Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.
A fractional-order model of COVID-19 considering the fear effect of the media and social networks on the community

Fatma Bozkurt\textsuperscript{a,b}, Ali Yousef\textsuperscript{a}, Thabet Abdeljawad\textsuperscript{c,d,e,f}, Adem Kalinli\textsuperscript{b,f}, Qasem Al Mdallal\textsuperscript{g}

\textsuperscript{a} Kuwait College of Science and Technology, Department of Mathematics, 27235 Kuwait City, Kuwait
\textsuperscript{b} Department of Mathematics, Erciyes University, 38039 Kayseri, Turkey
\textsuperscript{c} Department of Mathematics and General Sciences, Prince Sultan University, Riyadh 11586, Saudi Arabia
\textsuperscript{d} Department of Medical Research, China Medical University, Taichung, 40402, Taiwan
\textsuperscript{e} Department of Computer Science and Information Engineering, Asia University, Taichung, Taiwan
\textsuperscript{f} Rectorate, Middle East Technical University, 06800 Cankaya-Ankara, Turkey
\textsuperscript{g} United Arab Emirates University, Department of Mathematics, Al Ain, UAE


corresponding author.
E-mail address: tabdeljawad@psu.edu.sa (T. Abdeljawad).

abstract
Since December 2019, the world has experienced from a virus, known as Covid-19, that is highly transmittable and is now spread worldwide. Many mathematical models and studies have been implemented to work on the infection and transmission risks. Besides the virus’s transmission effect, another discussion appears in the community: the fear effect. People who have never heard about coronavirus, face every day uncertain and different information regarding the effect of the virus and the daily death rates from sources like the media, the medical institutions or organizations. Thus, the fear of the virus in the community can possibly reach the point that people become scared and confused about information polluted from different networks with long-term trend discussions. In this work, we use the Routh-Hurwitz Criteria to analyze the local stability of two essential critical points: the disease-free and the co-existing critical point. Using the discretization process, our analysis has shown that one should distinguish between the spread of “awareness” or “fear” in the community through the media and others to control the virus’s transmission. Finally, we conclude our theoretical findings with numerical simulations.

© 2021 Elsevier Ltd. All rights reserved.

1. Introduction

1.1. A brief information about SARS-CoV-2

Coronaviruses are a large group of viruses in the Coronaviridae family. This Coronaviridae is sub-divided into four groups of coronaviruses: \( \alpha \)–Coronavirus, \( \beta \)–Coronavirus, \( \gamma \)– Coronavirus, and \( \delta \)–Coronavirus [1]. These viruses were not seen as an attractive research field in applied sciences, since they are not highly pathogenic to humans until the outbreak of SARS-CoV in 2003 and MERS-CoV in 2012. Both viruses were transmitted directly from civets and dromedary camels (intermediate hosts) to the human hosts, respectively, and they showed the same natural host of bats in the epidemic spread [2,3]. Other coronaviruses such as HCoV-OC43 and HCoV-HKU1 that belong to the \( \beta \)-Coronaviruses are mostly harmless. These viruses showed endemic behaviors, and therefore the related studies were restricted to some areas such as medicine and biology, rarely also applied mathematics [4,5].

In 2003, the community started to hear about the coronavirus with the epidemic outbreak of SARS-CoV in China. This virus was transmitted to humans through intermediate hosts such as market civets. After that, people faced another infection in the Middle East in 2012, known as MERS-CoV, which spilled over to dromedary camels. These viruses showed severe respiratory syndromes in humans, including fever, dizziness, and cough [6–8]. However, all the coronaviruses mentioned above were endemic in the human populations causing 15–30% of respiratory tract infections each year. In December 2019, a virus of the Coronaviridae was announced in China-Wuhan. The spread was reported from the fishmarket in Wuhan using reptilians as intermediate hosts and bats as a natural host. A scenario was designed that the spread started in Wuhan’s local fish market, where the people used to buy bats. Apart from this point, another scenario was established in the community that a mutated virus from a Wuhan laboratory was spread to the world. The WHO later designated the novel coronavirus as COVID-19. Studies have shown that COVID-19 was characterized by two members of \( \beta \)-coronavirus; the human-origin coronavirus (SARS-CoV Tor2) and bat-origin coronavirus (bat-\( \beta \)-CoVZC45) [9]. A virus that was expected to show a similar epidemic behavior like SARS
or MERS reached a pandemic point worldwide with many open questions such as; how it started and how we can stop its spread.

1.2. A pandemic located virus in the era and the media fear effect on the civilians

A novel Coronavirus that appeared in December 2019 in China was able to spread in a few months worldwide so that on March 16th, 2020, the WHO upgraded the status of COVID-19 from epidemic to pandemic. To prevent the spread of infections brought after that, several fundamental protections such as quarantines, culling, heavy travel restrictions, and social distancing [10]. Health institutions and organizations took high responsibilities to engage the public with ‘healthy practices’ such as hand washing, keeping social distance, and staying at home during the lock-down period. Simultaneously, the governments started to collaborate with the WHO, and the media played a significant role in informing the civilians about the infection rates, the necessary health protections, and lock-down announcements from the government. Besides this, many practical and useless (unrealistic) information were also spread by the internet. People started to create their scenarios related to the virus, which leads to a fear effect in the community. Even the vaccine was suddenly a hope in a tunnel, according to The Economist [11], and not the end of the pandemic spread.

Considering all these discussions related to COVID-19, we want to emphasize the reality that the virus exists and is transmittable from human to human with a high reproduction number of $R[1,5]$. Until March 8th, 2021, which is more than one year after the spread in Wuhan, the number of infected reaches 177,476,407 cases with 2,606,051 deaths and 92,981,853 recovered. The USA shows the highest infection rate with 25,696,250, followed by India with 11,229,398 infections, and Brazil that hits 11,019,344 total cases. On the other side, an infection that started in China reached 89,994 infections, where the daily new case is recorded as 19 individuals [10]. Interestingly, a spread in China (Wuhan) did not show infections and fatalities reported in Europe, the USA, or other countries. Additionally, the mutated form of the virus is recorded mainly in Europe and the USA, which increased the mortality rate more than before.

In our study, we want to investigate the fear effect spread through the media and associations to the human in giving different daily information about the virus, the vaccines, and the protection rules. Considering some psychological problems such as anxiety, fear, or worry, it is seen that there is a positive correlation between the epidemiological spread and the psychological health among the students in China [12].

If we define now the virus as a predator and the civilians as prey, we can transform an experimental study to our case, which showed that apart from direct killing, the fear of the virus (predation fears) itself can reduce the prey growth rate (psychological effect) by 4% [13]. Finally, many studies in medicine and related fields started to concern their researches on the psychosocial impact of COVID-19, which shows similar dangerous results as the virus itself [14–20]. Unfortunately, uncontrollable information about the virus, the spread, mutation, and the vaccines lead people to suffer from uncertainty and activate a fear of the present and future. Therefore, we want to analyze and formulate an essential research topic related to the balance between controlling the people with various mechanisms and the community's fear during the lock-down period. Beyond that, we want to analyze the psychological pressure on civilians. Significantly, one should see that the change of “the normal life” reaches a psychological breakdown because of several waves of “lock-downs” and information pollutions so that people started to ask what is the “new normal life” with and after corona.

2. Mathematical model

It has been seen that many biological and medical phenomena can be characterized via mathematical models. Some mathematical models are formulated to analyze biology and medicine events such as infections, treatments, or environmental phenomena [21–24]. The study of these phenomena has been restricted to models of ODEs. However, it is realized that many problems in biology and other applied fields can be successfully formulated by the fractional-order differential equations (FDEs) that summarize the memory effect of the dynamical behavior. The nonlocal property of fractional-order models not only depends on the current state but also depends on its prior historical states. The transformation of an integer-order model into a fractional-order model needs to be precise with respect to the order of differentiation $\alpha$. However, a small change in $\alpha$ may cause a big change in the behavior of the solutions. Thus, it is preferred to use FDEs in biological models since they are relevant to memory and hereditary systems [25–29].

In this paper, we established SEIR + D model that describes the pandemic infection, in which the virus is located in the human body and continues to transmit the infection from human to human. The system is divided into six compartments. $S(t)$ shows the susceptible class that does not have any resistance to COVID-19. $E(t)$ shows the exposed compartment that has been infected, but since the virus is in the incubation period, they carry and transmit the virus without showing any symptoms. The $I(t)$ compartment is the infected group determined as COVID-19 positive, and $Q(t)$ shows the isolated class under the quarantine. $R(t)$ is the recovered compartment, while $D(t)$ denotes the death class. We established our model considering that humans show two types of fear during the movement from one compartment to the other: the fear of the susceptible class to be infected and the fear of the individuals under quarantine who are worried about the daily updated death rates. Therefore, in the system, $\alpha_1$ and $\alpha_2$ denote the level of the fear to be infected and the death from COVID-19. From a biological point,

$$f_1(\alpha_1, I) = \frac{1}{1 + \alpha_1 I}$$

and

$$f_2(\alpha_2, D) = \frac{1}{1 + \alpha_2 D}$$

can be reasonably assumed to satisfy the following statements;

2.1. Fear from infection COVID-19

$\alpha_1(0, I) = 1$: if there is no fear of the infection, then the susceptible class does not have the psychological pressure.

$\alpha_1(\alpha_1, 0) = 1$: if the virus disappears, then the fear and the psychological effect disappear on the susceptible class.

$\lim_{\alpha_1 \to \infty} f_1(\alpha_1, I) = 0$: if the virus's fear continues to expand more for a long-term period, then the “mentally healthy” susceptible class decreases to extinct.

$\lim_{\alpha_2 \to \infty} f_1(\alpha_2, I) = 0$: if the virus stays for a long-term period in a pandemic spread, the susceptible non-infected decreases to extinct.

$\frac{\partial f_1(\alpha_2, D)}{\partial \alpha_2} < 0$: if the fear effect increases, the offspring might be affected, and thus it decreases.

$\frac{\partial f_1(\alpha_2, D)}{\partial \alpha_2} < 0$: if the infection increases, the offspring might be affected, and thus it decreases.

2.2. Fear from the death of COVID-19

$\alpha_2(0, D) = 1$: if there is no fear of death from COVID-19, then the individuals under quarantine do not have the psychological pressure.

$\alpha_2(\alpha_2, 0) = 1$: if the death from COVID-19 decreases or extinct, then the fear and the psychological effect of it disappear on the individuals in the quarantine compartment.
\[ \lim_{\alpha \to \infty} f_2(\omega, D) = 0; \text{ if the fear of death from COVID-19 increases and expands more for a long-term period, then the "mentally healthy" class of the under quarantine decreases to extinct.} \]

\[ \lim_{0 \to \infty} f_2(\omega, D) = 0; \text{ if the death rate from COVID-19 increases in daily records, the quarantine compartment decreases to extinct because of death.} \]

\[ \frac{\partial f_2(\omega, D)}{\partial \omega} < 0; \text{ if the fear effect increases, the carrying capacity of the compartment under quarantine decreases.} \]

\[ \frac{\partial f_2(\omega, D)}{\partial D} < 0; \text{ if the death rate from COVID-19 increases, the compartment is carrying capacity under quarantine decreases.} \]

Thus, the mathematical model is modeled as follows:

\[
D^\alpha S(t) = \Lambda + S(t)r \left( 1 - \frac{S(t)}{K_S} \right) - \beta_1 E(t)S(t) - \gamma_1 I(t)S(t) - \eta S(t)
\]

\[
D^\alpha E(t) = E(t) \left( 1 - \frac{E(t)}{K_E} \right) + \beta_1 (1 - \varepsilon_1) E(t)S(t) - \theta E(t) - \eta E(t) - \mu E(t)
\]

\[
D^\alpha I(t) = I(t) \left( 1 - \frac{I(t)}{K_I} \right) + \beta_1 \varepsilon_1 E(t)S(t) + \gamma_1 I(t)S(t) + \theta E(t) - \beta_2 I(t) - \eta I(t) - \mu I(t)
\]

\[
D^\alpha Q(t) = Q(t) \left( 1 - \frac{Q(t)}{K_Q} \right) + \beta_2 I(t) - \gamma_2 Q(t) - \mu Q(t) - \gamma_2 Q(t)
\]

\[
D^\alpha R(t) = \gamma_2 Q(t) - \eta R(t)
\]

\[
D^\alpha D(t) = \mu (E(t) + I(t) + Q(t)) - \mu I(t)
\]

and

\[
S(0) = S_0, \quad E(0) = E_0, \quad I(0) = I_0, \quad Q(0) = Q_0, \quad R(0) = R_0 \quad \text{and} \quad D(0) = D_0.
\]

where the parameters are positive real numbers, \(0 < \alpha \leq 1\). \(D^\alpha\) denotes the Caputo derivative and \((S, E, I, Q, R, D) \in \mathbb{R}^6_+\).

The susceptible \(S(t)\) comprises individuals who have not contacted any infected person, but can get infected from COVID-19 infected people. \(S(t)\) can get infected through \(E(t)\), who do not know they are infected. It is also possible that the class \(S(t)\) noticed they were in contact with the infected class \(I(t)\). \(r\) is the growth rate of \(S(t)\), while \(\Lambda\) denotes the susceptible population’s offspring per spring. \(K_S\) shows the carrying capacity of \(S(t)\). The susceptible class lost their population density following contacts with infectious \(E(t)\) and \(I(t)\) at a rate of \(\beta_1\) and \(\gamma_1\) respectively. \(\eta\) shows natural death rate of the susceptible class \(S(t)\).

The \(E(t)\) class does not know that they have COVID-19 because of the late appeared symptoms of the infection. In this equation \(K_E\) is the carrying capacity of \(E(t)\). This class population decreases after being informed about the health organizations’ virus and becomes aware of screening at a rate \(\theta\). Another possibility is that after the symptoms appear with a rate of \(\varepsilon_1\) the class moves to the infected compartment \(I(t)\). For the exposed compartment, \(\eta\) shows the natural death and \(\mu\) death from COVID-19.

\(I(t)\) is the infected COVID-19 class. The carrying capacity is given by \(K_I\). The population of this class increases with \(\beta_1\varepsilon_1\) who noticed in the exposed class from the appeared symptoms that they are infected. Another parametric increase comes from the screening rate \(\theta\) when the test shows COVID-19 positive. Moreover, any contact between \(S(t)\) and \(I(t)\) is realized by the susceptible class with \(\gamma_1\) that increase the population of the infected \(I(t)\) group. We assume that the individuals are aware to inform the health organizations when they feel the infection symptoms. From the infected compartment, a rate of \(\beta_2\) moves to a hospitalized compartment or stays under quarantine at home.

The \(Q(t)\) has a carrying capacity of \(K_Q\). This class shows a fear effect \(\omega_2\) of the daily recorded death rates. The population density of the compartment increases with \(\beta_2\) \((t)\) who shows mainly severe symptoms and are hospitalized under quarantine. \(\gamma_2\) is the rate of recovered individuals during the quarantine period that changes the class from \(Q(t)\) to \(R(t)\). In this compartment, we expect two types of recorded deaths; natural death and death from COVID-19.

The \(R(t)\) compartment shows the class of recovered individuals. \(\gamma_2 Q(t)\) shows the increase of the compartment that is expected from class \(Q(t)\) after successful treatment, the recovered class shows only the natural death of a rate \(\eta\).

The most discussed part in the community was the case of “death from COVID-19" and “death with COVID-19”.

This case in the compartment of \(D(t)\), the death of the infected population is subtracted from death who died from other symptoms that affect the immune system, which is denoted as \(\mu_1\); this means, \(D(t)\) denotes the group that died from corona itself.

This study focuses mainly on five essential and sensitive parameters. These are; \(\alpha_1\), the fear to be infected, \(\alpha_2\), the fear to die from COVID-19, \(\theta\), the rate of screening, \(\varepsilon_1\), the rate of awareness and \(\gamma_2\), rate of recovering of successful treatment. In this work, we want to distinguish between “to fear" and "to get the awareness". Specific and precise information from professional institutions without any confusion would lead civilians to be aware and not scared of the virus. To get used to considering how to live in this pandemic spread and to take the necessary actions can be explained without terrifying people such as “you may die”. Professional screening tests would reach to successful movements between the compartments. For example, why does the media do not show successful treatments and all networks are spread from the same source to show hope and, after that, to take the hope from a human? This study will analyze if the lock-down, pollution of information related to COVID-19, and daily announced record from only deaths and infections lead to awareness or fears

**Definition 2.1.** [30] Given a function \(\varphi(t)\), the fractional integral with order \(\alpha > 0\) is given by Abel’s formula as

\[
I_0^\alpha \varphi(t) = \frac{1}{\Gamma(\alpha)} \int_0^t (x-t)^{\alpha-1} \varphi(x) dx, \quad x > 0.
\]

**Definition 2.2.** [30] Let \(\varphi : R^+ \rightarrow R\) be a continuous function. The Caputo fractional derivative of order \(\alpha \in (n - 1, n)\), where \(n\) is a positive integer is defined as

\[
D_0^\alpha \varphi(x) = \frac{1}{\Gamma(n-\alpha)} \int_0^x \frac{\varphi^{(n)}(t)}{(x-t)^{\alpha+1-n}} dt.
\]

When \(n\), the derivatives are defined to be the usual \(nth\) order derivatives.

**Definition 2.3.** [31] The Mittag-Leffler function of one variable is

\[
E_\alpha(\lambda, z) = E_\alpha(\lambda z^\alpha) = \sum_{k=0}^{\infty} \frac{\lambda^k z^k}{\Gamma(1+\alpha k)}, \quad (\lambda \neq 0, z \in C : Re(\alpha) > 0).
\]
The table of the parametric description is given as follows;

3. Equilibrium points of system (2.1) and the existence-uniqueness of the solutions in the system

To consider the system’s biological validity, we need to show that for all non-negative initial values, the solutions of system (2.1) remain non-negative. Thus, the preliminary studies include proving that the domain is positive and that the IVP system has a unique solution in \( \mathbb{R}^6_+ \). After that, two equilibrium points are defined to establish the core studies; the disease-free equilibrium point and positive co-existing point.

Denote \( \mathbb{R}^6_+ = \{ B \in \mathbb{R}^6 : B \geq 0 \} \) and let \( B(t) = (S(t), E(t), I(t), Q(t), R(t), D(t))^T \). For the proof of Theorem 3.1, we need the following lemma [32].

Lemma 3.1. [32] (Generalized Mean Value Theorem)

Let \( f(x) \in \mathcal{C}[a, b] \) and \( D^\alpha f(x) \in \mathcal{C}(a, b) \) for \( 0 < \alpha \leq 1 \), then we have

\[
f(x) = f(a) + \frac{1}{\Gamma(\alpha)} D^\alpha f(\xi)(x-a)^\alpha
\]

with \( 0 \leq \xi \leq x, \forall x \in (a, b) \).

Lemma 3.2. [33] Suppose \( f(x) \in \mathcal{C}[0, b] \) and \( D^\alpha f(x) \in \mathcal{C}[0, b] \) for \( 0 < \alpha \leq 1 \). Then, from Lemma 3.1, we have the following statements.

(i) \( f \) is non-decreasing if \( D^\alpha f(x) \geq 0, \forall x \in (0, b) \)
(ii) \( f \) is non-increasing if \( D^\alpha f(x) \leq 0, \forall x \in [0, b] \)

Theorem 3.1. The solution of the IVP in (2.1)-(2.2) is unique and the solutions are in \( \mathbb{R}^6_+ \).

Proof. Lemma 3.1. and Lemma 3.2. show the existence and uniqueness of (2.1)-(2.2) in \((0, \infty )\). Thus, we want to show that the domain \( \mathbb{R}^6_+ \) is positively invariant following the technique of [34]. Notice that

\[
D^\alpha S(t)|_{t=0} = \Lambda \geq 0
\]

\[
D^\alpha E(t)|_{t=0} = 0
\]

\[
D^\alpha I(t)|_{t=0} = \beta_1 \epsilon_1 E(t) S(t) + \theta E(t) \geq 0
\]

\[
D^\alpha Q(t)|_{t=0} = \beta_2 I(t) \geq 0
\]

\[
D^\alpha R(t)|_{t=0} = \gamma_2 Q(t) \geq 0
\]

\[
D^\alpha D(t)|_{t=0} = \mu (E(t) + I(t) + Q(t)) \geq 0
\]

on each hyperplane bounding the nonnegative orthant. Hence the domain \( \mathbb{R}^6_+ \) is positively invariant. This completes the proof.

Let us rewrite the system

\[
D^\alpha S(t) = f_1(S(t), E(t), I(t), Q(t), R(t), D(t)) = \Lambda + S(t) \epsilon_1 \left( 1 - \frac{S(t)}{K_1} \right) \frac{1}{1 + \alpha_1 I(t)} - \beta_1 E(t) S(t) - \gamma_1 I(t) S(t) - \eta S(t)
\]

\[
D^\alpha E(t) = f_2(S(t), E(t), I(t), Q(t), R(t), D(t)) = E(t) \left( 1 - \frac{E(t)}{K_2} \right) + \beta_1 (1 - \epsilon_1) E(t) S(t) - \theta E(t) - \eta E(t) - \mu E(t)
\]

\[
D^\alpha I(t) = f_3(S(t), E(t), I(t), Q(t), R(t), D(t)) = I(t) \left( 1 - \frac{I(t)}{K_3} \right) + \beta_1 \epsilon_1 E(t) S(t) + \gamma_1 I(t) S(t) + \theta E(t) - \beta_2 I(t) - \eta I(t) - \mu I(t)
\]

\[
D^\alpha Q(t) = f_4(S(t), E(t), I(t), Q(t), R(t), D(t)) = Q(t) \left( 1 - \frac{Q(t)}{K_4} \right) + \beta_2 I(t) - \eta Q(t) - \mu Q(t) - \gamma_2 Q(t)
\]

\[
D^\alpha R(t) = f_5(S(t), E(t), I(t), Q(t), R(t), D(t)) = \gamma_2 Q(t) - \eta R(t)
\]

\[
D^\alpha D(t) = f_6(S(t), E(t), I(t), Q(t), R(t), D(t)) = \mu (E(t) + I(t) + Q(t)) - \mu_1 D(t)
\]

To analyze the stability of (3.2), we perturb the equilibrium points by \( \epsilon_i(t) > 0, i = 1, 2, 3, 4, 5, 6 \), that is

\[
S(t) - \tilde{S} = \epsilon_1(t) \quad E(t) - \tilde{E} = \epsilon_2(t) \quad I(t) - \tilde{I} = \epsilon_3(t) \quad Q(t) - \tilde{Q} = \epsilon_4(t) \quad R(t) - \tilde{R} = \epsilon_5(t) \quad D(t) - \tilde{D} = \epsilon_6(t)
\]

Thus, we have

\[
D^\alpha (\epsilon_1(t)) \approx \frac{\partial f_1(\tilde{S}, \tilde{E}, \tilde{I}, \tilde{Q}, \tilde{R}, \tilde{D})}{\partial S} \epsilon_1(t) + \frac{\partial f_1(\tilde{S}, \tilde{E}, \tilde{I}, \tilde{Q}, \tilde{R}, \tilde{D})}{\partial E} \epsilon_2(t) + \frac{\partial f_1(\tilde{S}, \tilde{E}, \tilde{I}, \tilde{Q}, \tilde{R}, \tilde{D})}{\partial I} \epsilon_3(t) + \frac{\partial f_1(\tilde{S}, \tilde{E}, \tilde{I}, \tilde{Q}, \tilde{R}, \tilde{D})}{\partial Q} \epsilon_4(t) + \frac{\partial f_1(\tilde{S}, \tilde{E}, \tilde{I}, \tilde{Q}, \tilde{R}, \tilde{D})}{\partial R} \epsilon_5(t) + \frac{\partial f_1(\tilde{S}, \tilde{E}, \tilde{I}, \tilde{Q}, \tilde{R}, \tilde{D})}{\partial D} \epsilon_6(t)
\]
\[ D^\alpha (e_2(t)) \simeq f_2(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D}) + \frac{\partial f_2(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial S} e_1(t) + \frac{\partial f_2(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial E} e_2(t) + \frac{\partial f_2(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial I} e_3(t) \]
\[ + \frac{\partial f_2(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial Q} e_4(t) + \frac{\partial f_2(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial R} e_5(t) + \frac{\partial f_2(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial D} e_6(t). \]

\[ D^\alpha (e_3(t)) \simeq f_3(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D}) + \frac{\partial f_3(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial S} e_1(t) + \frac{\partial f_3(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial E} e_2(t) + \frac{\partial f_3(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial I} e_3(t) \]
\[ + \frac{\partial f_3(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial Q} e_4(t) + \frac{\partial f_3(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial R} e_5(t) + \frac{\partial f_3(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial D} e_6(t). \]

\[ D^\alpha (e_4(t)) \simeq f_4(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D}) + \frac{\partial f_4(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial S} e_1(t) + \frac{\partial f_4(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial E} e_2(t) + \frac{\partial f_4(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial I} e_3(t) \]
\[ + \frac{\partial f_4(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial Q} e_4(t) + \frac{\partial f_4(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial R} e_5(t) + \frac{\partial f_4(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial D} e_6(t). \]

\[ D^\alpha (e_5(t)) \simeq f_5(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D}) + \frac{\partial f_5(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial S} e_1(t) + \frac{\partial f_5(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial E} e_2(t) + \frac{\partial f_5(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial I} e_3(t) \]
\[ + \frac{\partial f_5(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial Q} e_4(t) + \frac{\partial f_5(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial R} e_5(t) + \frac{\partial f_5(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial D} e_6(t). \]

\[ D^\alpha (e_6(t)) \simeq f_6(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D}) + \frac{\partial f_6(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial S} e_1(t) + \frac{\partial f_6(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial E} e_2(t) + \frac{\partial f_6(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial I} e_3(t) \]
\[ + \frac{\partial f_6(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial Q} e_4(t) + \frac{\partial f_6(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial R} e_5(t) + \frac{\partial f_6(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D})}{\partial D} e_6(t). \]

The perturbation around the equilibrium point is to linearize the nonlinear system based on a fractional-order Lotka-Volterra logistic equation. We use the property of equilibrium point that
\[ f_i(\dot{S}, \dot{E}, \dot{I}, \dot{Q}, \dot{R}, \dot{D}) = 0, \quad i = 1, 2, 3, 4, 5, 6, \]
and therefore, a linearized system about the equilibrium point is obtained, such as
\[ D^\alpha V = JF, \]
where \( V = (e_1(t), e_2(t), e_3(t), e_4(t), e_5(t), e_6(t)) \). Furthermore, \( J \) is the Jacobian matrix at the equilibrium point. Moreover, we have \( W^{-1}JV = C \) such that \( C \) is the diagonal matrix of \( \lambda_i(1 = 1, 2, 3, 4, 5, 6) \) and \( W \) shows the eigenvectors of \( J \). Thus, we have
\[
\begin{align*}
D^\alpha \psi_1 &= \lambda_1 \psi_1, \\
D^\alpha \psi_2 &= \lambda_2 \psi_2, \\
D^\alpha \psi_3 &= \lambda_3 \psi_3, \quad \text{where} \quad \psi = \begin{pmatrix} \psi_1 \\ \psi_2 \\ \psi_3 \\ \psi_4 \\ \psi_5 \\ \psi_6 \end{pmatrix}, \\
D^\alpha \psi_4 &= \lambda_4 \psi_4, \\
D^\alpha \psi_5 &= \lambda_5 \psi_5, \\
D^\alpha \psi_6 &= \lambda_6 \psi_6,
\end{align*}
\]
and the solutions are given by Mittag-Leffler functions such as
\[
\begin{align*}
\psi_1(t) &= \sum_{n=0}^{\infty} \frac{\lambda_1^n t^{\alpha n}}{\Gamma(n\alpha + 1)} \psi_1(0) = E_{\alpha}(\lambda_1 t^\alpha) \psi_1(0), \\
\psi_2(t) &= \sum_{n=0}^{\infty} \frac{\lambda_2^n t^{\alpha n}}{\Gamma(n\alpha + 1)} \psi_2(0) = E_{\alpha}(\lambda_2 t^\alpha) \psi_2(0), \\
\psi_3(t) &= \sum_{n=0}^{\infty} \frac{\lambda_3^n t^{\alpha n}}{\Gamma(n\alpha + 1)} \psi_3(0) = E_{\alpha}(\lambda_3 t^\alpha) \psi_3(0), \\
\psi_4(t) &= \sum_{n=0}^{\infty} \frac{\lambda_4^n t^{\alpha n}}{\Gamma(n\alpha + 1)} \psi_4(0) = E_{\alpha}(\lambda_4 t^\alpha) \psi_4(0), \\
\psi_5(t) &= \sum_{n=0}^{\infty} \frac{\lambda_5^n t^{\alpha n}}{\Gamma(n\alpha + 1)} \psi_5(0) = E_{\alpha}(\lambda_5 t^\alpha) \psi_5(0), \\
\psi_6(t) &= \sum_{n=0}^{\infty} \frac{\lambda_6^n t^{\alpha n}}{\Gamma(n\alpha + 1)} \psi_6(0) = E_{\alpha}(\lambda_6 t^\alpha) \psi_6(0).
\end{align*}
\]
and
\[ \psi(t) = \sum_{n=0}^{\infty} \left( \frac{\lambda_n^{1+n\alpha}}{(n\alpha + 1)^{1+n\alpha}} \right) \psi(0) = E_{\alpha,\delta}^{\alpha}(t) \psi(0). \]

Matignon studied in [35] a system of fractional order differential equations involving the Caputo derivative
\[ D^\alpha y(t) = Ay(t) \] (3.7)
with initial values \( y(0) = y_0 = (y_{01}, y_{20}, \ldots, y_{n0})^T \), where \( y = (y_1, y_2, \ldots, y_n)^T, \alpha \in (0, 1) \) and \( A \in \mathbb{R}^{n \times n} \). The stability of the system given in (3.7) was defined by Matignon as follows.

**Definition 3.1.** The autonomous system (3.7) is said to be
(i) stable if and only if for any \( y_0 \), there exists \( \delta > 0 \) such that \( \|y(t)\| \leq \delta \) for \( t \geq 0 \).
(ii) asymptotically stable if and only if \( \lim_{n \to \infty} \|y(t)\| = 0 \).

**Theorem 3.2.** [36] The system (3.7) is
(i) asymptotically stable if and only if \( |\arg(\alpha_i)| > \frac{\pi}{\alpha} \).
(ii) stable \iff either it is asymptotically stable or the critical values which satisfy \( |\arg(\alpha_i)| = \frac{\pi}{\alpha} \) have geometric multiplicity one.

Matignon sketched the proof of Theorem 3.2 in [35], and Zeng et al. proved the theorem using the Mittag-Leffler functions [37]. Thus, considering system (3.5), we can say that if
\[ |\arg(\alpha_i)| > \frac{\pi}{\alpha} \] (i = 1, 2, 3, 4, 5, 6), then \( \psi_i (i = 1, 2, 3, 4, 5, 6) \) are decreasing and therefore, \( \varepsilon^i (i = 1, 2, 3, 4, 5, 6) \) are decreasing. In other words, let the solution \( V = (\varepsilon_1(t), \varepsilon_2(t), \varepsilon_3(t), \varepsilon_4(t), \varepsilon_5(t), \varepsilon_6(t) \) of (2.10) exist. If the solution of (3.5) is increasing, then the equilibrium point \((\bar{S}, \bar{E}, \bar{I}, \bar{Q}, \bar{R}, \bar{D})\) of the system is unstable. Similarly, if the solution of (3.5) is decreasing, then the equilibrium point \((\bar{S}, \bar{E}, \bar{I}, \bar{Q}, \bar{R}, \bar{D})\) is locally asymptotically stable.

This study focuses on two equilibrium points; the disease-free equilibrium point and the co-existing equilibrium point. These are given as follows;

**Disease-free equilibrium point:** \( \chi_1 = (\bar{S}_1, 0, 0, 0, 0, 0) \), where
\[ \bar{S}_1 = \frac{K_1(r - \eta) + \sqrt{K_1^2(r - \eta)^2 + 4r\lambda}}{2r} \] or \( r > \eta \). (3.8)

**Co-existing equilibrium point:** \( \chi_2 = (\bar{S}_2, \bar{E}_2, \bar{I}_2, \bar{Q}_2, \bar{R}_2, \bar{D}_2) \).

**4. Local stability analysis of the disease-free and co-existing equilibrium points**

This section analyzes the local stability of system (3.2) around each equilibrium point defined as \( \chi_1 \) and \( \chi_2 \). Hereafter, we assume that \( r > \eta \).

The Jacobian matrix of the disease-free point \( \chi_1 = (\bar{S}_1, 0, 0, 0, 0, 0) \) is given by
\[ J(\chi_1) = \begin{pmatrix} a_{11} & a_{12} & a_{13} & 0 & 0 & 0 \\ 0 & a_{22} & 0 & 0 & 0 & 0 \\ 0 & a_{31} & a_{33} & 0 & 0 & 0 \\ 0 & 0 & a_{43} & a_{44} & 0 & 0 \\ 0 & 0 & 0 & a_{54} & a_{55} & 0 \\ 0 & a_{62} & a_{63} & a_{64} & 0 & a_{66} \end{pmatrix} \] (4.1)

where
\[ a_{11} = r - \frac{2}{K_1} \bar{S}_1 - \eta, \quad a_{12} = -\beta_1 \bar{S}_1, \quad a_{13} = -\gamma_1 \bar{S}_1 - \alpha_1 \bar{S}_1 r (1 - \frac{\bar{S}_1}{K_1}), \quad a_{14} = a_{15} = a_{16} = 0, \quad a_{21} = a_{22} = a_{23} = a_{24} = a_{25} = a_{26} = 0, \quad a_{27} = 1 + \beta_2 (1 - \varepsilon_1) \bar{S}_1 - (\theta + \varepsilon_1 + \mu) \],
\[ a_{31} = a_{34} = a_{35} = a_{36} = 0, \quad a_{32} = \beta_1 \varepsilon_1 \bar{S}_1 + \theta, \quad a_{33} = 1 + \gamma_1 \bar{S}_1 - (\beta_2 + \eta + \mu), \quad a_{41} = a_{42} = a_{45} = a_{46} = 0, \quad a_{43} = a_{44} = 1 - (\eta + \mu + \gamma_2), \quad a_{51} = a_{52} = a_{53} = a_{56} = 0, \quad a_{54} = a_{55} = \gamma_2, \quad a_{55} = -\eta, \quad a_{61} = a_{65} = 0, \quad a_{62} = a_{63} = a_{64} = \mu \] and
\[ a_{66} = -\mu. \]

Thus, the Eq. (4.1) around the positive equilibrium point \( \chi_1 \) is given by
\[ (a_{11} - \lambda)(a_{22} - \lambda)(a_{33} - \lambda)(a_{44} - \lambda)(a_{55} - \lambda)(a_{66} - \lambda) = 0. \] (4.2)

**Theorem 4.1.** Let \( \chi_1 \) be the disease-free equilibrium point of system (3.2). Assume that \( \theta > \beta_2 > \gamma_2 > 1, \beta_1 > \gamma_1 \) and \( \varepsilon_1 > \frac{\beta_1 - \gamma_1}{\beta_1} \). Then \( \chi_1 \) is stable local asymptotic if and only if
\[ \frac{\bar{S}_1}{\gamma_1} < \frac{\beta_2 + \eta + \mu - 1}{\gamma_1}. \] (4.3)

**Proof.** From (4.2), it follows that
(i) \( \lambda_1 = r - \frac{2}{K_1} \bar{S}_1 - \eta < 0 \), since \( \bar{S}_1 = \frac{K_1(r - \eta) + \sqrt{K_1^2(r - \eta)^2 + 4r\lambda}}{2r} \) for \( r > \eta \).
(ii) \( \lambda_2 = 1 + \beta_1 (1 - \varepsilon_1) \bar{S}_1 - (\theta + \varepsilon_1 + \mu) < 0 \), if
\[ \bar{S}_1 < \frac{\theta + \eta + \mu - 1}{\beta_1(1 - \varepsilon_1)} \] for \( \theta + \eta + \mu > 1 \) and \( \varepsilon_1 < 1 \). (4.4)
(iii) \( \lambda_3 = 1 + \gamma S_1 - (\beta_2 + \eta + \mu) < 0. \) if
\[
\tilde{S}_1 > \frac{\beta_2 + \eta + \mu - 1}{\gamma_1} \quad \text{for} \quad \beta_2 + \eta + \mu > 1.
\] (4.5)

(iv) \( \lambda_4 = 1 - (\eta + \mu + \gamma_2) < 0, \) if \( \eta + \mu + \gamma_2 > 1. \)

(v) \( \lambda_5 = -\eta < 0. \)

(vi) \( \lambda_5 = -\mu_1 < 0. \)

In considering both (4.4) and (4.5), we obtain
\[
\tilde{S}_1 < \frac{\beta_2 + \eta + \mu - 1}{\gamma_1} < \frac{\theta + \eta + \mu - 1}{\beta_1 (1 - \varepsilon_1)}
\] (4.6)
since \( \theta > \beta_2 \) and \( \varepsilon_1 > \frac{\beta_2 + \eta}{\beta_1} \) for \( \beta_1 > \gamma_1. \)

**Remark 4.1.** Theorem 4.1 shows how the epidemiological phenomena can be reduced to a disease-free event. The increase of the screening rate will detect the infected people before transmitting the disease to others. Thus, to reduce infection transmission, it is expected to increase the screening denoted as \( \theta. \) The next stage is to expect successful isolation of the infected people and recovering in stabilizing the immune system of the human body. The awareness of the civilians, given as \( \varepsilon_1, \) shows a critical role in minimizing the spread of infection. This awareness is to apply the rules like keeping social distance to individuals, wearing a mask,… The major risk of transmitting the disease to the susceptible group is seen from the exposed compartment, which does not show its symptoms. Therefore, we conclude that one cannot know or predict who is infected, so permanent health practices are advised to reach a disease-free community.

One can notice that the disease-free stability criteria do not contain any fear of the infection or die from the infection. The stability shows up in two primary parametric forms, the screening rate and the infection’s awareness.

The Jacobian matrix of the co-existing equilibrium point \( X_2 = (S_2, E_2, I_2, Q_2, R_2, D_2) \) is given as
\[
J(X_2) = \begin{pmatrix}
11 & 12 & 13 & 0 & 0 & 0 \\
21 & 22 & 0 & 0 & 0 & 0 \\
31 & 32 & 33 & 0 & 0 & 0 \\
0 & 0 & 44 & 0 & 0 & 0 \\
0 & 0 & 0 & 64 & 65 & 0 \\
0 & 62 & 63 & 64 & 0 & 66
\end{pmatrix}
\] (4.7)

where
\[
a_{11} = \frac{1}{1 - \alpha_1 I_2} - \beta_1 E_2 - \gamma_1 I_2 - \eta, a_{12} = -\beta_2 S_2, a_{13} = -\gamma_1 S_2 - \alpha_1 f \left(1 - \frac{S_1}{R_1}\right),
a_{21} = \beta_1 (1 - \varepsilon_1) E_2, a_{22} = 1 - \frac{2}{\alpha_2} E_2 + \beta_1 (1 - \varepsilon_1) S_2 - (\theta + \eta + \mu), a_{31} = \beta_1 E_1 + \gamma_1 I_2, a_{32} = \beta_1 E_1 S_2 + \theta,
a_{33} = \gamma_1 S_2 - (\beta_2 + \eta + \mu), a_{43} = \beta_2, a_{44} = 1 - \frac{2}{\alpha_2} \tilde{S}_2 \left[1 - \frac{\tilde{S}_2}{(1 + \alpha_2 \tilde{S}_2)}\right], a_{45} = \gamma_2, a_{55} = -\eta, a_{62} = a_{63} = a_{64} = \mu, \text{ and } a_{66} = -\mu_1.
\]

From the characteristic equation of (4.7), we obtain
\[
\{(a_{44} - \lambda)(a_{66} - \lambda) - a_{46} a_{64}\} \{(a_{22} - \lambda)(a_{11} - \lambda)(a_{33} - \lambda) - a_{13} a_{31}\} + a_{21} a_{43} a_{31} = a_{12} (a_{33} - \lambda) = 0.
\] (4.8)

where
\[
\lambda_5 = -\eta < 0.
\] (4.9)

In (4.8), we consider at first the characteristic equation
\[
\lambda^2 - (a_{44} + a_{66})\lambda + a_{44}a_{66} - a_{46}a_{64} = 0.
\] (4.10)

which shows a basic reproduction number of \( R_{01} = \frac{a_{44}a_{66}}{a_{46}a_{64}}. \) This has an equation of a form such as
\[
\lambda^2 - (a_{44} + a_{66})\lambda + a_{44}a_{66}(1 - R_{01}) = 0.
\] (4.11)

The reproduction number \( R_{01} \) is dependent mainly on the carrying capacity of the individuals who are under quarantine. The increase of the numbers in the Q compartment activates the fear of deaths of COVID-19 infected. The further studies in section 4 will show that the fear of human focus exactly on the total number of deaths announced from WHO (with and from COVID-19) and not the deaths of the infected groups who died from COVID-19.

Considering the characteristic equation (4.8) again, we want to incorporate a second reproduction number \( R_{02} = \frac{a_{13}a_{31}}{a_{11}a_{33}}, \) which belongs to the susceptible and infected compartments, which is
\[
\lambda^2 - (a_{11} + a_{33})\lambda + a_{11}a_{33}(1 - R_{02}) = 0.
\] (4.12)

The basic reproduction number \( R_{02} \) shows that a fear of the susceptible class to get infected with the novel coronavirus is proportional to the population density loss. If the infection rate increases, the fear of the susceptible compartment increases. This fear of the susceptible class being infected by COVID-19 started to exists after considering the total number of deaths of infected people (with and from COVID-19).
In this scenario, we assume a total awareness of $\varepsilon_1 = 1$ focuses mainly on the screening effect and the fear caused by different sources giving pollution of information. Thus, the characteristic equation in (4.8) changes to
\[
(\lambda^2 - (a_{44} + a_{66})\lambda + a_{44}a_{66}(1 - R_{01})) (\lambda^2 - (a_{11} + a_{33})\lambda + a_{11}a_{33}(1 - R_{02})) = 0, \tag{4.13}
\]
and
\[
\lambda_2 < 1 \quad \text{for} \quad \tilde{E}_2 < \frac{K_4(1 - (\theta + \eta + \mu))}{2} \quad \text{(existence of the exposed compartment)}, \tag{4.14}
\]
where $\theta + \eta + \mu < 1$.

We assume in the following theorem that the compartments cannot exceed their carrying capacity.

**Theorem 4.2.** Let $\gamma_2$ be the positive equilibrium point of system (3.2) and assume that $R_{01} < 1$ and $R_{02} < 1$. Moreover, let the conditions $\eta + \mu + \gamma_2 + \mu_1 < 1$ and $\beta_2 + \eta + \mu > 1$ hold. If
\[
1 \quad \frac{D_2}{D_2} \left( \frac{K_4 - 2\tilde{Q}_2}{K_4(\eta + \mu + \gamma_2 + \mu_1)} - 1 \right) < \alpha_2 < \frac{1}{D_2} \left( \frac{K_4 - 2\tilde{Q}_2}{K_4(\eta + \mu + \gamma_2)} - 1 \right) \quad \text{for} \quad \tilde{Q}_2 < \frac{K_4(1 - (\eta + \mu + \gamma_2 + \mu_1))}{2}, \tag{4.15}
\]
\[
1 \quad \frac{D_2}{D_2} \left( \frac{K_1 r - 2r\tilde{S}_2}{K_1(\beta_1\tilde{E}_2 + \gamma_1\tilde{I}_2 + \beta_2 + 2\eta + \mu - 1)} - 1 \right) < \alpha_1 < \frac{1}{D_2} \left( \frac{K_1 r - 2r\tilde{S}_2}{K_1(\beta_1\tilde{E}_2 + \gamma_1\tilde{I}_2 + \eta)} - 1 \right) \quad \text{for} \quad \tilde{S}_2 < \frac{2}{K_3\gamma_1} \tilde{I}_2, \tag{4.16}
\]
where
\[
\beta_1\tilde{E}_2 + \gamma_1\tilde{I}_2 < r - \eta \quad \text{and} \quad K_1K_3 > \frac{4r}{\gamma_1} \quad \text{from} \quad \gamma_1((r - \eta - \beta_1\tilde{E}_2 - \gamma_1\tilde{I}_2) + (1 - \beta_2 - \eta - \mu)).
\]

then the roots of (4.13) are real or complex conjugates with negative real parts and $|\arg(\lambda_i)| > \frac{\alpha}{i}$ (i = 1, 2, 3, 4). $\alpha \in (0, 1)$ is equivalent to the Routh-Hurwitz Criteria. This implies that $\gamma_2$ is locally asymptotically stable.

**Proof.** Let us consider at first the isolated (under quarantine) and the death compartment, where
\[
\Delta_1 = (a_{44} + a_{66})^2 - 4a_{44}a_{66}(1 - R_{01}) > 0. \tag{4.17}
\]

From (4.17), we have
\[
\alpha_2 < \frac{1}{D_2} \left( \frac{K_4 - 2\tilde{Q}_2}{K_4(\eta + \mu + \gamma_2)} - 1 \right) \quad \text{and} \quad \tilde{Q}_2 < \frac{K_4(1 - (\eta + \mu + \gamma_2))}{2}, \tag{4.18}
\]
where $R_{01} < 1$ and $\eta + \mu + \gamma_2 < 1$.

Moreover, computations show that
\[
a_{44} + a_{66} < 0 \Rightarrow \left( 1 - \frac{2}{K_4} \tilde{Q}_2 \right) \frac{1}{1 + \alpha_2 D_2} - (\eta + \mu + \gamma_2) - \mu_1 < 0, \tag{4.19}
\]
if
\[
\alpha_2 > \frac{1}{D_2} \left( \frac{K_4 - 2\tilde{Q}_2}{K_4(\eta + \mu + \gamma_2 + \mu_1)} - 1 \right) \quad \text{and} \quad \tilde{Q}_2 < \frac{K_4(1 - (\eta + \mu + \gamma_2 + \mu_1))}{2}, \tag{4.20}
\]
where $\eta + \mu + \gamma_2 + \mu_1 < 1$.

From both (4.18) and (4.20), we obtain
\[
1 \quad \frac{D_2}{D_2} \left( \frac{K_4 - 2\tilde{Q}_2}{K_4(\eta + \mu + \gamma_2 + \mu_1)} - 1 \right) < \alpha_2 < \frac{1}{D_2} \left( \frac{K_4 - 2\tilde{Q}_2}{K_4(\eta + \mu + \gamma_2)} - 1 \right), \tag{4.21}
\]
where $\tilde{Q}_2 < \frac{K_4(1 - (\eta + \mu + \gamma_2 + \mu_1))}{2}$ and $\eta + \mu + \gamma_2 + \mu_1 < 1$.

Considering now the discriminant of the characteristic equation of the $S - I$ compartments, we have
\[
\Delta_2 = (a_{11} + a_{33})^2 - 4a_{11}a_{33}(1 - R_{02}) > 0. \tag{4.22}
\]

If $R_{02} < 1$ and the following statements hold:
\[
\alpha_1 < \frac{1}{D_2} \left( \frac{K_1 r - 2r\tilde{S}_2}{K_1(\beta_1\tilde{E}_2 + \gamma_1\tilde{I}_2 + \eta)} - 1 \right) \quad \text{for} \quad \tilde{S}_2 < \frac{K_1(r - \eta - \beta_1\tilde{E}_2 - \gamma_1\tilde{I}_2)}{2r}, \tag{4.23}
\]
where $\beta_1\tilde{E}_2 + \gamma_1\tilde{I}_2 < r - \eta$, and
\[
\tilde{S}_2 \frac{2}{K_3\gamma_1} \tilde{I}_2 < r - \eta \quad \text{and} \quad \gamma_1((r - \eta - \beta_1\tilde{E}_2 - \gamma_1\tilde{I}_2) + (1 - \beta_2 - \eta - \mu)).
\]

hold. From (4.23) and (2.24), we have $K_1K_3 > \frac{4r}{\gamma_1(r - \eta - \beta_1\tilde{E}_2 - \gamma_1\tilde{I}_2)}$ such that
\[
\tilde{S}_2 < \frac{2}{K_3\gamma_1} \tilde{I}_2 < \frac{K_1(r - \eta - \beta_1\tilde{E}_2 - \gamma_1\tilde{I}_2)}{2r}.
\]
Furthermore, we get that
\[
\alpha_{11} + a_{33} < 0 \Rightarrow r \left[ 1 - \frac{2}{K_1} \tilde{S}_2 \right] \cdot \frac{1}{1 + \alpha_1 \tilde{I}_2} - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2 - \frac{2}{K_3} \tilde{I}_2 + \gamma_1 \tilde{S}_2 - (\beta_2 + 2\eta + \mu - 1) < 0
\]
if
\[
\alpha_1 > \frac{1}{\tilde{I}_2} \left( \frac{K_1 r - 2\tilde{S}_2}{K_1(\beta_1 \tilde{E}_2 + \gamma_1 \tilde{I}_2 + \beta_2 + 2\eta + \mu - 1) - 1} \right),
\]
where \( \tilde{S}_2 < \frac{K_1 \left[ r + 1 - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2 - (\beta_2 + 2\eta + \mu) \right]}{2r} \). (4.25)

From both (4.23) and (2.25), we get at the end that
\[
\frac{1}{\tilde{I}_2} \left( \frac{K_1 r - 2\tilde{S}_2}{K_1(\beta_1 \tilde{E}_2 + \gamma_1 \tilde{I}_2 + \beta_2 + 2\eta + \mu - 1) - 1} \right) < \alpha_1 < \frac{1}{\tilde{I}_2} \left( \frac{K_1 r - 2\tilde{S}_2}{K_1(\beta_1 \tilde{E}_2 + \gamma_1 \tilde{I}_2 + \eta) - 1} \right)
\]
where \( \tilde{S}_2 < \frac{K_1 \left[ r + 1 - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2 - (\beta_2 + 2\eta + \mu) \right]}{2r} \). This completes the proof.

**Remark 4.2.** In the local stability of the co-existing equilibrium point, we assumed that the recognition of the infection is high enough such that \( \epsilon_1 = 1 \). Still, we expect that the individuals’ screening rate is sufficient to keep a stable environment; however, the screening is applied after the symptoms appear, and thus, we can predict that the exposed compartment can continue to infect the susceptible class. In Theorem 4.2, we noticed that the fear of the death of COVID-19 is dependent on the carrying capacity of the isolated individuals (people under quarantine). The fear is under control when the isolation does not exceed half of the carrying capacity of Q and when the individuals noticed that the infected people found successful treatment. The infection rate from the interaction S – I compartments is mainly related to the infection’s transmission capacity and the updated news of fear of being infected with COVID-19. We see that the awareness, and thus without pollution of information related to COVID-19, show an essential role in keeping the co-existing equilibrium point stable. Another essential parameter is the screening rate that reduced the exposed compartment. The fear of being infected by COVID-19 and dying from this infection is related to the daily updated news of numbers of infected people, isolation, and deaths. When the number of the carrying capacity of infected and isolated people decreases, the fear minimizes.

To support the following theorem, we assume that (4.9) holds, while
\[
\lambda_3 > 1 \text{ for } \theta + \eta + \mu < 1 \text{ (existence of the exposed compartment).}
\]

**Theorem 4.3.** Let \( \chi_2 \) be the co-existing equilibrium point of system (3.2) and assume that \( R_{01} = 1 \) and \( R_{02} = 1 \). Moreover, suppose that \( \eta + \mu + \gamma_2 + \mu_1 < 1 \) and \( \beta_2 + \eta + \mu > 1 \). If
\[
\alpha_2 < \frac{1}{D_2} \left( \frac{K_4 - 2\tilde{Q}_2}{K_4(\eta + \mu + \gamma_2 + \mu_1) - 1} \right) \quad \text{for } \tilde{Q}_2 < \frac{K_4 \left( 1 - (\eta + \mu + \gamma_2 + \mu_1) \right)}{2}
\]
\[
\alpha_1 < \frac{1}{\tilde{I}_2} \left( \frac{K_1 r - 2\tilde{S}_2}{K_1(\beta_1 \tilde{E}_2 + \gamma_1 \tilde{I}_2 + \beta_2 + 2\eta + \mu - 1) - 1} \right) \quad \text{for } \tilde{S}_2 < \frac{K_1 \left[ r + 1 - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2 - (\beta_2 + 2\eta + \mu) \right]}{2r}
\]
where
\[
\beta_1 \tilde{E}_2 + \gamma_1 \tilde{I}_2(r - \eta) \quad \text{and} \quad K_1 K_3 \frac{4r \tilde{I}_2}{\gamma_1 \left( (r - \eta - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2) + (1 - \beta_2 - \eta - \mu) \right)}
\]
then the characteristic equation shows some non-negative eigenvalues such that \( |\arg(\lambda^*)| < \frac{\pi}{2} \), where \( \alpha \in (0, 1) \). Thus the equilibrium point \( \chi_2 \) is unstable.

**Proof.** Since \( R_{01} = 1 \), we have \( \Delta_1 = (a_{44} + a_{66})^2 > 0 \). Furthermore,
\[
a_{44} + a_{66} > 0 \Rightarrow \left[ 1 - \frac{2}{K_1} \tilde{Q}_2 \right] \cdot \frac{1}{1 + \alpha_2 \tilde{Q}_2} - (\eta + \mu + \gamma_2 + \mu_1) > 0
\]
if
\[
\alpha_2 < \frac{1}{D_2} \left( \frac{K_4 - 2\tilde{Q}_2}{K_4(\eta + \mu + \gamma_2 + \mu_1) - 1} \right) \quad \text{and} \quad \tilde{Q}_2 < \frac{K_4 \left( 1 - (\eta + \mu + \gamma_2 + \mu_1) \right)}{2}
\]
where \( \eta + \mu + \gamma_2 + \mu_1 < 1 \).

On the other side, since \( R_{02} = 1 \), we obtain \( \Delta_1 = (a_{11} + a_{33})^2 > 0 \) while
\[
a_{11} + a_{33} > 0 \Rightarrow r \left[ 1 - \frac{2}{K_1} \tilde{S}_2 \right] \cdot \frac{1}{1 + \alpha_1 \tilde{S}_2} - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2 - \frac{2}{K_3} \tilde{I}_2 + \gamma_1 \tilde{S}_2 - (\beta_2 + 2\eta + \mu - 1) > 0
\]
if \( \tilde{S}_2 > \frac{2}{K_1 K_3} \tilde{I}_2 \), \( \beta_2 + \eta + \mu > 1 \) and
\[
\alpha_1 < \frac{1}{\tilde{I}_2} \left( \frac{K_1 r - 2\tilde{S}_2}{K_1(\beta_1 \tilde{E}_2 + \gamma_1 \tilde{I}_2 + \beta_2 + 2\eta + \mu - 1) - 1} \right)
\]
where \( \tilde{S}_2 < \frac{K_1 \left[ r + 1 - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2 - (\beta_2 + 2\eta + \mu) \right]}{2r} \). This completes the proof.
Remark 4.3. In Theorem 4.3, we consider the unstable (uncontrollable) case of the co-existing equilibrium point. (4.27) emphasizes the screening tools; this means, if the screening rate is low or insufficient, we expect undetected infectious cases. These infected individuals can continue to transmit the infection to the susceptible class with a rate of $\beta_1$ and $\gamma_1$. The increase of the infected cases (exposed and COVID-19 detected classes) decreases the susceptible density, which leads to a fear of mental health problems of the susceptible class. The trust in screening tests and belief in treatment decreases in this scenario. The basic reproduction numbers are assumed as $R_{01} = 1$ and $R_{02} = 1$. Moreover, we see that the susceptible group focuses mainly on the susceptible and infected class’s carrying capacity. The fear to die from corona, which is given as $\alpha_2$, is based on the density capacity of the isolated compartment and the announcements of unsuccessful treatment alternatives.

**Theorem 4.4.** Let $\chi_2$ be the co-existing equilibrium point of system (3.2). Assume that $\eta + \mu + \gamma_2 + \mu_1 < 1$ and $\beta_2 + 2\eta + \mu > 1$. Let the basic reproduction numbers be

$$R_{01} > 1 + \frac{\left(1 - \frac{2}{K_{4} \tilde{Q}_2} \cdot \frac{1}{1 + \alpha_2 D_2} - (\eta + \mu + \gamma_2 + \mu_1)\right)^2}{4 \mu_1 \left(1 - \frac{2}{K_{4} \tilde{Q}_2} \cdot \frac{1}{1 + \alpha_2 D_2} - (\eta + \mu + \gamma_2)\right)}$$

and

$$1 < R_{02} < 1 + \frac{\left(r\left(1 - \frac{2}{K_{4} \tilde{Q}_2} \cdot \frac{1}{1 + \alpha_2 D_2} - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2 - \eta + 1 - \frac{2}{K_{1}} \tilde{I}_2 + \gamma_1 \tilde{S}_2 - (\beta_2 + \eta + \mu)\right)^2}{4 \left(r\left(1 - \frac{2}{K_{4} \tilde{Q}_2} \cdot \frac{1}{1 + \alpha_2 D_2} - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2 - \eta\right)((\beta_2 + \eta + \mu) - 1 + \frac{2}{K_{1}} \tilde{I}_2 - \gamma_1 \tilde{S}_2)\right)}$$

such that

$$\alpha_2 < \frac{1}{D_2} \left(\frac{K_4 - 2 \tilde{Q}_2}{K_4(\eta + \mu + \gamma_2 + \mu_1)} - 1\right)$$

for $\tilde{Q}_2 < \frac{K_4(1 - (\eta + \mu + \gamma_2 + \mu_1))}{2}$  

(4.33)

$$\alpha_1 < \frac{1}{I_2} \left(\frac{K_1 r - 2 r \tilde{S}_2}{K_1(\beta_1 \tilde{E}_2 + \gamma_1 \tilde{I}_2 + \beta_2 + 2\eta + \mu - 1)} - 1\right)$$

for $\tilde{S}_2 < \frac{2}{K_3 \gamma_1} I_2$  

(4.34)

where

$$\beta_1 \tilde{E}_2 + \gamma_1 \tilde{I}_2 (r - \eta)$$

and $K_1 \gamma_1 \gamma_1 (r - \eta - \tilde{E}_2 - \gamma_1 \tilde{I}_2) + (1 - \beta_2 - \eta - \mu)$.

Then the eigenvalues of (4.11) and (4.12) show complex conjugate, which implies that the $S - I$ and $Q - D$ interaction show an asymptotic stable behavior such that

$$|\arg(\lambda_{1,3})| = \tan^{-1} \left[\frac{\left(4\left(1 - \frac{2}{K_{4} \tilde{Q}_2} \cdot \frac{1}{1 + \alpha_2 D_2} - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2 - \eta + 1 - \frac{2}{K_{1}} \tilde{I}_2 + \gamma_1 \tilde{S}_2 - (\beta_2 + \eta + \mu)\right)^2}{4 \left(1 - \frac{2}{K_{4} \tilde{Q}_2} \cdot \frac{1}{1 + \alpha_2 D_2} - (\eta + \mu + \gamma_2)\right) \mu_1 (R_{01} - 1)} - 1\right] \alpha \pi \frac{2}{2}$$

and

$$|\arg(\lambda_{4,6})| = \tan^{-1} \left[\frac{\left(1 - \frac{2}{K_{4} \tilde{Q}_2} \cdot \frac{1}{1 + \alpha_2 D_2} - (\eta + \mu + \gamma_2)\right) \mu_1 (R_{01} - 1) - 1\right] \alpha \pi \frac{2}{2}$$

Proof. Let us consider the isolated (under quarantine) and death compartment where

$$\Delta_1 = (a_{44} + a_{66})^2 - 4a_{44}a_{66}(1 - R_{01}) < 0.$$

(4.36)

From (4.36), we have

$$R_{01} > 1 + \frac{\left(1 - \frac{2}{K_{4} \tilde{Q}_2} \cdot \frac{1}{1 + \alpha_2 D_2} - (\eta + \mu + \gamma_2 + \mu_1)\right)^2}{4 \mu_1 \left(1 - \frac{2}{K_{4} \tilde{Q}_2} \cdot \frac{1}{1 + \alpha_2 D_2} - (\eta + \mu + \gamma_2)\right)}$$

(4.37)

where

$$\alpha_2 < \frac{1}{D_2} \left(\frac{K_4 - 2 \tilde{Q}_2}{K_4(\eta + \mu + \gamma_2 + \mu_1)} - 1\right)$$

and $\tilde{Q}_2 < \frac{K_4(1 - (\eta + \mu + \gamma_2))}{2}$ for $\eta + \mu + \gamma_2 < 1.$

(4.38)

Furthermore, straightforward computations show that

$$a_{44} + a_{66} > 0 \Rightarrow \left(1 - \frac{2}{K_4 \tilde{Q}_2} \cdot \frac{1}{1 + \alpha_2 D_2} - (\eta + \mu + \gamma_2)\right) - \mu_1 > 0.$$

(4.39)

if

$$\alpha_2 < \frac{1}{D_2} \left(\frac{K_4 - 2 \tilde{Q}_2}{K_4(\eta + \mu + \gamma_2 + \mu_1)} - 1\right)$$

and $\tilde{Q}_2 < \frac{K_4(1 - (\eta + \mu + \gamma_2 + \mu_1))}{2}$ for $\eta + \mu + \gamma_2 + \mu_1 < 1.$

(4.40)

This completes the proof of the $Q - D$ interaction.

Considering now the discriminant of the characteristic equation includes the interaction of the $S - I$ compartments

$$\Delta_2 = (a_{11} + a_{33})^2 - 4a_{11}a_{33}(1 - R_{02}) < 0.$$

(4.41)
we obtain

\[ R_{02} < 1 + \left\{ \frac{\beta_1 \tilde{E}_2 - \gamma_2 \tilde{I}_2 - \eta + 1 - \frac{2}{\kappa_1} \tilde{I}_2 + \gamma_1 \tilde{S}_2 - (\beta_2 + \eta + \mu)}{4 \left( \beta_1 \tilde{E}_2 - \gamma_2 \tilde{I}_2 - \eta \right) \left( (\beta_2 + \eta + \mu) - 1 + \frac{2}{\kappa_1} \tilde{I}_2 - \gamma_1 \tilde{S}_2 \right)} \right\}^2 \]

(4.42)

\[ \tilde{S}_2 \left( \frac{2}{K_3 \gamma_1} \tilde{I}_2 \right) \text{ for } \beta_2 + \eta + \mu \]

(4.43)

and

\[ \alpha_1 < \frac{1}{\tilde{I}_2} \left( \frac{\kappa_1 r - 2\tilde{S}_2}{K_1 (\beta_1 \tilde{E}_2 + \gamma_1 \tilde{I}_2 + \beta_2 + 2\eta + \mu - 1)} - 1 \right) \text{ for } \tilde{S}_2 < \frac{K_1 \left( r - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2 \right)}{2r} \]

(4.44)

Furthermore, we get that

\[ a_{11} + a_{33} > 0 \Rightarrow r \left( 1 - \frac{2}{K_3} \tilde{S}_2 \right) \left( \frac{1}{1 + \alpha_1 \tilde{I}_2} - \beta_1 \tilde{E}_2 - \gamma_2 \tilde{I}_2 - \frac{2}{K_3} \tilde{I}_2 + \gamma_1 \tilde{S}_2 - (\beta_2 + 2\eta + \mu - 1) > 0 \right. \] if

\[ \alpha_1 < \frac{1}{\tilde{I}_2} \left( \frac{\kappa_1 r - 2\tilde{S}_2}{K_1 (\beta_1 \tilde{E}_2 + \gamma_1 \tilde{I}_2 + \beta_2 + 2\eta + \mu - 1)} - 1 \right) \text{ for } \tilde{S}_2 < \frac{2}{K_3 \gamma_1} \tilde{I}_2 < \frac{K_1 \left( r + 1 - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2 - (\beta_2 + 2\eta + \mu) \right)}{2r} \]

(4.45)

where

\[ \beta_1 \tilde{E}_2 + \gamma_1 \tilde{I}_2 \left( r - \eta - K_1 K_3 \right) \gamma_1 \left( \left( r - \eta - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2 \right) + (1 - \beta_2 - \eta - \mu) \right) \]

(4.46)

From (4.44) and (4.45), we obtain at the end that

\[ \alpha_1 < \frac{1}{\tilde{I}_2} \left( \frac{\kappa_1 r - 2\tilde{S}_2}{K_1 (\beta_1 \tilde{E}_2 + \gamma_1 \tilde{I}_2 + \beta_2 + 2\eta + \mu - 1)} - 1 \right) < \frac{1}{\tilde{I}_2} \left( \frac{\kappa_1 r - 2\tilde{S}_2}{K_1 (\beta_1 \tilde{E}_2 + \gamma_1 \tilde{I}_2 + \beta_2 + 2\eta + \mu - 1)} - 1 \right) \]

Remark 4.4. In Theorem 4.4, we proved that the characteristic equations' eigenvalues in (4.11) and (4.12) show complex eigenvalues under specific conditions. In this scenario, the treatment of recovering from COVID-19 is limited or needs a long time for a successful result; however, the death rate from the corona is less. The basic reproduction number of both $R_{01} > 1$ and $R_{02} > 1$. We discovered that the death caused by different symptoms during corona does not affect the susceptible class, and the main concern is the death in total without noticing the difference. The isolated compartment shows an increase, while the interaction of the susceptible and infected group is high. Thus, the fear to get infected $\alpha_1$ exists and is dependent on the carrying capacity of the $S-I$ compartments. While the fear to die from corona, which is $\alpha_2$ shows an upper bound according to the decrease of death from corona, and even though the treatment takes time, hope exists, which keeps the compartments stable.

Theorem 4.5. Let $\chi_2$ be the co-existing equilibrium point of system (3.2). Assume that $\eta + \mu + \gamma_2 + \mu_1 \left( \beta_2 + 2\eta + \mu \right)$. Let the basic reproduction numbers be

\[ R_{01} > 1 + \left\{ \frac{\left( 1 - \frac{2}{\kappa_2} \tilde{Q}_2 \right)}{\kappa_4 (\eta + \mu + \gamma_2 + \mu_1)} - (\eta + \mu + \gamma_2 + \mu) \right\}^2 \]

(4.47)

\[ 1 < R_{02} < 1 + \frac{\left( \frac{\kappa_4 - 2\tilde{Q}_2}{\kappa_2 (\eta + \mu + \gamma_2 + \mu_1)} - 1 \right)}{\kappa_2 (\eta + \mu + \gamma_2 + \mu_1)} \]

(4.48)

such that

\[ \frac{K_4 - 2\tilde{Q}_2}{2\tilde{Q}_2} \left( \frac{K_4 (\eta + \mu + \gamma_2 + \mu_1)}{\kappa_4 (\eta + \mu + \gamma_2 + \mu_1) - 1} \right) < \alpha_2 < \frac{1}{\tilde{I}_2} \left( \frac{K_4 - 2\tilde{Q}_2}{\kappa_2 (\eta + \mu + \gamma_2 + \mu_1) - 1} \right) \]

or \( \tilde{Q}_2 < K_4 \left( 1 - (\eta + \mu + \gamma_2 + \mu_1) \right) \)

(4.49)

\[ \frac{K_1 r - 2\tilde{S}_2}{K_1 (\beta_1 \tilde{E}_2 + \gamma_1 \tilde{I}_2 + \beta_2 + 2\eta + \mu - 1)} - 1 \]

(4.48)

where

\[ \beta_1 \tilde{E}_2 + \gamma_1 \tilde{I}_2 \left( r - \eta \right) \gamma_1 \left( \left( r - \eta - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2 \right) + (1 - \beta_2 - \eta - \mu) \right) \]

(4.49)

Then the $S-I$ and $Q-D$ compartments are asymptotic stable such that

\[ 0 < \alpha < 2 \left\lfloor \frac{2}{\pi} \tan^{-1} \left( \frac{\kappa_2 \tilde{Q}_2}{\kappa_2 (\eta + \mu + \gamma_2 + \mu_1) - 1} \right) \right\rfloor \]

(4.49)
and
\[
0 < \alpha < 2 - \frac{2}{\pi} \tan^{-1}\left(\sqrt{\frac{4\left(1 - \frac{2}{K_4} \hat{Q}_2 \cdot \frac{1}{1 + \alpha_2 D_2} - (\eta + \mu + \gamma_2)\right) \mu_1 (R_{01} - 1)}{\left(1 - \frac{2}{K_4} \hat{Q}_2 \cdot \frac{1}{1 + \alpha_2 D_2} - (\eta + \mu + \gamma_2 + \mu_1)\right)^2 - 1}}\right).
\]

**Proof.** From
\[
\Delta_1 = (a_{44} + a_{66})^2 - 4a_{44}a_{66}(1 - R_{01}) < 0,
\]
we have
\[
R_{01} > 1 + \left(\frac{1 - \frac{2}{K_4} \hat{Q}_2 \cdot \frac{1}{1 + \alpha_2 D_2} - (\eta + \mu + \gamma_2 + \mu_1)}{4 \mu_1 \left(1 - \frac{2}{K_4} \hat{Q}_2 \cdot \frac{1}{1 + \alpha_2 D_2} - (\eta + \mu + \gamma_2)\right)}\right)^2,
\]
where
\[
\alpha_2 < \frac{1}{D_2} \left(\frac{K_4 - 2 \hat{Q}_2}{K_4 (\eta + \mu + \gamma_2 + \mu_1) - 1}\right) \text{ and } \hat{Q}_2 < \frac{K_4 (1 - (\eta + \mu + \gamma_2))}{2} \text{ for } \eta + \mu + \gamma_2 < 1.
\]
Furthermore, we have
\[
a_{44} + a_{66} < 0 \Rightarrow \left(1 - \frac{2}{K_4} \hat{Q}_2 \cdot \frac{1}{1 + \alpha_2 D_2} - (\eta + \mu + \gamma_2) - \mu_1 < 0,
\]
if
\[
\alpha_2 > \frac{1}{D_2} \left(\frac{K_4 - 2 \hat{Q}_2}{K_4 (\eta + \mu + \gamma_2 + \mu_1) - 1}\right) \text{ and } \hat{Q}_2 < \frac{K_4 (1 - (\eta + \mu + \gamma_2 + \mu_1))}{2} \text{ for } \eta + \mu + \gamma_2 + \mu_1 < 1.
\]

From (4.52) and (4.54), we obtain
\[
\frac{1}{D_2} \left(\frac{K_4 - 2 \hat{Q}_2}{K_4 (\eta + \mu + \gamma_2 + \mu_1) - 1}\right) < \alpha_2 < \frac{1}{D_2} \left(\frac{K_4 - 2 \hat{Q}_2}{K_4 (\eta + \mu + \gamma_2) - 1}\right).
\]

Considering now the discriminant of the characteristic equation includes the interaction of the \(S - I\) compartments
\[
\Delta_2 = (a_{11} + a_{33})^2 - 4a_{11}a_{33}(1 - R_{02}) < 0,
\]
we obtain
\[
R_{02} < 1 + \left(\frac{r \left(1 - \frac{2}{K_3} \hat{S}_2 \cdot \frac{1}{1 + \alpha_1 I_2} - \beta_1 \hat{E}_2 - \gamma_1 \hat{I}_2 - \eta + 1 - \frac{2}{K_1} \hat{I}_2 + \gamma_1 \hat{S}_2 - (\beta_2 + \eta + \mu)\right)}{4 \left(1 - \frac{2}{K_3} \hat{S}_2 \cdot \frac{1}{1 + \alpha_1 I_2} - \beta_1 \hat{E}_2 - \gamma_1 \hat{I}_2 - \eta\right) \left((\beta_2 + \eta + \mu) - 1 + \frac{2}{K_1} \hat{I}_2 - \gamma_1 \hat{S}_2\right)}\right)^2,
\]
where \(\hat{S}_2 < \frac{2}{K_1 \gamma_1} \hat{I}_2, \beta_2 + \eta + \mu > 1\) and
\[
\alpha_1 < \frac{1}{I_2} \left(\frac{K_1 r - 2 r \hat{S}_2}{K_1 (\beta_1 E_2 + \gamma_1 I_2 + \eta)} - 1\right) \text{ for } \hat{S}_2 < \frac{K_1 \left(r - \eta - \beta_1 \hat{E}_2 - \gamma_1 \hat{I}_2\right)}{2r}.
\]
Moreover, we get that
\[
a_{11} + a_{33} < 0 \Rightarrow r \left(1 - \frac{2}{K_1} \hat{S}_1 \cdot \frac{1}{1 + \alpha_1 I_2} - \frac{2}{K_3} \hat{I}_2 - \gamma_1 \hat{S}_2 - (\beta_2 + 2 \eta + \mu - 1)\right) < 0
\]
if
\[
\alpha_1 > \frac{1}{I_2} \left(\frac{K_1 r - 2 r \hat{S}_2}{K_1 (\beta_1 E_2 + \gamma_1 I_2 + \beta_2 + 2 \eta + \mu - 1)} - 1\right)
\]
where
\[
\beta_1 \hat{E}_2 + \gamma_1 \hat{I}_2 (r - \eta \text{ and } K_1 K_3) \frac{4r \hat{I}_2}{\gamma_1 \left((r - \eta - \beta_1 \hat{E}_2 - \gamma_1 \hat{I}_2) + (1 - \beta_2 - \eta - \mu)\right)}.
\]

**Remark 4.5.** In Theorem 4.5, we show that both fears, \(\alpha_1\) and \(\alpha_2\), are restricted to a bounded interval depending on the carrying capacity of the isolated and infected compartments that do not exceed almost half of their carrying capacity. Additionally, the transmission from the susceptible compartment to the infected compartment is reduced. The fear effect \(\alpha_2\) recognizes the difference of death from corona and with corona, which keeps the compartments stable. Still, a long-term treatment exists; however, the death rate from the corona is low.

**Theorem 4.6.** Let \(X_2\) be the co-existing equilibrium point of system (3.2). Assume that \(\eta + \mu + \gamma_2 + \mu_1 (1 + \beta_2 + 2 \eta + \mu)1\) and \(K_1 K_3 > \frac{2 \eta K_5 \beta_5}{I_3 \beta_3}\). Let the basic reproduction numbers be \(R_{01} > 1\) and \(R_{02} > 1\).
such that
\[
\frac{1}{D_2} \left( \frac{K_4 - 2 \tilde{Q}_2}{K_4(\eta + \mu + \gamma_2 + \mu_1)} - 1 \right) = \alpha_2 < \frac{1}{D_2} \left( \frac{K_4 - 2 \tilde{Q}_2}{K_4(\eta + \mu + \gamma_2 + \mu_1)} - 1 \right) \quad \text{for } \tilde{Q}_2 < K_4 \left( \frac{1 - (\eta + \mu + \gamma_2 + \mu_1)}{2} \right),
\]  
(4.60)

\[
\frac{1}{I_2} \left( \frac{K_1 r - 2 r \tilde{S}_2}{K_1(\beta_1 E_2 + \gamma_1 I_2 + \beta_2 + 2 \eta + \mu - 1)} - 1 \right) = \alpha_1 < \frac{1}{I_2} \left( \frac{K_1 r - 2 r \tilde{S}_2}{K_1(\beta_1 E_2 + \gamma_1 I_2 + \eta)} - 1 \right) \quad \text{for } \tilde{S}_2 = \frac{2}{K_1 \gamma_1} I_2
\]  
(4.61)

where

\[
\beta_1 \tilde{E}_2 + \gamma_1 \tilde{I}_2 < r - \eta \quad \text{and } K_1 K_3 > \frac{4r \ I_2}{\gamma_1 \left( (r - \eta - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2) + (1 - \beta_2 - \eta - \mu) \right)}.
\]

Then the $S - I$ and $Q - D$ compartments are asymptotic stable such that

\[
\tan^{-1} \left( \frac{4 \left( r - \frac{2}{K_1} \tilde{Q}_2 \cdot \frac{1}{1 + \alpha_2 I_2} - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2 - \eta \right) \left( (\beta_2 + \eta + \mu) - \frac{1}{R_02 - 1} \right)}{r \left( 1 - \frac{2}{K_4} \tilde{Q}_2 \cdot \frac{1}{1 + \alpha_2 I_2} - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2 - \eta \right) + \frac{1}{\alpha_2 I_2} - (\beta_2 + \eta + \mu) \right)^2 - 1} = \frac{\pi}{2}
\]

and

\[
\tan^{-1} \left( \frac{4 \left( \frac{1}{1 + \alpha_2 I_2} \cdot \frac{1}{K_4} \tilde{Q}_2 - (\eta + \mu + \gamma_2) \cdot \frac{1}{\alpha_2 I_2} - (\eta + \mu + \gamma_2 + \mu_1) \right) \mu_1 \left( R_01 - 1 \right)}{\left( 1 - \frac{2}{K_4} \tilde{Q}_2 \cdot \frac{1}{1 + \alpha_2 I_2} - (\eta + \mu + \gamma_2) \right)^2 - 1} = \frac{\pi}{2}
\]

**Proof.** If $R_{01} > 1$, then $\Delta_1 = 4 a_{44} a_{66} (R_{01} - 1) < 0$.

\[
\alpha_2 < \frac{1}{D_2} \left( \frac{K_4 - 2 \tilde{Q}_2}{K_4(\eta + \mu + \gamma_2 + \mu_1)} - 1 \right) \quad \text{and } \tilde{Q}_2 < \frac{K_4 \left( \frac{1 - (\eta + \mu + \gamma_2 + \mu_1)}{2} \right)}{2}
\]  
(4.62)

In addition, we obtain
\[
a_{44} + a_{66} = 0 \Rightarrow \left( 1 - \frac{2}{K_4} \tilde{Q}_2 \cdot \frac{1}{1 + \alpha_2 I_2} - (\eta + \mu + \gamma_2) - \mu_1 = 0.
\]

if

\[
\alpha_2 = \frac{1}{D_2} \left( \frac{K_4 - 2 \tilde{Q}_2}{K_4(\eta + \mu + \gamma_2 + \mu_1)} - 1 \right) \quad \text{and } \tilde{Q}_2 < \frac{K_4 \left( \frac{1 - (\eta + \mu + \gamma_2 + \mu_1)}{2} \right)}{2}
\]  
(4.63)

which means that
\[
\frac{1}{D_2} \left( \frac{K_4 - 2 \tilde{Q}_2}{K_4(\eta + \mu + \gamma_2 + \mu_1)} - 1 \right) = \alpha_2 < \frac{1}{D_2} \left( \frac{K_4 - 2 \tilde{Q}_2}{K_4(\eta + \mu + \gamma_2 + \mu_1)} - 1 \right).
\]

On the other side if $R_{02} > 1$, then $\Delta_2 = 4 a_{11} a_{33} (R_{02} - 1) < 0$, since $\tilde{S}_2 = \frac{2}{K_1 \gamma_1} I_2$ and $\beta_2 + \eta + \mu > 1$. while

\[
\alpha_1 < \frac{1}{I_2} \left( \frac{K_1 r - 2 r \tilde{S}_2}{K_1(\beta_1 E_2 + \gamma_1 I_2 + \eta)} - 1 \right) \quad \text{for } \tilde{S}_2 < \frac{K_1 \left( r - \eta - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2 \right)}{2 r}
\]  
(4.64)

Furthermore, we get that
\[
a_{11} + a_{33} = 0 \Rightarrow r \left( 1 - \frac{2}{K_4} \tilde{S}_2 \cdot \frac{1}{1 + \alpha_1 I_2} - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2 - (\beta_2 + 2 \eta + \mu - 1) = 0,
\]

if

\[
\alpha_1 = \frac{1}{I_2} \left( \frac{K_1 r - 2 r \tilde{S}_2}{K_1(\beta_1 E_2 + \gamma_1 I_2 + \beta_2 + 2 \eta + \mu - 1)} - 1 \right)
\]  
(4.65)

where

\[
\beta_1 \tilde{E}_2 + \gamma_1 \tilde{I}_2 (r - \eta \quad \text{and } K_1 K_3 > \frac{4r \ I_2}{\gamma_1 \left( (r - \eta - \beta_1 \tilde{E}_2 - \gamma_1 \tilde{I}_2) + (1 - \beta_2 - \eta - \mu) \right)}.
\]

This completes the proof.

**Remark 4.6.** Theorem 4.6 shows the reality of high transmission with $R_{01} > 1$ and $R_{02} > 1$. The screening rate exists sufficiently to detect the exposed class; however, the transmission spread continues. Individuals under quarantine perceive death rates mainly related to corona. The uncertain treatment alternatives keep a limited trust and fear in the compartments. On the other side, the fear of the susceptible class, which is given as $\alpha_1$ is split into two forms; the increase of new cases scares the $S$ compartment to be infected from the undetected exposed and or infected classes, and another form of the fear focuses on the isolation strategies.
5. Existence of flip bifurcation

In this part, we want to apply the discretization process to analyze Flip Bifurcation. We modify our model in (2.1) in considering the discrete-time effect. In this part, we assume an awareness of $\varepsilon_1 = 1$. The discretization of the system (2.1) is as follows, where $z = \left[ \frac{t}{h} \right]$:

\[
\begin{align*}
D^\alpha S(t) &= \Lambda + S(z) r \left( 1 - \frac{S(z)}{K_1} \right) \frac{1}{1 + \alpha_1 I(z)} - \beta_1 E(z) S(z) - \gamma_1 I(z) S(z) - \eta S(z) \\
D^\alpha E(t) &= E(z) \left( 1 - \frac{E(z)}{K_2} \right) - (\theta + \eta + \mu) E(z) \\
D^\alpha I(t) &= I(z) \left( 1 - \frac{I(z)}{K_3} \right) + \beta_1 E(z) S(z) + \gamma_1 I(z) S(z) + \theta E(z) - (\beta_2 + \eta + \mu) I(z) \\
D^\alpha Q(t) &= Q(z) \left( 1 - \frac{Q(z)}{K_4} \right) \frac{1}{1 + \alpha_2 D(z)} + \beta_2 I(z) - (\eta + \mu + \gamma_2) Q(z) \\
D^\alpha R(t) &= \gamma_2 Q(z) - \eta R(z) \\
D^\alpha D(t) &= \mu (E(z) + I(z) + Q(z)) - \mu_1 D(z).
\end{align*}
\]

For $t \in [0, h)$, $\frac{t}{h} \in [0, 1)$, we have

\[
\begin{align*}
D^\alpha S(t) &= \Lambda + S_0 r \left( 1 - \frac{S_0}{K_1} \right) \frac{1}{1 + \alpha_1 I_0} - \beta_1 E_0 S_0 - \gamma_1 I_0 S_0 - \eta S_0 \\
D^\alpha E(t) &= E_0 \left( 1 - \frac{E_0}{K_2} \right) - (\theta + \eta + \mu) E_0 \\
D^\alpha I(t) &= I_0 \left( 1 - \frac{I_0}{K_3} \right) + \beta_1 E_0 S_0 + \gamma_1 I_0 S_0 + \theta E_0 - (\beta_2 + \eta + \mu) I_0 \\
D^\alpha Q(t) &= Q_0 \left( 1 - \frac{Q_0}{K_4} \right) \frac{1}{1 + \alpha_2 D_0} + \beta_2 I_0 - (\eta + \mu + \gamma_2) Q_0 \\
D^\alpha R(t) &= \gamma_2 Q_0 - \eta R_0 \\
D^\alpha D(t) &= \mu (E_0 + I_0 + Q_0) - \mu_1 D_0.
\end{align*}
\]

The solution of (5.1) reduces to

\[
\begin{align*}
S_1(t) &= S_0 + \frac{t^\alpha}{\Gamma(\alpha + 1)} \left\{ \Lambda + S_0 r \left( 1 - \frac{S_0}{K_1} \right) \frac{1}{1 + \alpha_1 I_0} - \beta_1 E_0 S_0 - \gamma_1 I_0 S_0 - \eta S_0 \right\} \\
E_1(t) &= E_0 + \frac{t^\alpha}{\Gamma(\alpha + 1)} \left\{ E_0 \left( 1 - \frac{E_0}{K_2} \right) - (\theta + \eta + \mu) E_0 \right\} \\
I_1(t) &= I_0 + \frac{t^\alpha}{\Gamma(\alpha + 1)} \left\{ I_0 \left( 1 - \frac{I_0}{K_3} \right) + \beta_1 E_0 S_0 + \gamma_1 I_0 S_0 + \theta E_0 - (\beta_2 + \eta + \mu) I_0 \right\} \\
Q_1(t) &= Q_0 + \frac{t^\alpha}{\Gamma(\alpha + 1)} \left\{ Q_0 \left( 1 - \frac{Q_0}{K_4} \right) \frac{1}{1 + \alpha_2 D_0} + \beta_2 I_0 - (\eta + \mu + \gamma_2) Q_0 \right\} \\
R_1(t) &= R_0 + \frac{t^\alpha}{\Gamma(\alpha + 1)} \left\{ \gamma_2 Q_0 - \eta R_0 \right\} \\
D_1(t) &= D_0 + \frac{t^\alpha}{\Gamma(\alpha + 1)} \left\{ \mu (E_0 + I_0 + Q_0) - \mu_1 D_0 \right\}
\end{align*}
\]

For $t \in [h, 2h)$, $\frac{t}{h} \in [1, 2)$ we get

\[
\begin{align*}
S_2(t) &= S_1 + \frac{(t-h)^\alpha}{\Gamma(\alpha + 1)} \left\{ \Lambda + S_1 r \left( 1 - \frac{S_1}{K_1} \right) \frac{1}{1 + \alpha_1 I_1} - \beta_1 E_1 S_1 - \gamma_1 I_1 S_1 - \eta S_1 \right\} \\
E_2(t) &= E_1 + \frac{(t-h)^\alpha}{\Gamma(\alpha + 1)} \left\{ E_1 \left( 1 - \frac{E_1}{K_2} \right) - (\theta + \eta + \mu) E_1 \right\} \\
I_2(t) &= I_1 + \frac{(t-h)^\alpha}{\Gamma(\alpha + 1)} \left\{ I_1 \left( 1 - \frac{I_1}{K_3} \right) + \beta_1 E_1 S_1 + \gamma_1 I_1 S_1 + \theta E_1 - (\beta_2 + \eta + \mu) I_1 \right\} \\
Q_2(t) &= Q_1 + \frac{(t-h)^\alpha}{\Gamma(\alpha + 1)} \left\{ Q_1 \left( 1 - \frac{Q_1}{K_4} \right) \frac{1}{1 + \alpha_2 D_1} + \beta_2 I_1 - (\eta + \mu + \gamma_2) Q_1 \right\} \\
R_2(t) &= R_1 + \frac{(t-h)^\alpha}{\Gamma(\alpha + 1)} \left\{ \gamma_2 Q_1 - \eta R_1 \right\} \\
D_2(t) &= D_1 + \frac{(t-h)^\alpha}{\Gamma(\alpha + 1)} \left\{ \mu (E_1 + I_1 + Q_1) - \mu_1 D_1 \right\}
\end{align*}
\]
Repeating the process \( n \) times, we obtain

\[
\begin{align*}
S_{n+1}(t) &= S_n + \frac{(t-n)\alpha}{\Gamma(\alpha + 1)} \left( \Lambda + S_n \frac{\alpha}{\alpha + 1} - \frac{S_n}{\alpha + 1} \right) + \rho S_n \xi - \beta_1 I_n S_n - \gamma_1 I_n S_n - \eta I_n S_n \\
E_{n+1}(t) &= E_n + \frac{(t-n)\alpha}{\Gamma(\alpha + 1)} \left( \frac{1}{\alpha + 1} - \frac{E_n}{\alpha + 1} - (\theta + \eta + \mu) E_n \right) \\
I_{n+1}(t) &= I_n + \frac{(t-n)\alpha}{\Gamma(\alpha + 1)} \left( I_n - \frac{I_n}{\alpha + 1} + \beta_1 E_n S_n + \gamma_1 I_n S_n + \theta E_n - (\beta_2 + \eta + \mu) I_n \right) \\
Q_{n+1}(t) &= Q_n + \frac{(t-n)\alpha}{\Gamma(\alpha + 1)} \left( Q_n - \frac{Q_n}{\alpha + 1} \right) - \beta_2 I_n - (\eta + \mu + \gamma_2) Q_n \\
R_{n+1}(t) &= R_n + \frac{(t-n)\alpha}{\Gamma(\alpha + 1)} \{ \gamma_2 Q_n - \eta R_n \} \\
D_{n+1}(t) &= D_n + \frac{(t-n)\alpha}{\Gamma(\alpha + 1)} \{ \mu (E_n + I_n + Q_n) - \mu I_n \}.
\end{align*}
\]

(5.4)

For \( t \in [n\hbar, (n+1)\hbar) \), while \( t \to (n+1)\hbar \) and \( \alpha \to 1 \), we have

\[
\begin{align*}
S_{n+1}(t) &= S_n + \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \left( \Lambda + S_n \frac{\alpha}{\alpha + 1} - \frac{S_n}{\alpha + 1} \right) + \rho S_n \xi - \beta_1 I_n S_n - \gamma_1 I_n S_n - \eta I_n S_n \\
E_{n+1}(t) &= E_n + \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \left( \frac{1}{\alpha + 1} - \frac{E_n}{\alpha + 1} - (\theta + \eta + \mu) E_n \right) \\
I_{n+1}(t) &= I_n + \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \left( I_n - \frac{I_n}{\alpha + 1} + \beta_1 E_n S_n + \gamma_1 I_n S_n + \theta E_n - (\beta_2 + \eta + \mu) I_n \right) \\
Q_{n+1}(t) &= Q_n + \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \left( Q_n - \frac{Q_n}{\alpha + 1} \right) + \beta_2 I_n - (\eta + \mu + \gamma_2) Q_n \\
R_{n+1}(t) &= R_n + \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \{ \gamma_2 Q_n - \eta R_n \} \\
D_{n+1}(t) &= D_n + \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \{ \mu (E_n + I_n + Q_n) - \mu I_n \}.
\end{align*}
\]

(5.5)

The Jacobian matrix of (5.5) around the co-existing equilibrium point \( \chi_2 = (S^*, E^*, I^*, Q^*, R^*, D^*) \) is given by

\[
J(\chi_2) = 
\begin{pmatrix}
\frac{1}{2} & 0 & 0 & 0 & 0 & 0 \\
0 & \frac{1}{2} & 0 & 0 & 0 & 0 \\
0 & 0 & 1 & 0 & 0 & 0 \\
0 & 0 & 0 & \frac{1}{2} & 0 & 0 \\
0 & 0 & 0 & 0 & 1 & 0 \\
0 & 0 & 0 & 0 & 0 & 1
\end{pmatrix}
\]

(5.6)

\[
b_{11} = 1 + \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \left( \frac{\alpha}{\alpha + 1} - \frac{\alpha}{\alpha + 1} \right), \quad b_{12} = \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \left( \frac{\alpha}{\alpha + 1} \right)
\]

\[
b_{13} = \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \left( -\alpha \frac{\alpha}{\alpha + 1} - \frac{\alpha}{\alpha + 1} \right), \quad b_{22} = 1 + \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \left( \frac{\alpha}{\alpha + 1} - \frac{\alpha}{\alpha + 1} \right), \quad b_{23} = \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \left( \frac{\alpha}{\alpha + 1} \right)
\]

\[
b_{31} = \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \left( \frac{\alpha}{\alpha + 1} \right), \quad b_{32} = \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \left( \frac{\alpha}{\alpha + 1} \right), \quad b_{33} = \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \left( \frac{\alpha}{\alpha + 1} \right)
\]

\[
b_{43} = \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \left( \frac{\alpha}{\alpha + 1} \right), \quad b_{44} = 1 + \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \left( \frac{\alpha}{\alpha + 1} \right), \quad b_{45} = \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \left( \frac{\alpha}{\alpha + 1} \right)
\]

\[
b_{54} = \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \left( \frac{\alpha}{\alpha + 1} \right), \quad b_{55} = 1 - \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \eta, \quad b_{64} = \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \mu, \quad b_{66} = 1 - \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \mu_1.
\]

The characteristic equation of (5.5) is given as

\[
\lambda_2 = 1 + \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \left( \frac{\alpha}{\alpha + 1} \right), \\
\lambda_5 = 1 - \frac{\hbar^\alpha}{\Gamma(\alpha + 1)} \eta.
\]

(5.7)

(5.8)

(5.9)

(5.10)

Theorem 5.1. [38,39] For system (5.11), one of the eigenvalues is \(-1\), and the other eigenvalue lies inside the unit circle if and only if the following statements hold:
Proof. Let \( \chi_2 \) be the co-existing equilibrium point of system (5.5) and assume that \( R_{01}^* = 1 \) and \( R_{02}^* = 1 \). Moreover, suppose that

\[
1 > \theta + \eta + \mu, \quad 1 > \eta + \mu + \gamma_2 > \mu_1 \in \left( \frac{\Gamma(\alpha + 1)}{h^\alpha}, \frac{2\Gamma(\alpha + 1)}{h^\alpha} \right) \text{ and } 1 + \frac{\Gamma(\alpha + 1)}{h^\alpha} < \beta_2 + \eta + \mu < 1 + \frac{2\Gamma(\alpha + 1)}{h^\alpha},
\]

\[
E^* = K_2\{1 - (\theta + \eta + \mu)\} + \frac{2\Gamma(\alpha + 1)}{h^\alpha}
\]

Then the system undergoes a Flip Bifurcation.

\[
1 + \frac{h^\alpha}{\Gamma(\alpha + 1)} \left( 1 - \frac{E^*}{K_2} \right) - (\theta + \eta + \mu) = -1 \Rightarrow E^* = K_2\{1 - (\theta + \eta + \mu)\} + \frac{2\Gamma(\alpha + 1)}{h^\alpha}.
\]

Theorem 5.2. Let \( \chi_2 \) be the co-existing equilibrium point of system (5.5) and assume that \( R_{01}^* = 1 \) and \( R_{02}^* = 1 \). Moreover, suppose that

\[
1 > \theta + \eta + \mu, \quad 1 > \eta + \mu + \gamma_2 > \mu_1 \in \left( \frac{\Gamma(\alpha + 1)}{h^\alpha}, \frac{2\Gamma(\alpha + 1)}{h^\alpha} \right) \text{ and } 1 + \frac{\Gamma(\alpha + 1)}{h^\alpha} < \beta_2 + \eta + \mu < 1 + \frac{2\Gamma(\alpha + 1)}{h^\alpha},
\]

\[
E^* = K_2\{1 - (\theta + \eta + \mu)\} + \frac{2\Gamma(\alpha + 1)}{h^\alpha}
\]

1. \[
\alpha_2 = \frac{1}{D^*} \left( \frac{K_4 - 2Q^*}{K_4(\eta + \mu + \gamma_2 - \frac{\Gamma(\alpha + 1)}{h^\alpha})} - 1 \right)
\]

where

\[
K_1K_3 > \frac{2r^*}{\gamma_1(r + \frac{\Gamma(\alpha + 1)}{h^\alpha} - \beta_1E^* - \gamma_1r^* - \eta)} \quad \text{and} \quad \beta_1E^* + \gamma_1r^* < r - \eta.
\]

Proof. From (5.8), it is evident that \( \lambda_5 < 1 \) while

\[
1 + \frac{h^\alpha}{\Gamma(\alpha + 1)} \left( 1 - \frac{E^*}{K_2} \right) - (\theta + \eta + \mu) = -1 \Rightarrow E^* = K_2\{1 - (\theta + \eta + \mu)\} + \frac{2\Gamma(\alpha + 1)}{h^\alpha}.
\]

Theorem 5.1. (a), if \( R_{01}^* = 1 \) and

\[
1 + \frac{h^\alpha}{\Gamma(\alpha + 1)} \left( 1 - \frac{2Q^*}{K_4} \right) - \frac{1}{1 + \alpha_2D^*} - (\eta + \mu + \gamma_2) < 0,
\]

\[
1 - \frac{h^\alpha}{\Gamma(\alpha + 1)} \mu_1 < 0.
\]

Considering both (5.16) and (5.17), we obtain \( \mu_1 > \frac{\Gamma(\alpha + 1)}{h^\alpha} \) and

\[
\alpha_2 > \frac{1}{D^*} \left( \frac{K_4 - 2Q^*}{K_4(\eta + \mu + \gamma_2 - \frac{\Gamma(\alpha + 1)}{h^\alpha})} - 1 \right)
\]

where

\[
Q^* < \frac{K_4\{1 - (\eta + \mu + \gamma_2) + \frac{\Gamma(\alpha + 1)}{h^\alpha}\}}{2} \quad \text{for} \quad 1 > \eta + \mu + \gamma_2.
\]

Moreover, from Theorem 5.1. (b), we have

\[
3 + \frac{h^\alpha}{\Gamma(\alpha + 1)} \left( 1 - \frac{2Q^*}{K_4} \right) - \frac{1}{1 + \alpha_2D^*} - (\eta + \mu + \gamma_2 + \mu_1) = 0
\]

Thus, from (5.16) and (5.17), we get

\[
Q^* < \frac{K_4\{1 - (\eta + \mu + \gamma_2) + \frac{\Gamma(\alpha + 1)}{h^\alpha}\}}{2} \quad \text{for} \quad 1 > \eta + \mu + \gamma_2 + \mu_1.
\]

Thus, from (5.18) and (5.19), we get

\[
\alpha_2 > \frac{1}{D^*} \left( \frac{K_4 - 2Q^*}{K_4(\eta + \mu + \gamma_2 - \frac{\Gamma(\alpha + 1)}{h^\alpha})} - 1 \right)
\]

for \( \mu_1 < \frac{2\Gamma(\alpha + 1)}{h^\alpha} \) and \( Q^* < \frac{K_4\{1 - (\eta + \mu + \gamma_2) + \frac{\Gamma(\alpha + 1)}{h^\alpha}\}}{2} \). On the other side, since \( R_{01}^* = 1 \), (c) and (d) hold.
Let us now consider the characteristic equation of (5.10). Since \( R_0^{*} = 1 \) the conditions of Theorem 5.1 (c) and (d) hold directly. Furthermore, (a) holds if
\[
1 + \frac{h^p}{\Gamma(\alpha + 1)} \left\{ \frac{1}{1 + \alpha I^r} - \frac{1}{1 + \alpha I^r} - \beta_I^e - \gamma I^r - \eta \right\} < 0
\]
(5.20)
and
\[
1 + \frac{h^p}{\Gamma(\alpha + 1)} \left\{ 1 - \frac{2I^r}{K_1} + \gamma S^r - (\beta_2 + \eta + \mu) \right\} < 0.
\]
(5.21)

From (5.20), we have
\[
\alpha_1 > \frac{1}{\Gamma} \left\{ \frac{rK_1 - 2rS}{K_1(\beta_1 E^e + \gamma I^r + \eta - \frac{\Gamma(\alpha + 1)}{h^p})} - 1 \right\} \text{ for } S^r \left( \frac{K_1(r + \frac{\Gamma(\alpha + 1)}{h^p} - \beta_I^e - \gamma I^r - \eta)}{2r} \right) \text{ and } r - \eta \beta_I^e + \gamma I^r.
\]
(5.22)

Besides, we have \( S^r = \frac{2r}{\gamma K_3} \text{ and } \beta_2 + \eta + \mu > 1 + \frac{\Gamma(\alpha + 1)}{h^p} \) from (5.21). Moreover, we obtain the condition of (b);
\[
3 + \frac{h^p}{\Gamma(\alpha + 1)} \left\{ \frac{r}{K_1(\beta_2 + 2\eta + \mu + 1) + \beta_I^e + \gamma I^r + 1 - (\beta_2 + 2\eta + \mu)} - 1 \right\} = 0
\]
if
\[
\alpha_1 = \frac{1}{\Gamma} \left\{ \frac{r(K_1 - 2S)}{K_1(\beta_2 + 2\eta + \mu + 1) + \beta_I^e + \gamma I^r + \frac{3\Gamma(\alpha + 1)}{h^p}} - 1 \right\}.
\]
(5.23)

where \( S^r < \frac{K_1(r + \frac{\Gamma(\alpha + 1)}{h^p} - \beta_I^e - \gamma I^r)}{2r} \). One can find that
\[
\frac{2I^r}{\gamma K_3} < S^r \frac{K_1(r + \frac{\Gamma(\alpha + 1)}{h^p} - \beta_I^e - \gamma I^r - \eta)}{2r} < K_1 \frac{K_1(r - \eta + \frac{3\Gamma(\alpha + 1)}{h^p} - \beta_I^e - \gamma I^r)}{2r}
\]

Fig. 1. (a) Dynamical spread of system (3.2) (b) Stability of the susceptible and exposed compartment (c) Stability of the infected and quarantine individuals (d) Dynamical spread of the recovered and death compartment.
if \( K_1K_3 > \frac{2\beta^*}{\gamma_1(r + \frac{1}{\gamma_1}) - \beta_1^* - \gamma_1^* - \eta} \), where \( \beta_1^* + \gamma_1^* < r - \eta \). This completes the proof.

**Remark 5.1.** We used the discretization process to establish a difference equation system of order one. The flip bifurcation showed that the infected group’s death rate died from other symptoms activated by COVID-19 is one of the sensitive parameters, which means that the media or health institutions should emphasize the difference to control the fear effect of individuals. In all studies, we could see that the carrying capacity of the sensitive compartment and the compartment of people under quarantine determine humans’ psychological change. Any decrease of the susceptible class shows mental sickness to be one of the infected individuals, while the increase of the quarantine compartment because of high infection or decrease because of total death rates leads to human fear about the death from COVID-19. The density of the exposed class changes related to applying successful tools to detect the virus. The basic reproduction numbers are considered as \( R_{01}^* = 1 \) and \( R_{02}^* = 1 \) to analyze the conditions of Flip bifurcation.

Since the reproduction numbers nowadays are incredibly high, which does not represent a flip bifurcation, we need to think between the difference of “recognition-management” and “control-fear,” which is the major problem we humans are facing for more than one year.

6. Some numerical simulations by using MATLAB 2019

Finally, in this section, we want to illustrate the fear effect of \( \alpha_1 \) and \( \alpha_2 \) in using MATLAB 2019. On the other side, we will consider the effect of the parameters such as screening, recovering from the infection, and the death rates of infected people who die from different symptoms. Here we want to emphasize the psychological pressure on humans named from different organizations as controlling the spread. We determine the values of the parameters and the initial conditions of system (3.2), such as

\[
\begin{align*}
\Lambda &= 12000, \quad \beta_1 = 0.02, \quad \beta_2 = 0.3, \quad \gamma_1 = 0.0001, \quad \gamma_2 = 0.08, \\
\varepsilon_1 &= 1, \quad \theta = 0.3, \quad \mu = 0.005, \quad \eta = 0.0012, \quad \mu_1 = 0.002, \\
K_1 &= 10000, \quad K_2 = 500, \quad K_3 = 200, \quad K_4 = 180, \quad \text{where the fear effect is chosen} \quad \alpha_1 = \alpha_2 = 0 \quad \text{for the initial conditions} \quad S(0) = 2000, \quad E(0) = 80, \quad I(0) = 40, \quad Q(0) = 30, \quad R(0) = 10, \quad D(0) = 2.
\end{align*}
\]

Fig. 1(a)-(b)-(d) show a case of successful treatment alternatives. The density of compartment \( R(t) \) increases, followed by a stable form of the death compartment. The existence of the infection affected the mental health of the susceptible class first; however, after recognizing the treatments and the screening tools, the susceptible class shows a stable equilibrium point. In Fig. 1(c) we can see that the screening period is still not sufficient so that the spread of the infection continuous, while on the other side supplements to hospitalize people under quarantine doesn’t show the desired carrying capacity.
Fig. 2 shows the significant increase of the exposed and infected compartment when the awareness is decreased to %60. Thus, to recognize the spread effect of the virus is the major point in this pandemic phenomena. Fig. 2(a)-(d) show that the exposed and infected compartments will continue the epidemiological spread for a long time if people are not aware of the serious case and protect themselves from the virus. This is a critical result that shows the dominant parameter $\varepsilon_1$ to understand the circumstances of the environment that face a pandemic phenomenon.

In Fig. 3(a)-(d), we reduced the screening rate $\theta = 0.1$ considering the discrepancy information related to the PCR test. We assume in addition that confusing information spread by media and long-term lock-down periods lead to a fear of the compartments with $\alpha_1 = 0.3$ and $\alpha_2 = 0.3$. The figure shows a psychological breakdown of the susceptible class, which is restricted to a specific interval of time until the awareness $\varepsilon_1 = 1$ started to control the compartment.

Fig. 4(a)-(d) is applied to the same scenario when the community shows only %60 of awareness—both the exposed and the infected compartment increases. The fear is related to the screening rate and the treatment process; however, recognizing permanent health care regulations is applied in discrete time or ignored from the community.

7. Conclusion

In this study, we gave at first a brief information about the novel coronavirus COVID-19. Our main objective was to investigate the fear effect of the community spread through the media, social networks and the health organization. The psychological effect such as anxiety, fear, or worry showed a close relation to the epidemiological spread. Unrealistic information about the virus, the spread, mutation, and the vaccines lead people to suffer from uncertainty and activate a fear of the present and future. We formulated a fractional order system of a SEIR+cD model to analyze the balance between controlling the people with various mechanisms and the community’s fear during the lock-down period. The model showed two types of fear; the fear to get infected and the death from corona. The compartment of death was an essential part in the model, since from the statistical records it is still a discussion to distinguish between “death from corona” and “death with corona”.

From our theoretical and numerical findings we conclude that permanent health care regulations are essential to stabilize the community’s recognition of the virus COVID-19. However, as necessary as the awareness, we emphasized improvements of the screening tools and treatment processes. Information from different sources causes fears about the virus and the consequences.
pandemic phenomena should not be seen as a trend of creating unrealistic stories, which affects human mental health during this challenging event (Table 1).

### 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.

### 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. **Adem Kalinli:** Investigation, Visualization, Writing – original draft, Supervision. **Qasem Al Mdallal:** Investigation, Visualization, Writing – original draft, Supervision.

---

**Table 1**

| Notation | Description of Parameter |
|----------|--------------------------|
| $\alpha_1$ | The fear effect of the susceptible class to be infected by COVID-19 |
| $\alpha_2$ | The fear effect of individuals under quarantine to die from COVID-19 |
| $\beta_1$ | Infection rate from the $S$ – $E$ interaction |
| $\gamma_1$ | Infection rate from the $S$ – $I$ interaction |
| $\epsilon_1$ | Recognition of infection |
| $\Theta$ | Rate of screening |
| $\beta_2$ | The rate of infected people being isolated |
| $\gamma_2$ | The rate of recovering from the infection |
| $K_s$ | Carrying capacity of the susceptible class |
| $K_e$ | Carrying capacity of the exposed class |
| $K_i$ | Carrying capacity of the infected class of COVID-19 |
| $K_a$ | Carrying capacity of individuals under quarantine |
| $\Lambda$ | The rate of offspring per year |
| $\mu_1$ | The death rate of COVID-19 infected |
| $\mu_2$ | The death rate of the infected group died from different symptoms that was activated by the virus COVID-19 |
| $\eta$ | The natural death rate |
References

[1] Fehr AR, Perlman S. Coronaviruses: an overview of their replication and pathogenesis. Methods Mol Biol 2015;1282:1–12.
[2] Wu PC, Lai SK, Huang Y, Yuen KY. Coronavirus diversity, phylogeny, and interspecies jumping. Exp Biol Med (Maywood) 2009;234:1117–27.
[3] Su S, et al. Epidemiology, genetic recombination, and pathogenesis of coronaviruses. Trends Microbiol 2016;24:490–502.
[4] Foroni D, Caglioni R, Clerici M, Sirinou M. Molecular evolution of human coronavirus genomes. Trends Microbiol 2017;25:35–48.
[5] Al-Asaad N, Rong L, Alsalaw S, Shillor M. Mathematical model and simulations of MERS outbreak: predictions and implications for control measures. Biomath 2017;5(2):1–21.
[6] Masood N, Malik SS, Raja MN, Mubarak S, Yu C. Unraveling the epidemiology, geographical distribution, and genomic evolution of potentially lethal coronaviruses (SARS, MERS, and SARS-CoV-2). Front Cellular Infect Microbiol 2020;10:499;1–8.
[7] Krishnamoorthy S, Swain B, Verma RS, Gunthe SS. SARS-CoV, MERS-CoV, and 2019-nCoV viruses: an overview of origin, evolution, and genetic variations. Virus Dis 2020;31:411–23.
[8] Rawazir A, Yenugachati N, Dazir OB, Jadi H. Epidemiological trends, characteristics, and distribution of COVID-19: lessons from SARS and MERS outbreaks and way forward. J Infect Dis Epidemiol 2020;6(127):1–6.
[9] Wu F, Su Z, Yu B, Chen Y-M, Wang W, et al. A new coronavirus associated with human respiratory disease in China. Nature 2020;579:265–9.
[10] World Health Organization. Novel Coronavirus (2019-nCoV) Situation Reports, https://www.who.int/emergencies/diseases/novel-coronavirus-2019/situation-reports.
[11] The Economist. Suddenly hope, Nov 12th Ed., 2020, https://www.economist.com/weeklyedition/2020-11-14.
[12] Chao W, Fang Z, Hou G, Han M, Xu X, et al. Economic and social consequences of human mobility restrictions under COVID-19. Proc Natl Acad Sci 2020;117(27):15530–5.
[13] Samal SK. Population dynamics with multiple Allee effects induced by fear factors – A mathematical study on prey-predator interactions. Appl Math Modell 2018;64:1–14.
[14] Guisepppe MD, Zilcha-mano S, Prout TA, Perry JC, Orru G, et al. Psychological impact of coronavirus disease 2019 among Italians during the first week of lockdown. Front Psychiatry 2020;11(576597):1–9.
[15] Luo M, Guo L, Yu M, Jiang W, Wang H. The psychological and mental impact of coronavirus disease 2019 (COVID-19) on medical staff and general public – A systematic review and meta-analysis. Psychiatry Res 2020;291(111390):1–10.
[16] Wang Y, Ma S, Yang C, Cai Z, Hu S, et al. Acute psychological effects of Coronavirus Disease 2019 outbreak among healthcare workers in China: a cross-sectional study. Transl Psychiatry 2020;10(348):1–10.
[17] Umm Min Allah N, Arshad S, Mahmood H, Abbas H. The psychological impact of coronavirus outbreak in Pakistan. Asia-Pacif Psychiay 2020;12(4):1.
[18] Maji C. Impact of media-induced fear on the control of COVID-19 outbreak: a mathematical study. Int J Differ Eq 2021 Article ID 2129490, 1–11, 2021.
[19] Johnston MD, Pell B. A dynamical framework for modeling fear of infection and social distancing in COVID-19 spread. Math Biosci Eng 2020;17(5):7892–915.
[20] Mpeshe SC, Nyerere N. Modeling the dynamics of coronavirus disease pandemic coupled with fear epidemics. Comput Math Methods Med 2021;1–9 Article ID 66474252021.
[21] Liu X, Xiao D. Complex dynamic behaviors of a discrete-time predator-prey system. Chaos Solutions Fractals 2007;32:80–94.
[22] Bozkurt F, Yousef A, Abdeljawad T. Analysis of the outbreak of the novel coronavirus Covid-19 dynamic model with control mechanisms. Results Phys 2020;10:103586;1–10.
[23] Huang Y, Zhu Z, Li Z. Modeling the Allee effect and fear effect in predator-prey system incorporating a prey refuge. Adv Differ Eq 2020;321:1–13.
[24] Johnson MD, Pell B. A dynamical framework for modeling fear of infection and frustration with social distancing in Covid-19 spread. Math Biosci Eng 2020;17(6):7892–915.
[25] Yousef A, Bozkurt F. Bifurcation and stability analysis of a system of fractional-order differential equations for a plant-herbivore model with Allee Effect. Mathematics 2019;7(5):1–18.
[26] Mandal M, Jana S, Nandi SK. Modelling and control of the fractional-order epidemic model with fear effect. Energy Ecol Environ 2020;5(6):421–32.
[27] Ozdemir N, Ucar E. Investigating of an immune system-cancer mathematical model with Mittag-Leffler kernel. AIMS Press 2020;5(2):1519–31.
[28] Ahmad WM, Sprott JC. Chaos in fractional order autonomous nonlinear systems. Chaos Solutions Fractals 2003;16:339–51.
[29] Yousef A, Bozkurt F, Abdeljawad T. Qualitative analysis of a fractional pandemic spread model of the novel coronavirus (Covid-19). Comput Mater Continua 2021;66(1):1–27.
[30] Li L, Liu JG. A generalized definition of Caputo derivatives and its application to fractional ODEs. SIAM J Math Anal 2016;50(3):2867–900.
[31] Kilbas AA, Srivastava HM, Trujillo JJ. Theory and applications of fractional differential equations, 204. Elsevier, 2006. 1st EditionAbdeljawad, T., Baleanu, D., On the fractional derivatives with generalized Mittag-Leffler kernels, Adv Differ Eqs 2018, 468, 2018.
[32] Odibat ZM, Shawagfeh NT. Generalized Taylor’s formula. Appl Math Comput 2007;186:286–93.
[33] Ozalp N, Demirsoy A. A fractional-order SEIR model with vertical transmission. Math Comput Modell 2011;54:1–6.
[34] Qwuusu-Mensah I, Kinyemi L, Oduro B, Hlyoa OS. Fractional-order approach to modeling and simulations of the novel COVID-19. Adv Differ Eq 2020:683:1–21.
[35] Matignon D. Stability results for fractional-order differential equations with applications to control processing. Comput Eng Syst Appl 1996;2:1–6.
[36] Qian D, Li C, Agarwal RP, Wang PY. Stability analysis of fractional differential system with Riemann-Liouville derivatives. Math Comput Model 2010;52:862–74.
[37] Zeng QS, Cao GY, Zhu XJ. The asymptotic stability on sequential fractional-order systems. J Shanghai Jiaotong Univ 2005;39:346–8.
[38] Li X, Mao C, Niu W, Wang D. Stability analysis for discrete biological models using algebraic methods. Math Comput Sci 2011;5:247–62.
[39] Elaydi S. An introduction to difference equations. New York, NY: Springer; 2005.