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Abstract  To curtail and control the pandemic coronavirus (Covid-19) epidemic, there is an urgent need to understand the transmissibility of the infection. Mathematical model is an important tool to describe the transmission dynamics of any disease. In this research paper, we present a mathematical model consisting of a system of nonlinear fractional order differential equations, in which bats were considered as the origin of the virus that spread the disease into human population. We proved the existence and uniqueness of the solution of the model by applying Banach contraction mapping principle. The equilibrium solutions (disease free & endemic) of the model were found to be locally asymptotically stable. The key parameter (Basic reproduction number) describing the number of secondary infections was obtained. Furthermore, global stability analysis of the solutions was carried out using Lyapunov candidate function. We performed numerical simulation, which shows the changes that occur at every time instant due to the variation of $\alpha$. From the graphs, we can see that FODEs have rich dynamics and are better descriptors of biological systems than traditional integer–order models.

1. Introduction

The zoonotic Coronaviruses (which were likely originated from bats) were identified in the mid-1960 and are known to infect human and variety of animals including birds and mammals. Since 2002, two corona viruses infecting animals evolved and caused outbreak in humans: severe acute respiratory syndrome (SARS-COV) and middle-east respiratory syndrome (MERS-COV) [1].

In December 2019, a novel coronavirus which is genetically related to SARS-COV was first isolated from three patients with pneumonia, connected to cluster acute respiratory illness cases from Wuhan, China [1]. Since then, the outbreak spread and become global pandemic, as at the time of this write up the outbreak spread across 150 countries, infected over 700,000 individuals and claimed the lives of over 30,000.

The coronavirus (COVID-19) spread from an infected to healthy person through eye, nose and mouth, via droplets produced on coughing or sneezing. Contact with contaminated surfaces, objects, or items of personal use [2]. The common signs of infection include respiratory symptoms, fever, cough, and shortness of breath and breathing difficulties. In more sev...
ere cases, infection can cause pneumonia, SARS, kidney failure and even death [3].

The time between catching the virus and showing the symptoms is between 2 and 14 days, while the mortality rate ranges between 2 and 3%. It is significantly less than 2003 SARS (10%) or MERS (35%). Almost 80% of people with mild symptoms recover from the disease in two weeks following timely medical care [2].

There is not yet treatment and vaccine against the infection, but the standard recommendations to prevent infection spread at individual level, is the compliance with an orientation strategies that include reporting of suspected case, self-isolation, regular hand washing using sanitizers, covering mouth and nose when coughing and sneezing, and avoiding contact with any one showing symptoms of respiratory illness such as coughing and sneezing [3].

However, to curtail the spread of the infection many governments undertake the following policies: widely public orientation on distancing from public gathering that include social and religious, banning both the local and international air trip, closing both public and private institutions that may attract large gathering, contact tracing and isolation of infected individuals, providing sanitizers at public domains like markets and car park, fumigating the area where an infected individuals comes from, and to the large extent imposing stay at home curfew.

In order to have an effective control of the spread of the infection, we need to know how many people on average does an individual infect (reproduction number), do the measures taken results significant impacts? What is the duration from time onset of the disease to infection (incubation period)? Meanwhile, there is urgent need to develop a mathematical model to estimate the transmissibility and dynamics of the infection.

M. Tahir et al. [4] developed mathematical model (for MERS) in form of nonlinear system of differential equations, in which he considered a camel to be the source of infection that spread the virus to infective human population, then human to human transmission, then to clinic center then to care center. However, they constructed the Lyapunov candidate function to investigate the local and global stability analysis of the equilibrium solutions and subsequently obtained the basic reproduction number or roughly, a key parameter describing transmission of the infection.

T. M Chen et al. [5] developed a Bats-Hosts-Reservoir-People (BHRP) transmission network model for the potential transmission from the infection source (probably bats) to the human infection, which focus on calculating $R_0$.

Q. lin et al. [6] modeled (based on SEIR) the outbreak in Wuhan with individual reaction and governmental action (holiday extension, city lockdown, hospitalization and quarantine) in which they estimated the preliminary magnitude of different effect of individual reaction and governmental action.

Nowadays, many mathematicians use fractional derivative to model epidemic diseases [14–19]. This is because the precision of fractional order supersede the integer order due to its changes at every instant of time and nonlocal behavior, since the derivative is obtained as a result of evaluating integral over the region, while the value of integer order derivative evaluated at a point depend only on that point.

To mimic the ongoing outbreak, we modified the model of M. Tahir et al. in which we incorporated the susceptible human and bat population. We also used fractional derivative in the Caputo sense due to the fact that fractional order derivative gives better result than the integer order.

Numerous fractional derivative operators were developed, but the Riemann-Liouville and the Caputo fractional derivative operators are the most widely used due to their similarities and simplicity to handle. Other derivatives are Atangana-Baleanu, Caputo-Fabrizio, Katugampola, Hadamard e.t.c. [7].

The paper is arranged as follows: chapter one gives introduction, chapter two gives important definitions and preliminaries, chapter three gives model formulation, chapter four gives existence and uniqueness of solution of the model, chapter five gives local and global stability analyses and the derivation of basic reproduction ratio, and finally chapter six gives the numerical simulation result to support the analytic result together with discussion and conclusion.

2. Preliminary definitions and theorems

Definition 1 [8]: A gamma function of $p > 0$ is defined as,

$$\Gamma(p) = \int_0^\infty x^{p-1}e^{-x}dx.$$

Definition 2 [8]: The Riemann-Liouville fractional derivative of order $\alpha \in [n-1, n)$ of $f(x)$ is defined as,

$$aRLD_\alpha^xf(x) = \frac{1}{\Gamma(n-\alpha)} \frac{d^n}{dx^n} \int_a^x (x-t)^{n-\alpha-1}f(t)dt, n = [\alpha] + 1.$$

Definition 3 [8]: The Caputo fractional derivative of order $\alpha \in (n-1, n]$ of $f(x)$ is defined as

$$aXD_\alpha^xf(x) = \frac{1}{\Gamma(n-\alpha)} \int_a^x (x-t)^{n-\alpha-1}f(t)dt, n = [\alpha] + 1.$$

Definition 4 [9]: (Linearity of fractional derivative)

Let $f, g$ be continuous and $b, c$ be scalars, then

$$aRLD_\alpha^n[bf(x) + dg(x)] = b \ aRLD_\alpha^n f(x) + d \ aRLD_\alpha^n g(x);$$

$$aCD_\alpha^n[bf(x) + dg(x)] = b \ aCD_\alpha^n f(x) + d \ aCD_\alpha^n g(x).$$

Definition 5 [10]: (Contraction)

An operator $f: X \rightarrow X$ that maps a metric space onto itself is said to be contractive if for $0 < q < 1$,

$$d(f(x), f(y)) = q d(x, y), \forall x, y \in X.$$

Theorem 1 [10]: (Picard-Banach fixed point or Banach contraction mapping principle)

Any contractive operator that maps a metric space onto itself has a unique fixed point. Furthermore, if $f: X \rightarrow X$ is a contractive operator that maps a metric space onto itself and $a$ is its fixed point: $f(a) = a$; then for any iterative sequence,

$$x_0, x_1 = f(x_0), x_2 = f(x_1), \cdots, x_{n+1} = f(x_n), \cdots,$$

converges to $a$.  

In other words \( \alpha \) is a solution or an equilibrium for continuous dynamical system and fixed point for discrete dynamical system.

**Theorem 2** [11]: The equilibriums solutions \( x_0 \) of the system (\( * \)) is locally asymptotically stable if all the eigenvalues \( \lambda_i \) of the Jacobian matrix \( \frac{\partial \mathbf{f}}{\partial x} \) evaluated at the equilibrium points satisfy

\[
|\arg(\lambda_i)| > \frac{\pi}{2}, 0 < \epsilon < 1.
\]

**Theorem 3** [12]: Let \( x = 0 \) be an equilibrium of system (\( * \)), let \( \Omega \subseteq \mathbb{R}^n \) be a domain containing \( x = 0 \).

Let \( V(t, x) : [0, \infty) \times \Omega \rightarrow \mathbb{R} \) be continuously differentiable function such that,

\[
i - W_i(x) \leq V(t, x) \leq W_j(x),
\]

\[
i - \frac{\partial^\alpha V(t, x)}{\partial t^\alpha} \leq -W_j(x), \text{for} t \geq 0, x \in \Omega
\]

Where \( W_i(x), W_j(x) \) are continuous positive definite function on \( \Omega \) and \( V \) is a Lyapunov candidate function, then \( x = 0 \) is globally asymptotically stable.

**Theorem 4** [13]: Let \( x(t) \in \mathbb{R}^n \) be continuous and derivable function. Then, for any time instant \( t \geq t_0 \) and \( x(t) \in \mathbb{R}^n \),

\[
0\frac{\partial^\alpha V(t, x)}{\partial t^\alpha} \leq \frac{x(t) - x^*}{x^*} - x^* \ln \left( \frac{x(t)}{x^*} \right) - \frac{x^*}{x^*} \frac{\partial^\alpha V(t, x)}{\partial t^\alpha},
\]

\( x^* \in \mathbb{R}^n \).

3. Model formulation

With bats as the origin of the novel Covid-19 virus, it is assumed that the new born of bats are born into susceptible class \( S_b \) at the rate \( \lambda_b \). Which joined the infectious class at rate \( \beta_3 \). It is also assumed that the new born of humans are born into susceptible class \( S_h \) which later became infectious \( I_h \) as a result of contact with an infected bats at the rate \( \beta_2 \). Then the virus spreads from an infected human to human \( H_h \) to a family member \( F_m \), then to clinic center \( P_c \) and care center \( C_c \) at the rates \( \beta_3, \beta_4, \beta_5 \) and \( \beta_6 \) respectively. Fig. 1 gives the schematic diagram of the dynamics of the disease. Table 1 and table 2 give the descriptions of variables and parameters respectively.

The transmission dynamics can be described by the nonlinear system of fractional order differential equations (FODE) below;

\[
0\varpi \frac{\partial^\alpha S_b(t)}{\partial t^\alpha} = \lambda_b S_b(t) - \mu_3 S_b(t) - \beta_3 S_b(t) I_h(t),
\]

\[
0\varpi \frac{\partial^\alpha I_h(t)}{\partial t^\alpha} = \beta_3 S_b(t) I_h(t) - (\mu_4 + \delta_2) I_h(t) - \beta_4 I_h(t) H_h(t),
\]

\[
0\varpi \frac{\partial^\alpha H_h(t)}{\partial t^\alpha} = \beta_4 I_h(t) H_h(t) - (\mu_5 + \delta_3) H_h(t) - \beta_5 H_h(t) F_m(t),
\]

\[
0\varpi \frac{\partial^\alpha F_m(t)}{\partial t^\alpha} = \beta_5 H_h(t) F_m(t) - (\mu_6 + \delta_4) F_m(t) - \beta_6 F_m(t) P_c(t),
\]

\[
0\varpi \frac{\partial^\alpha P_c(t)}{\partial t^\alpha} = \beta_6 F_m(t) P_c(t) - (\mu_7 + \delta_5) P_c(t) - \beta_7 P_c(t) C_c(t),
\]

\[
0\varpi \frac{\partial^\alpha C_c(t)}{\partial t^\alpha} = \beta_7 P_c(t) C_c(t) - \mu_8 C_c(t).
\]

**Table 1** Model variables and their descriptions.

| Model Variables | Descriptions |
|-----------------|--------------|
| \( S_b = S_b(t) \) | Susceptible bats population |
| \( I_b = I_b(t) \) | Infected bats population |
| \( S_h = S_h(t) \) | Susceptible human population |
| \( I_h = I_h(t) \) | Infected human population |
| \( H_h = H_h(t) \) | Human to human transmission population |
| \( F_m = F_m(t) \) | Infected individual to family members population |
| \( P_c = P_c(t) \) | Patient to clinic center transmission population |
| \( C_c = C_c(t) \) | Patient to care center transmission population |
| \( Z = Z(t) \) | Total model population |

**Table 2** Model parameters and their descriptions.

| Model Parameters | Description |
|------------------|-------------|
| \( \mu_i, i = 1, 2, \ldots, 8 \) | Natural death rates in 5 compartments |
| \( S_0, S_1, S_2, I_0, I_1, H_0, F_m, P_c, and C_c \) | compartments |
| \( \delta_i \) | Disease induced death in \( I_0 \) |
| \( \delta_i, i = 2, 3, \ldots, 5 \) | Disease induced death in \( S_0, I_0, H_0, F_m, P_c, and C_c \) compartments |
| \( \lambda_b \) | Birth rate of bats |
| \( \beta_3 \) | Birth rate of human |
| \( \beta_4 \) | Transmission rates |

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**Fig. 1** Schematic diagram showing the dynamics of COVID-19 pandemic.
4. Existence and uniqueness of the solutions

**Theorem 6:** The kernels of equation (1) – (8) satisfy Lipschitz continuity for $L_t \geq 0, i = 1, 2, \ldots, 8$.

**Proof:** Let the kernel kernels be

\[ f_i(t, S_0) = \lambda^0_i - \mu^0_i S_0 - \beta^0_i S_0 I_0, \]

\[ f_2(t, I_0) = \beta^1_0 S_0 I_0 - (\mu^1 + \delta^1_i) I_0 - \beta^1_0 S_0 I_0, \]

\[ f_3(t, S_0) = \lambda^0_i - \mu^0_i S_0 - \beta^0_i S_0 I_0, \]

\[ f_4(t, I_0) = \beta^1_0 S_0 I_0 - (\mu^1 + \delta^1_i) I_0 - \beta^1_0 S_0 I_0, \]

\[ f_5(t, H_0) = \beta^2_0 H_0 I_0 - (\mu^2 + \delta^2_i) I_0 - \beta^2_0 H_0 F_0, \]

\[ f_6(t, F_0) = \beta^3_0 H_0 I_0 - (\mu^3 + \delta^3_i) F_0 - \beta^3_0 P_0 C_0, \]

\[ f_7(t, C_0) = \beta^4_0 P_0 C_0 - (\mu^3 + \delta^3_i) C_0. \]

Now,

\[ |f_i(t, S_0) - f_i(t, S'_0)| = |(\mu^0_i + \beta^0_i I_0)(S_0 - S'_0)| \]

\[ \leq (|\mu^0_i| + |\beta^0_i I_0|)||S_0 - S'_0|| \]

\[ \leq \left( |\mu^0_i| + \beta^0_i \max_{I_0 \in [0, h]} |I_0(t)| \right)||S_0 - S'_0|| \]

\[ \leq L_1||S_0 - S'_0||, \]

where

\[ \|f_i(t, \cdot) - f_i(t, \cdot)\| \leq L_1\|S_0 - S'_0\|. \]

In a similar way, we obtain

\[ \|f_j(t, \cdot) - f_j(t, \cdot)\| \leq L_2\|S_0 - S'_0\|. \]

For

\[ |f_2(t, I_0) - f_2(t, I'_0)| = |\beta^1_0 S_0 I_0 - (\mu^1 + \delta^1_i) + \beta^1_0 S_0 I_0 - \beta^1_0 S_0 I'_0| \]

\[ \leq \left( \beta^1_0 \max_{I_0 \in [0, h]} |S_0(t)| + |(\mu^1 + \delta^1_i) + \beta^1_0 S_0 I_0(t)| \right)||I_0 - I'_0||, \]

which implies;

\[ \|f_2(t, I_0) - f_2(t, I'_0)\| \leq L_2\|I_0 - I'_0\|. \]

Similarly

\[ \|f_2(t, I_0) - f_2(t, I'_0)\| \leq L_4||I_0 - I'_0||, \]

\[ \|f_3(t, H_0) - f_3(t, H'_0)\| \leq L_3||H_0 - H'_0||, \]

\[ \|f_6(t, F_0) - f_6(t, F'_0)\| \leq L_6||F_0 - F'_0||, \]

\[ \|f_7(t, C_0) - f_7(t, C'_0)\| \leq L_8||C_0 - C'_0||. \]

**Lemma 1:** The continuous system (1) through (8) can be transformed to equivalent Volterra-integral equations.

**Proof:** Consider

\[ 0CD_T S_0(t) = f_1(t, S_0(t)). \]

On integrating fractionally, we get;

\[ 0CD_T^{-\alpha}[0CD_T S_0(t)] = 0CD_T^{-\alpha}[f_1(t, S_0(t))]. \]

\[ S_0(t) = S_0(0) + \frac{1}{\Gamma(\alpha)} \int_0^t (t - \tau)^{\alpha-1} f_1(\tau, S_0(\tau))d\tau; \]

Similarly,

\[ I_0(t) = I_0(0) + \frac{1}{\Gamma(\alpha)} \int_0^t (t - \tau)^{\alpha-1} f_2(\tau, I_0(\tau))d\tau; \]

\[ H_0(t) = H_0(0) + \frac{1}{\Gamma(\alpha)} \int_0^t (t - \tau)^{\alpha-1} f_3(\tau, H_0(\tau))d\tau; \]

\[ F_0(t) = F_0(0) + \frac{1}{\Gamma(\alpha)} \int_0^t (t - \tau)^{\alpha-1} f_6(\tau, F_0(\tau))d\tau; \]

\[ P_0(t) = P_0(0) + \frac{1}{\Gamma(\alpha)} \int_0^t (t - \tau)^{\alpha-1} f_7(\tau, P_0(\tau))d\tau; \]

\[ C_0(t) = C_0(0) + \frac{1}{\Gamma(\alpha)} \int_0^t (t - \tau)^{\alpha-1} f_7(\tau, C_0(\tau))d\tau. \]

**Theorem 7:** as in [9] Let $0 < \alpha < 1, I \in [0, h^*] \subseteq \mathbb{R}$ and $J = [S_0(t) - S_0(0)] \leq k_1$

Let $f_j : I \times J \rightarrow \mathbb{R}$ be continuous bounded function, that is $\forall M > 0$ such that $|f_j(t, S_0)| \leq M_1$, assume that $f_j$ satisfies Lipschitz conditions. If $L_3k_1 < M_1$, then there exist unique $S_0 \in C[0, h^*]$, such that $h^* = \min \left[ h, \left( \frac{M_1}{L_3k_1} \right)^{1/\alpha} \right]$.

**Proof:** let $T = \{ S_0 \in C[0, h^*] : \|S_0(t) - S_0(0)\| \leq k_1 \}$. 


Fractional order epidemic model for the dynamics of novel COVID-19

Since $T \subseteq \mathbb{R}$ and its closed set, then $T$ is complete metric space.
Recall that,
\[
S_b(t) = S_b(0) + \frac{1}{\Gamma(z)} \int_0^t (t-\tau)^{z-1} f_1(\tau, S_b(\tau))d\tau.
\]  
(25)

Define operator $F$ in $T$ as;
\[
FS_b(t) = S_b(0) + \frac{1}{\Gamma(z)} \int_0^t (t-\tau)^{z-1} f_1(\tau, S_b(\tau))d\tau.
\]  
(33)

To show that (40) satisfies theorem 1 (Picard-Banach)
First,
\[
|FS_b(t) - S_b(0)| = \left| \frac{1}{\Gamma(z)} \int_0^t (t-\tau)^{z-1} f_1(\tau, S_b(\tau))d\tau \right|
\leq \frac{1}{\Gamma(z)} \int_0^t (t-\tau)^{z-1} |f_1(\tau, S_b(\tau))|d\tau
\leq \frac{1}{\Gamma(z)} \int_0^t (t-\tau)^{z-1} M_1 d\tau
= \frac{M_1}{\Gamma(z+1)} t^z
= \frac{M_1}{\Gamma(z+1)} (h')^z
\leq \frac{M_1}{\Gamma(z+1)} k_1 \Gamma(z+1) \frac{k_1 \Gamma(z+1)}{M_1}
= k_1.
\]  
(34)

This implies;
\[
|FS_b(t) - S_b(0)| \leq k_1.
\]  
(35)

Similarly,
\[
|FI_b(t) - I_b(0)| \leq k_2,
\]  
(36)

\[
|FS_b(t) - S_b(0)| \leq k_3,
\]  
(37)

\[
|FI_b(t) - I_b(0)| \leq k_4,
\]  
(38)

\[
|FF_m(t) - F_m(0)| \leq k_6,
\]  
(39)

\[
|FP_c(t) - P_c(0)| \leq k_7,
\]  
(40)

\[
|FC_c(t) - C_c(0)| \leq k_8.
\]  
(41)

Therefore $F$ maps $T$ onto itself.
Secondly, to show that $T$ is contractive
\[
FS_b - FS_b = S_b(t) - S_b(0) + \frac{1}{\Gamma(z)} \int_0^t (t-\tau)^{z-1} \left[ f_1(\tau, S_b(\tau)) - f_1(\tau, S_b'(\tau)) \right]d\tau.
\]
Since $S_b(0) = S_b'(0)$, then
\[
|FS_b - FS_b| = \left| \frac{1}{\Gamma(z)} \int_0^t (t-\tau)^{z-1} \left[ f_1(\tau, S_b(\tau)) - f_1(\tau, S_b'(\tau)) \right]d\tau \right|
\leq \frac{1}{\Gamma(z)} \int_0^t (t-\tau)^{z-1} |f_1(\tau, S_b(\tau)) - f_1(\tau, S_b'(\tau))|d\tau.
\]
By Substituting (24) we get
\[
\leq \frac{1}{\Gamma(z)} \int_0^t (t-\tau)^{z-1} L_4 ||S_b - S_b'||d\tau
= \frac{L_4}{\Gamma(z)} ||S_b - S_b'|| \int_0^t (t-\tau)^{z-1} \tau^b d\tau
= \frac{L_4}{\Gamma(z+1)} ||S_b - S_b'|| \frac{\Gamma(z)}{\Gamma(z+1)} \tau^b
\leq \frac{L_4}{\Gamma(z+1)} ||S_b - S_b'|| (h')^z
\leq \frac{L_4}{\Gamma(z+1)} ||S_b - S_b'|| \frac{k_1 \Gamma(z+1)}{M_1}.
\]
Therefore,
\[
||FS_b - FS_b|| \leq \frac{L_4}{M_1} ||S_b - S_b||.
\]  
(42)

Since by hypothesis $\frac{L_4 k_1}{M_1} < 1$, then $T$ is contractive and has a unique fixed point. Thus, equation (1) has unique solution.
In a similar way, we obtain
\[
||FI_b - FI_b|| \leq \frac{L_5 k_5}{M_5} ||I_b - I_b||,
\]  
(43)

\[
||FS_b - FS_b|| \leq \frac{L_6 k_6}{M_6} ||S_b - S_b||,
\]  
(44)

\[
||FI_b - FI_b|| \leq \frac{L_7 k_7}{M_7} ||I_b - I_b||,
\]  
(45)

\[
||FH_b - FH_b|| \leq \frac{L_8 k_8}{M_8} ||H_b - H_b||,
\]  
(46)

\[
||FF_m - FF_m|| \leq \frac{L_9 k_9}{M_9} ||F_m - F_m||,
\]  
(47)

\[
||FP_c - FP_c|| \leq \frac{L_4 k_4}{M_4} ||P_c - P_c||,
\]  
(48)

\[
||FC_c - FC_c|| \leq \frac{L_4 k_4}{M_4} ||C_c - C_c||.
\]  
(49)

5. Stability analysis and derivation of basic reproduction ratio

In this chapter, we find the equilibrium solutions and carry out local stability analysis of the solutions. We use the condition for the local stability to derive the basic reproduction ratio. Global stability analysis was also carried by constructing the appropriate Lyapunov function.
5.1. Equilibria solutions

To find the equilibrium solutions we equate the system (1) through (8) to zero

\[ \delta_k^2 - \mu S_k - \beta_k^2 S_k I_k = 0, \]  \hspace{1cm} (50)

\[ \beta_k^2 S_k I_k - (\mu_k + \delta_k) I_k = 0, \]  \hspace{1cm} (51)

\[ \delta_k^2 - \mu S_k - \beta_k^2 S_k I_k = 0, \]  \hspace{1cm} (52)

\[ \beta_k^2 S_k I_k - (\mu_k + \delta_k) I_k = 0, \]  \hspace{1cm} (53)

\[ \delta_k^2 - \mu S_k - \beta_k^2 S_k I_k = 0, \]  \hspace{1cm} (54)

\[ \beta_k^2 S_k I_k - (\mu_k + \delta_k) I_k = 0, \]  \hspace{1cm} (55)

\[ \beta_k^2 P, F_m = - (\mu_k + \delta_k) F_m - \beta_k^2 P, F_m = 0, \]  \hspace{1cm} (56)

\[ \beta_k^2 P, C_e = - (\mu_k + \delta_k) C_e = 0. \]  \hspace{1cm} (57)

The endemic equilibrium \( E_0 \) was obtained by equating \( I_k, I_h, H_k, F_m, P, \) and \( C \), to zero. Hence we obtain;

\[ E_0 = (S_0^k, I_0^k, F_0^k, H_0^k, F_0^m, P_0^k, C_0^e) = \left( \frac{\mu}{\mu_k}, \frac{\mu}{\mu_k}, 0, 0, 0, 0, 0, 0 \right). \]

N = \(-\mu_k - \beta_k^2 I_k, A = \beta_k^2 S_k, \)

\(- (\mu_k + \delta) - \beta_k S_k, B = \beta_k I_k, C = \beta_k I_k - (\mu_k + \delta_k) - \beta_k F_m, D = \beta_k H_k - (\mu_k + \delta_k) - \beta_k P, E = \beta_k F_m - (\mu_k + \delta_k) - \beta_k C, G = \beta_k P, - (\mu_k + \delta_k), \) and \( Q = \mu_k - \beta_k I_k. \)

The endemic equilibrium is obtained by taking all the variables to be different from zero.

Solving (50) – (57)

\[ P^* = \left( \frac{\mu_k}{\mu} \right), \]  \hspace{1cm} (57a)

\[ H^e = \left( \frac{\mu_k}{\mu_k} \right), \]  \hspace{1cm} (58a)

\[ S^e = \left( \frac{\mu_k}{\mu_k} \right), \]  \hspace{1cm} (50a)

\[ S^e = \left( \frac{\mu_k}{\mu_k} \right), \]  \hspace{1cm} (52a)

\[ \beta_k^2 S_k - (\mu_k + \delta_k) - \beta_k^2 S_k = 0, \]  \hspace{1cm} (51a)

Putting (50) and (52) into (51) yields

\[ \beta_k^2 \mu_k - \mu_k \]

\[ \mu_k \]

\[ \mu_k \]

\[ \mu_k \]

Theorem 8: The disease free equilibrium \( E_0 \) is locally asymptotically stable.

Proof: Consider the Jacobian matrix at disease free equilibrium;

\[ J_{E_0} = \begin{bmatrix}
-\mu_k^2 & -\mu_k^2 / \mu & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & Y_1 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & -\mu_k^2 / \mu & -\mu_k^2 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & Y_0 \\
\end{bmatrix}, \]

where

\[ Y_1 = \beta_k^2 / \mu_k^2 - (\mu_k + \delta_k), \]

\[ Y_2 = - (\mu_k^2 + \delta_k), \]

\[ Y_3 = - (\mu_k^2 + \delta_k), \]

\[ Y_4 = - (\mu_k^2 + \delta_k). \]

Then using the relation;

\[ \det J_{E_0} = 0, \]

we get the eigenvalues as;

\[ K_1 = -\mu_k. \]
\[ K_2 = \frac{\beta_1^2 S^2_b}{\mu_b^2} - (\mu_b^2 + \delta_b^2) - \frac{\beta_2^2 S^2_b}{\mu_b^2} \]
\[ K_3 = -\mu_b^3, \]
\[ K_4 = -\left(\mu_b^4 + \delta_b^4\right), \]
\[ K_5 = -\left(\mu_b^5 + \delta_b^5\right), \]
\[ K_6 = -\left(\mu_b^6 + \delta_b^6\right), \]
\[ K_7 = -\left(\mu_b^7 + \delta_b^7\right), \]
\[ K_8 = -\left(\mu_b^8 + \delta_b^8\right), \]

Since the Im(K) = 0, i = 1, 2, \ldots, 8, then clearly
\[ |\text{Arg}(K)| = \pi > \frac{2\pi}{3} \quad 0 < \alpha < 1. \]

Then by theorem [2], \( E_0 \) is locally asymptotically stable.

5.2.1. Basic reproduction number
For the DFE to be stable, we need the eigenvalue \( K_2 < 0 \)
\[ \beta_1^2 S^2_b - (\mu_b^2 + \delta_b^2) - \frac{\beta_2^2 S^2_b}{\mu_b^2} < 0. \]

Simplifying, we get;
\[ \frac{\beta_1^2 S^2_b}{\mu_b^2} - (\mu_b^2 + \delta_b^2) + \frac{\beta_2^2 S^2_b}{\mu_b^2} < 1. \]

We let basic reproduction ratio (\( R_0 \)) to be;
\[ R_0 = \frac{\beta_1^2 S^2_b}{\mu_b^2 (\mu_b^2 + \delta_b^2) + \beta_2^2 S^2_b}. \]

\textbf{Theorem 9:} The endemic equilibrium \( (S^*_b, I^*_b, S^*_b, I^*_b, H^*_b, F^*_m, P^*_c, C^*_c) \) is stable if \( R_0 > 1 \).

\textbf{Proof:} Since all the equilibrium points in the endemic equilibrium depend on \( I^*_b \), then it suffices to investigate the stability or otherwise of \( I^*_b \)

Since \( I^*_b > 0 \), then from (53+) either
\[ \frac{\lambda^2 - \lambda^2}{\mu_b^2 + \delta_b^2} + \frac{\mu_b^2}{\mu_b^2} > \frac{\lambda^2 - \lambda^2}{\mu_b^2 + \delta_b^2} + \frac{\mu_b^2}{\mu_b^2 (\mu_b^2 + \delta_b^2)} \left(1 - \frac{1}{R_0}\right) \]
\[ 0 > \frac{4\mu_b^2}{\beta_2^2 (\mu_b^2 + \delta_b^2) \left(1 - \frac{1}{R_0}\right)} \]
\[ 0 > \left(1 - \frac{1}{R_0}\right) \]
\[ R_0 < 1, \]

which may lead to a change of \( I^*_b \) to be complex.

or
\[ \frac{\lambda^2 - \lambda^2}{\mu_b^2 + \delta_b^2} + \frac{\mu_b^2}{\mu_b^2} > \frac{4\mu_b^2}{\beta_2^2 (\mu_b^2 + \delta_b^2) \left(1 - \frac{1}{R_0}\right)} \]
By theorem 4

\[
\begin{align*}
&\leq \left(1 - \frac{S_b(t)}{S_b}\right) 0CD_f^t S_b(t) + \left(1 - \frac{I_b(t)}{I_b}\right) 0CD_f^t I_b(t) \\
&+ \left(1 - \frac{S_b(t)}{S_b}\right) 0CD_f^t S_b(t) + \left(1 - \frac{I_b(t)}{I_b}\right) 0CD_f^t I_b(t) \\
&+ \left(1 - \frac{H_b(t)}{H_b}\right) 0CD_f^t H_b(t) + \left(1 - \frac{F_m(t)}{F_m}\right) 0CD_f^t F_m(t) \\
&+ \left(1 - \frac{P_c(t)}{P_c}\right) 0CD_f^t P_c(t) + \left(1 - \frac{C_c(t)}{C_c}\right) 0CD_f^t C_c(t) \\
&= \left(1 - \frac{S_b(t)}{S_b}\right) [\beta_1^2 S_b - \mu_1^2 S_b - \beta_1^2 H_b I_b] \\
&+ \left(1 - \frac{I_b(t)}{I_b}\right) [\beta_1^2 S_b I_b - (\mu_1^2 + \delta_1^2) I_b - \beta_1^2 H_b I_b] \\
&+ \left(1 - \frac{S_b(t)}{S_b}\right) [\beta_1^2 S_b I_b - (\mu_1^2 + \delta_1^2) I_b - \beta_1^2 H_b I_b] \\
&+ \left(1 - \frac{H_b(t)}{H_b}\right) [\beta_1^2 I_b H_b - (\mu_1^2 + \delta_1^2) H_b - \beta_1^2 H_b F_m] \\
&+ \left(1 - \frac{F_m(t)}{F_m}\right) [\beta_1^2 H_b F_m - (\mu_1^2 + \delta_1^2) F_m - \beta_1^2 P_c F_m] \\
&+ \left(1 - \frac{P_c(t)}{P_c}\right) [\beta_1^2 P_c F_m - (\mu_1^2 + \delta_1^2) P_c - \beta_1^2 P_c C_c] \\
&+ \left(1 - \frac{C_c(t)}{C_c}\right) [\beta_1^2 P_c C_c - (\mu_1^2 + \delta_1^2) C_c].
\end{align*}
\]

Recall from the positive equilibrium, we have:

\[
\begin{align*}
\lambda_b^2 &= \mu_1^2 S_b + \beta_1^2 S_b I_b, (11 \ast \ast) \\
\lambda_h^2 &= \mu_1^2 S_b + \beta_1^2 S_b I_b, (12 \ast \ast) \\
\mu_1^2 + \delta_1^2 &= \beta_1^2 S_b - \beta_1^2 S_b = 0, (13 \ast \ast) \\
\mu_1^2 + \delta_1^2 &= \beta_1^2 S_b - \beta_1^2 S_b = 0, (14 \ast \ast) \\
\mu_1^2 + \delta_1^2 &= \beta_1^2 I_b - \beta_1^2 P_c, (15 \ast \ast) \\
\mu_1^2 + \delta_1^2 &= \beta_1^2 I_b - \beta_1^2 P_c, (16 \ast \ast) \\
\mu_1^2 + \delta_1^2 &= \beta_1^2 F_m - \beta_1^2 C_c, (17 \ast \ast) \\
\mu_1^2 + \delta_1^2 &= \beta_1^2 P_c, (18 \ast \ast)
\end{align*}
\]

Therefore, we have:

\[
\begin{align*}
&= \left(1 - \frac{S_b(t)}{S_b}\right) [\beta_1^2 (S_b - S_b) + \beta_1^2 (S_b I_b - S_b I_b)] \\
&+ \left(1 - \frac{I_b(t)}{I_b}\right) [\beta_1^2 (S_b - S_b) I_b - \beta_1^2 (S_b I_b - S_b I_b)] \\
&+ \left(1 - \frac{S_b(t)}{S_b}\right) [\beta_1^2 (S_b I_b - S_b I_b)] \\
&+ \left(1 - \frac{H_b(t)}{H_b}\right) [\beta_1^2 (S_b I_b - S_b I_b)] \\
&+ \left(1 - \frac{F_m(t)}{F_m}\right) [\beta_1^2 (S_b I_b - S_b I_b)] \\
&+ \left(1 - \frac{P_c(t)}{P_c}\right) [\beta_1^2 (S_b I_b - S_b I_b)] \\
&+ \left(1 - \frac{C_c(t)}{C_c}\right) [\beta_1^2 (S_b I_b - S_b I_b)].
\end{align*}
\]

\[
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\]
Since for any \( a, b \in \mathbb{R}, 2ab \leq a^2 + b^2 \) or \( ab \leq \frac{1}{2}a^2 + \frac{1}{2}b^2 \), then:

\[
aCDV^\alpha \leq \frac{a^2}{b^2} (S_a - S_b)^2 + \frac{1}{a^2} (S_a - S_b)^2 + \frac{1}{2} \frac{b^2}{a^2} (S_a - S_b)^2, \\
+ \frac{b^2}{a^2} (I_m - I_m)^2 \frac{2}{m} \frac{b^2}{a^2} (S_a - S_b)^2 - \frac{b^2}{a^2} (I_m - I_m)^2 - \frac{b^2}{a^2} (I_m - I_m)^2, \\
+ \frac{b^2}{a^2} (S_a - S_b)^2 + \frac{b^2}{a^2} (S_a - S_b)^2 - \frac{b^2}{a^2} (I_m - I_m)^2, \\
- \frac{b^2}{a^2} (S_a - S_b)^2 \frac{2}{m} \frac{b^2}{a^2} (I_m - I_m)^2 + \frac{b^2}{a^2} (I_m - I_m)^2, \\
+ \frac{b^2}{a^2} (S_a - S_b)^2 \frac{2}{m} \frac{b^2}{a^2} (I_m - I_m)^2, \\
+ \frac{b^2}{a^2} (I_m - I_m)^2 (S_m - S_m)^2 - (I_m - I_m)^2, \\
(S_a - S_b)^2 (S_a - S_b)^2 \frac{2}{m} \frac{b^2}{a^2} (I_m - I_m)^2, \\
+ \frac{b^2}{a^2} (S_a - S_b)^2 \frac{2}{m} \frac{b^2}{a^2} (I_m - I_m)^2. \\
\]

\[
\left\{ (S_a - S_b)^2 \left[ \frac{b^2}{a^2} \frac{I_m}{2m} + \frac{b^2}{a^2} \frac{S_m}{2m} \right] - \frac{b^2}{a^2} (S_a - S_b)^2 \frac{2}{m} \frac{b^2}{a^2} (I_m - I_m)^2, \\
+ \frac{b^2}{a^2} (S_a - S_b)^2 \frac{2}{m} \frac{b^2}{a^2} (I_m - I_m)^2, \\
- \frac{b^2}{a^2} (S_a - S_b)^2 \frac{2}{m} \frac{b^2}{a^2} (I_m - I_m)^2, \\
+ \frac{b^2}{a^2} (S_a - S_b)^2 \frac{2}{m} \frac{b^2}{a^2} (I_m - I_m)^2. \\
\right\}
\]

Let \( W = \left\{ (S_a - S_b)^2 \left[ \frac{b^2}{a^2} \frac{I_m}{2m} + \frac{b^2}{a^2} \frac{S_m}{2m} \right] - \frac{b^2}{a^2} (S_a - S_b)^2 \frac{2}{m} \frac{b^2}{a^2} (I_m - I_m)^2, \\
+ \frac{b^2}{a^2} (S_a - S_b)^2 \frac{2}{m} \frac{b^2}{a^2} (I_m - I_m)^2, \\
- \frac{b^2}{a^2} (S_a - S_b)^2 \frac{2}{m} \frac{b^2}{a^2} (I_m - I_m)^2, \\
+ \frac{b^2}{a^2} (S_a - S_b)^2 \frac{2}{m} \frac{b^2}{a^2} (I_m - I_m)^2. \\
\right\}
\]

We can verify that \( W(S_a, I_a, S_b, I_b, H_m, F_m, P_c, C_c) \) is continuous on \( \mathbb{R}^5 \) and \( W(S_a, I_a, S_b, I_b, H_m, F_m, P_c, C_c) > 0 \), for all \( (S_a, I_a, S_b, I_b, H_m, F_m, P_c, C_c) \) \( \in \mathbb{R}^8 \). \( W(S_a, I_a, S_b, I_b, H_m, F_m, P_c, C_c) \) is positive definite function. Thus, by \textit{Theorem 3} the equilibrium solution \( (S_a^e, I_a^e, S_b^e, I_b^e, H_m^e, F_m^e, P_c^e, C_c^e) \) is globally asymptotically stable.

6. Numerical simulations

Numerical simulations using the variables and parameter values as given in Table 3 and 4 respectively, are carried out.
From the graphs, we can see that FODEs have rich dynamics and are better descriptors of biological systems than traditional integer – order models. From Fig. 2 (\( \alpha = 1 \))to Fig. 6 (\( \alpha = 0.2 \)), we can observe that the number of infection get to zero with time. This is true as per as any epidemic disease is concern. We note that that the solution of the model, with various values of \( \alpha \), continuously depends on the time – fractional derivative, but arrives to the equilibrium points. The displayed solution in Figs. 2 – 6 confirm that the fractional order plays the role of time – delay in the systems.

7. Conclusion

In conclusion, it was observed that indeed FODE is the generalization of integer order differential equation. Using fractional order differential equations can help us to reduce the errors arising from the neglected parameters in modeling biological systems with memory and system distributed parameters. In this paper, we presented a fractional order differential order model in Caputo sense to study Covid – 19. We proved the existence and uniqueness of the solution of the model by applying Banach contraction mapping principle. The equilibrium solutions (disease free & endemic) of the model were found to be locally asymptotically stable. The key parameter (Basic reproduction number) describing the number of secondary infections was obtained. Furthermore, global stability analysis of the solutions was carried out using Lyapunov candidate function. We performed numerical simulation, which shows the changes that occur at every time instant due to the variation of \( \alpha \).

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.

Table 3 parameter values.

| Variable | Value       | Reference |
|----------|-------------|-----------|
| \( S_0 \) | 0 – 600     | As in [4]  |
| \( I_0 \) | 200 – 500   | As in [4]  |
| \( S_0(0) \) | 10,000,000 | As in [4]  |
| \( I_0 \) | 240 – 440   | As in [4]  |
| \( H_0 \) | 100 – 400   | As in [4]  |
| \( F_m \) | 40 – 200    | As in [4]  |
| \( P_e \) | 0 – 300     | As in [4]  |
| \( C_e \) | 0 – 300     | As in [4]  |

Table 4 parameter values.

| Parameter value | Reference |
|-----------------|-----------|
| \( \beta_1 \)   | 1.2300    | As in [4]  |
| \( \beta_2 \)   | 0.1000    | As in [4]  |
| \( \beta_3 \)   | 0.0060    | As in [4]  |
| \( \beta_4 \)   | 1.0090    | As in [4]  |
| \( \beta_5 \)   | 0.0040    | As in [4]  |
| \( \beta_6 \)   | 0.0900    | As in [4]  |
| \( \lambda_2 \) | 1.5000    | As in [4]  |
| \( \lambda_3 \) | 1.25      | Assumed    |
| \( \mu_1 \)     | 1.7000    | As in [4]  |
| \( \mu_2 \)     | 0.1340    | As in [4]  |
| \( \mu_3 \)     | 0.5       | Assumed    |
| \( \mu_4 \)     | 0.1343    | As in [4]  |
| \( \mu_5 \)     | 0.0024    | As in [4]  |
| \( \mu_6 \)     | 0.0074    | As in [4]  |
| \( \mu_7 \)     | 0.3440    | As in [4]  |
| \( \mu_8 \)     | 0.501410  | As in [4]  |
| \( \delta_1 \)  | 0.0143    | As in [4]  |
| \( \delta_2 \)  | 0.3002    | As in [4]  |
| \( \delta_3 \)  | 0.0054    | As in [4]  |
| \( \delta_4 \)  | 0.0019    | As in [4]  |
| \( \delta_5 \)  | 0.0640    | As in [4]  |
| \( \delta_6 \)  | 0.4400    | As in [4]  |

Fig. 2 Numerical simulations of the system of FODE (1) – (8) with \( \alpha = 1 \)
Fig. 3  Numerical simulations of the system of FODE (1) – (8) with $\alpha = 0.8$

Fig. 4  Numerical simulations of the system of FODE (1) – (8) with $\alpha = 0.6$

Fig. 5  Numerical simulations of the system of FODE (1) – (8) with $\alpha = 0.4$
Fig. 6  Numerical simulations of the system of FODE (1) – (8) with $x = 0.2$