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Fractional Order Model for the Role of Mild Cases in the Transmission of COVID-19

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Abstract
Most of the nations with deplorable health conditions lack rapid COVID-19 diagnostic test due to limited testing kits and laboratories. The un-diagnostic mild cases (who show no critical sign and symptoms) play the role as a route that spread the infection unknowingly to healthy individuals. In this paper, we present a fractional order SIR model incorporating individual with mild cases as a compartment to become SMIR model. The existence of the solutions of the model is investigated by solving the fractional Gronwall’s inequality using the Laplace transform approach. The equilibrium solutions (DFE & Endemic) are found to be locally asymptotically stable, and subsequently the basic reproduction number is obtained. Also the global stability analysis is carried out by constructing Lyapunov function. Lastly, numerical simulations that support analytic solution follow. It was also shown that when the rate of infection of the mild cases increases, there is equivalent increase in the overall population of infected individuals. Hence to curtail the spread of the disease there is need to take care of the Mild cases as well.

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1. Introduction

The outbreak of the novel strain of Corona viruses (COVID-19) started late December in the Wuhan province in China [1]. It became a global pandemic causing the devastating impact in terms of morbidity, infections and fatality in addition to socio-economic disaster. The virus source which is yet to be identified is said to have genetic linkages with Severe Acute Respiratory Syndrome (SARS-COV) but less severe than Middle East Respiratory Syndrome (MERS-COV) [2].

The virus is transmitted to healthy persons via eyes, mouth and nose when an infected person produced respiratory droplets of cough and sneeze or as a result of contact with contaminated surfaces. The average incubation period from catching the virus to time onset of major symptoms (like fever, cough and sneeze) is between 2 – 14 days [3].

As the vaccine is not yet found, the control measures such as: social distancing, quarantine of suspected case, use of personal protective equipment (like face Mask, hand globes, gown), regular hand sanitation using antibacterial agents (like soaps, sanitizer) and imposing the lockdown curfew (when necessary) are the effective intervention that mitigate the transmission of the infection.

To execute these measures effectively, there is need to have an in depth study about the number of persons that each infected individual can infect, meanwhile a mathematical model describing the transmission dynamics of the disease should be established. In this regard, Zhao and Chen [4] developed a Susceptible, Un-quarantined infected, Quarantined infected, Confirmed infected (SUQC) model to characterize the dynamics of COVID-19 and explicitly parameterized the intervention effects of control measures. Similarly, Song et al. [5] developed a mathematical model based on the epidemiology of COVID-19, incorporating the isolation of healthy people, confirmed cases and close contacts.

Tahir et al. [6] developed a 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 equilibriums solution and subsequently obtained the basic reproduction number or roughly, a key parameter describing transmission of the infection. Also, Chen et al. [7] 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 focuses on calculating $R_0$.

To suit Korean outbreak, Sunhwa and Moran [8] established deterministic mathematical model (in form of SEIKR), in which they estimated the reproduction number and assessed the effect of pre-
ventive measures. Similarly, Lin et al. [9] 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 primary magnitude of different effect of individual reaction and governmental action. Also Yang and Wang [10] proposed a mathematical model to investigate the current outbreak of the coronavirus disease (COVID-19) in Wuhan, China. The model described the multiple transmission pathways in the infection dynamics, and emphasized the role of environmental reservoir in the transmission and spread of the disease. However, the model employed non-constant transmission rates which change with the epidemiological status and environmental conditions and which reflect the impact of the ongoing disease control measures.

Non-locality is one of the main drivers of interest in fractional calculus applications. There are interesting phenomena that have what are called memory effects, meaning their state does not depend solely on time and position but also on previous state. Such system can be very difficult to model and analyze with classical differential equations, but non-locality gives fractional derivative built-in ability to incorporate memory effects [11]. Fractional differential equations appear naturally in numerous fields of study including physics, polymer rheology, regular variation in thermodynamics, biophysics, blood flow phenomena, aerodynamics, electrodynamics of complex medium, viscoelasticity, capacitor theory, electrical circuits, electron-analytical chemistry, biology, control theory, and fitting of experimental data [12–14,22–26]. Recently there are many studies on epidemiological disease modeling using fractional order differential equations [27–33].

The Riemann-Liouville fractional derivative is mostly used by mathematicians, but this approach is not suitable for real world physical problems since it requires the definition of fractional order initial conditions, which have no physically meaningful explanation yet. Caputo introduced an alternative definition, which has the advantage of defining integer order initial condition for fractional differential equations [15].

In mostly poor and underdeveloped territories where there is no capacity of rapid diagnostic COVID-19 test due to insufficiency of testing kits, the Mild cases (who usually show no symptoms of the infection due to their strong and active immune system up to their recovery period) play a major role as a route that spread the disease to healthy individuals. Here we build our model by incorporating the population of mild individuals into the compartmental SIR model to become SMIR model in form of system of fractional order differential equations (FODE) in the Caputo sense. It should be noted that to our knowledge no model in literature considered the contribution of the mild cases of COVID – 19 in the proliferation of the pandemic.

The paper is organized as follows: Section 1 is the introduction, Section 2 is the preliminary definitions and theorems, the model formulation followed in Section 3, Section 4 is the stability analysis, Section 5 gives numerical simulations and discussions and Section 6 gives the conclusion.

2. Preliminary Definitions and Theorems

**Definition 1.** Gamma function of $p > 0$ is defined as

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

**Definition 2** [16]. The Caputo fractional derivative of order $\alpha \in (n - 1, n]$ of $f(x)$ is defined as

$$C_\alpha^D_t f(x) = \frac{1}{\Gamma(n - \alpha)} \int_0^x (x-t)^{n-\alpha-1}f^n(t)dt, \quad n = [\alpha] + 1.$$  

**Definition 3** [17]. The Laplace transform of $f(t)$ denoted by $L\{f(t)\} = F(s)$ is defined by the integral transform as

$$F(s) = \int_0^\infty e^{-st}f(t)dt.$$  

**Lemma 1.** Notice that the Laplace transform of an $n$-th derivative operator is obtained as

$$L\{t^n f(t)\} = S^n L\{f(t)\} - \sum_{k=0}^{n-1} s^{n-k-1} f^{(k)}(t_0).$$

Similarly for $\alpha \in (n - 1, n]$ we obtain the Laplace transform of the Caputo fractional operator as

$$L\{C_\alpha^D_t f(t)\} = S^n L\{f(t)\} - \sum_{k=0}^{n-1} s^{n-k-1} f^{(k)}(t_0).$$

**Definition 4** [18]. An entire function called Mittag-Leffler is defined in the form of power series as

$$E_{\alpha, \beta}(z) = \sum_{n=0}^{\infty} \frac{z^n}{\Gamma(\alpha k + \beta)}, \quad \alpha > 0, \quad \beta > 0,$$

and

$$E_{\alpha, 1}(z) = E_{\alpha}(z) = \sum_{n=0}^{\infty} \frac{z^n}{\Gamma(\alpha k + 1)}, \quad \beta = 1.$$  

The notion of convergence of mittag-Leffler function is fully discussed in [15].

**Theorem 1** [19]. The equilibrium solutions $x^*$ of the system

$$C_\alpha^D_t x(t) = f(t, x), \quad x(t_0) = x_0,$$

is locally asymptotically stable if all the eigenvalues $\lambda_i$ of the Jacobian matrix $\frac{df}{dx}$ evaluated at the equilibrium points satisfy

$$|\arg(\lambda_i)| > \frac{\pi}{\alpha}, \quad 0 < \alpha < 1.$$  

**Theorem 2** [20]. Let $x = 0$ be an equilibrium of non-autonomous fractional order system

$$C_\alpha^D_t x(t) = f(t, x), \quad x(t_0) = x_0,$$

let $\Omega \subseteq \mathbb{R}^n$ be a domain containing $x = 0$.

Let $V(t, x) : [t_0, \infty) \times \Omega \rightarrow \mathbb{R}$ be continuously differentiable function such that

$$W_1(x) \leq V(t, x) \leq W_2(x)$$

$$C_\alpha^D_t V(t, x) \leq -W_2(x), \quad \text{for } t \geq t_0, \quad x \in \Omega.$$  

Where $W_1(x)$, $W_2(x)$ and $W_3(x)$ be continuous positive definite functions on $\Omega$ and $V$ is a Lyapunov candidate function, then $x = 0$ is globally asymptotically stable.

**Lemma 2** [21]. Let $x(t) \in \mathbb{R}$ be continuously differentiable function, then for any time instant $t \geq t_0$

$$\frac{1}{\Gamma(\alpha)} \int_{t_0}^{t} C_\alpha^D_t x^\alpha(t) \leq \int_{t_0}^{t} C_\alpha^D_t f(t), \quad \forall \alpha \in (0, 1).$$

3. Model Formulation

Despite the fact that almost 80% of the COVID – 19 cases are mild who recover naturally (due to stronger and active immune system that fight against the virus), they still play a role as a route of transmission of the infection [2]. The model was formulated based on the assumption that new born of human are recruited into susceptible class $S(t)$ at the rate $n$. The susceptible individual who had contact with an infected at rate $\beta$ can developed mild
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Fig. 1. Schematic diagram describing the COVID-19 transmission.

Table 1

| Model parameters | Interpretation | Mean Value | References |
|------------------|----------------|------------|------------|
| $\eta$           | Recruitment rate into susceptible population | 0.5 | Assumed |
| $\beta$          | Infectious transmission rate | 0.3567 | [5] |
| $k$              | Mild cases transmission rate | 0.2 | Assumed |
| $\mu$            | Natural death rate | 0.75 | Assumed |
| $\sigma$         | Average Incubation rate | $\frac{1}{14}$ | [2] |
| $\omega$         | Natural recovery rate of mild cases | $\frac{1}{14}$ | [2] |
| $\gamma$         | Disease induced death rate | 0.3002 | [6] |

3.1. Boundedness of the Solutions

With the total population

\[ N(t) = S(t) + M(t) + I(t) + R(t). \]  

\[ S(t) \geq 0, \ M(t) \geq 0, \ I(t) \geq 0, \ R(t) \geq 0, \ \forall \ t \in \mathbb{R}, \]  

\[ \frac{\partial}{\partial t} N(t) = \omega M(t) + \gamma I(t) - \mu R(t). \]  

\[ \frac{\partial}{\partial t} S(t) = \eta - \frac{\beta S(t)}{N(t)} (I(t) + k M(t)) - \mu S(t). \]  

\[ \frac{\partial}{\partial t} M(t) = \frac{\beta S(t)}{N(t)} (I(t) + k M(t)) - (\mu + \sigma + \omega) M(t). \]  

\[ \frac{\partial}{\partial t} I(t) = \sigma M(t) - (\mu + \delta + \gamma) I(t). \]  

Fig. 2. Dynamics of infected, mild and recovered populations.

symptoms and move to mild class $M(t)$. Based on [2], the mild patients with infectivity rate $k$ play the role as a routine that spread the infection, those with strong immunity recovered naturally at the rate $\omega$. While some with critical illness became infected and moved to infectious class $I(t)$ after the incubation period $\frac{1}{14}$. The infectious individual may then recovered $R(t)$ or died at the rates $\gamma$ and $\delta$ respectively. Fig. 1 gives the schematic diagram describing the transmission dynamics of the disease.
Fig. 3. Dynamics of Infected individuals for various values of $\alpha$.

Fig. 4. Dynamics of Mild individuals for various values of $\alpha$.

The linearity of the Caputo operator yields

\[
\begin{align*}
\frac{\mathcal{D}_t^\alpha N(t)}{\mathcal{D}_t^\alpha S(t)} + \frac{\mathcal{D}_t^\alpha M(t)}{\mathcal{D}_t^\alpha I(t)} + \frac{\mathcal{D}_t^\alpha R(t)}{\mathcal{D}_t^\alpha I(t)} \\
= \eta^\alpha - \delta^\alpha I(t) - \mu^\alpha N(t),
\end{align*}
\]

\[
\times \frac{\mathcal{D}_t^\alpha N(t)}{\mathcal{D}_t^\alpha S(t)} \leq \eta^\alpha - \mu^\alpha N(t).
\]

We apply the Laplace transform method to solve the Gronwall’s inequality (6) with initial condition $N(t_0) \geq 0$

\[
L\left\{\frac{\mathcal{D}_t^\alpha N(t)}{\mathcal{D}_t^\alpha S(t)} + \frac{\mathcal{D}_t^\alpha N(t)}{\mathcal{D}_t^\alpha S(t)}\right\} \leq L\{\eta^\alpha\}.
\]

Linearity property of the Laplace transform gives

\[
\begin{align*}
L\left\{\frac{\mathcal{D}_t^\alpha N(t)}{\mathcal{D}_t^\alpha S(t)} + \frac{\mathcal{D}_t^\alpha N(t)}{\mathcal{D}_t^\alpha S(t)}\right\} \leq \mu^\alpha L\{N(t)\} \leq \mu^\alpha L\{N(t)\} \leq \eta^\alpha S\left(1 + \frac{\mu^\alpha}{S^\alpha}\right),
\end{align*}
\]

\[
\begin{align*}
L\{N(t)\} \leq \frac{\eta^\alpha}{S^\alpha + \mu^\alpha} + \sum_{k=0}^{n-1} \frac{S^\alpha - 1}{S^\alpha + \mu^\alpha} N(t_0) + \mu^\alpha L\{N(t)\} \leq \eta^\alpha.
\end{align*}
\]

Splitting (7) to partial fraction gives

\[
L\{N(t)\} \leq \eta^\alpha \left(1 - \frac{S^\alpha - 1}{S^\alpha + \mu^\alpha}\right) + \sum_{k=0}^{n-1} \frac{S^\alpha - 1}{S^\alpha + \mu^\alpha} N(t_0)
\]

\[
= \eta^\alpha \left(1 - \frac{1}{S^\alpha + \mu^\alpha}\right) + \sum_{k=0}^{n-1} \frac{1}{S^\alpha - 1} \frac{1}{(1 + \frac{\mu^\alpha}{S^\alpha})} N(t_0).
\]
Using Taylor series expansion that
\[
\frac{1}{1 + \frac{\mu}{S^\alpha}} = \sum_{n=0}^{\infty} \left( -\frac{\mu}{S^\alpha} \right)^n.
\]
Therefore
\[
L[N(t)] \leq \eta^\alpha \left[ \frac{1}{3} \left( \frac{1}{3} \sum_{n=0}^{\infty} \left( -\frac{\mu}{S^\alpha} \right)^n \right) + \sum_{k=0}^{n-1} \frac{1}{S^{\alpha n+1}} N^{(k)}(t_0) \sum_{n=0}^{\infty} \left( -\frac{\mu}{S^\alpha} \right)^n \right]
\leq \eta^\alpha \left( \frac{1}{3} \sum_{n=0}^{\infty} \left( -\frac{\mu}{S^\alpha} \right)^n \right) + \sum_{k=0}^{n-1} \frac{1}{S^{\alpha n+1}} N^{(k)}(t_0) \sum_{n=0}^{\infty} \left( -\frac{\mu}{S^\alpha} \right)^n.
\]
Taking the inverse Laplace transform of (8)
\[
N(t) \leq \eta^\alpha L^{-1} \left\{ \frac{1}{3} \right\} - \eta^\alpha \sum_{n=0}^{\infty} (-\mu^\alpha)^n L^{-1} \left\{ \frac{1}{S^{\alpha n+1}} \right\} + \sum_{k=0}^{n-1} \sum_{n=0}^{\infty} (-\mu^\alpha)^n N^{(k)}(t_0) L^{-1} \left\{ \frac{1}{S^{\alpha n+k+1}} \right\}.
\]
Recall that
\[
L[t^m] = \frac{m!}{S^{\alpha+1}} = \frac{\Gamma(m+1)}{S^{\alpha+1}}, \text{ or } L^{-1} \left\{ \frac{1}{S^{\alpha+1}} \right\} = \frac{t^m}{\Gamma(m+1)}.
\]
Thus
\[ N(t) \leq \eta^a - \eta^a \sum_{n_0=0}^{\infty} \frac{(-\mu^a)^n}{\Gamma(a + n + 1)} + \sum_{k=0}^{n-1} \frac{(-\mu^a)^n}{\Gamma(a + k + 1)} + \sum_{k=0}^{n-1} \frac{(-\mu^a)^n}{\Gamma(a + k + 1)} \times (t_0,F^{a+n+k}) \cdot \]

\[ N(t) \leq \eta^a - \eta^a \sum_{n_0=0}^{\infty} \frac{(-\mu^a)^n}{\Gamma(a + n + 1)} + \sum_{k=0}^{n-1} \frac{(-\mu^a)^n}{\Gamma(a + k + 1)} \, t^kN^k(t_0). \]

Substituting the Mittag-Leffler function

\[ N(t) \leq \eta^a [1 - E_1(-\mu^a t^\alpha)] + \sum_{k=0}^{n-1} E_{k+1}(-\mu^a t^\alpha)N^{k+1}(t_0)t^k. \tag{9} \]

Where \(E_1(-\mu^a t^\alpha), E_{k+1}(-\mu^a t^\alpha)\) are the series of Mittag-Leffler function (as in definition 4) which converges for any argument, hence we say that the solution to the model is bounded. Thus,

\[ \left\{ (S(t), M(t), I(t), R(t)) \in \mathbb{R}^4 : S(t), M(t), I(t), R(t) \leq \eta^a [1 - E_1(-\mu^a t^\alpha)] + \sum_{k=0}^{n-1} E_{k+1}(-\mu^a t^\alpha)N^{k+1}(t_0)t^k \right\}. \]

3.2. Uniqueness of the Solution

Consider the system (1) through (4) written as

\[ \frac{d}{dt}x(t) = F(t, x), \quad x(0) = x_0. \tag{10} \]

\[ F(t, x) = A x + g(x) + b. \]

\[ x(t) = (S(t), M(t), I(t), R(t))^T, \quad b = (\eta, 0, 0, 0)^T, \]

\[ A = \begin{bmatrix} -\mu^a & 0 & 0 & 0 \\ 0 & -\mu^a - \sigma^a - \omega^a & 0 & 0 \\ 0 & \sigma^a & -\mu^a - \delta^a & \gamma^a \\ 0 & \omega^a & \gamma^a & -\mu^a \end{bmatrix} \]

\[ g(x(t)) = \begin{bmatrix} -\frac{\beta^u S(t)}{\Gamma(\alpha)} (I(t) + k^M M(t)) \\ -\frac{\beta^u S(t)}{\Gamma(\alpha)} (I(t) + k^M M(t)) \\ 0 \\ 0 \end{bmatrix} \]

Theorem 4. System (10) satisfies Lipschitz continuity

Proof.

\[ |F(t, x) - F(t, x')| = |A(x(t) - x'(t)) + g(x(t)) - g(x'(t))| \]

\[ \leq (||A|| + 1)||x(t) - x'(t)||. \]

\[ ||F(t, x(t)) - F(t, x'(t))|| \]

\[ \leq L||x(t) - x'(t)||, \quad L = ||A|| + 1 < \infty. \tag{11} \]

Clearly, \(F\) is continuous and bounded function.

Reference to Picard-Lindelof theorem [20] we establish the following theorem.

Theorem 5. Let \(0 < \alpha < 1, \ L = [0, h^a] \subseteq \mathbb{R} \) and \( f = |x(t) - x(0)| \leq k. \)

Let \( f : I \times \mathbb{R} \rightarrow \mathbb{R} \) be continuous bounded function, that is \( \exists M > 0 \) such that \( |f(t, x)| \leq M. \)

Since \( f \) satisfies Lipschitz conditions, if \( Lk < M, \) then there exists a unique \( x \in C^\alpha [0, h^a] \) that holds for the initial value problem (10).

Where and \( h^a = \min\{h, (\frac{k^M}{M+1})^\alpha\}. \)

Proof.

Let \( T = \{ x \in C[0, h^a] : ||x(t) - x(0)|| \leq k \}, \)Since \( T \subseteq \mathbb{R} \) and its closed set, then \( T \) is complete metric space.

The continuous system (10) can be transformed to equivalent integral equations as;

\[ \xi_D^a [\xi D^a x(t)] = \xi D^a f(t, x), \]

\[ x(t) - x(0) = \frac{1}{\Gamma(\alpha)} \int_0^t (t - \tau)^{\alpha - 1} f(\tau, x(\tau)) d\tau, \tag{12} \]

\[ x(t) = x_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t - \tau)^{\alpha - 1} f(\tau, x(\tau)) d\tau. \]

Equation (12) is equivalent to Volterra integral equation that solves (10).

Define an operator \( F \) in \( T \)

\[ F[x](t) = x_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t - \tau)^{\alpha - 1} f(\tau, x(\tau)) d\tau \tag{13} \]

Now, we need to verify that (13) satisfies the hypothesis of contraction mapping principle.

First to show \( F : T \rightarrow T \)

\[ ||F[x](t) - x(0)|| = \frac{1}{\Gamma(\alpha)} \int_0^t (t - \tau)^{\alpha - 1} ||f(\tau, x(\tau))|| d\tau \]

\[ \leq \frac{1}{\Gamma(\alpha)} \int_0^t (t - \tau)^{\alpha - 1} M d\tau \]

\[ = \frac{M}{\Gamma(\alpha + 1)} t^\alpha \]

\[ = \frac{M}{\Gamma(\alpha + 1)} (h^a)^\alpha \]

\[ \leq \frac{M}{\Gamma(\alpha + 1)} \frac{k^M (\alpha + 1)}{M} ||F[x](t) - x(0)|| \leq k. \tag{14} \]

Or equivalently, \( x(0) - k \leq F[x](t) \leq x(0) + k, \forall t \in [0, h^a]. \)

Hence the operator \( F \) maps \( T \) onto itself.

Secondly, to show that \( T \) is a contraction, we have

\[ ||F[x](t) - F[x'](t)|| \]

\[ = \frac{1}{\Gamma(\alpha)} \int_0^t (t - \tau)^{\alpha - 1} ||f(\tau, x(\tau)) - f(\tau, x'(\tau))|| d\tau \]

\[ \leq \frac{1}{\Gamma(\alpha)} \int_0^t (t - \tau)^{\alpha - 1} ||f(\tau, x(\tau)) - f(\tau, x'(\tau))|| d\tau. \]
By Substituting (11)
\[
\begin{align*}
\eta_a - \frac{\beta^a S(t)}{N(t)} (l(t) + k^a M(t)) - \mu^a S(t) &= 0, \\
\beta^a S(t) (l(t) + k^a M(t)) - (\mu^a + \sigma^a + \omega^a) M(t) &= 0, \\
\sigma^a M(t) - (\mu^a + \delta^a + \gamma^a) I(t) &= 0, \\
\omega^a R(t) + \gamma^a I(t) - \mu^a R(t) &= 0.
\end{align*}
\]

On putting \(M(t) = I(t) = 0\) we obtained the disease free equilibrium
\[E^0 = (S^0, M^0, I^0, R^0) = \left( \frac{\eta_t}{\mu^a}, 0, 0, 0 \right).\]
In addition \(N^0 = \bar{S}\)
Considering \(M(t) \neq 0, I(t) \neq 0\), in (11)-(14) we find the endemic equilibrium,
\[E^* = (S^*, M^*, I^*, R^*)\]

\[S^* = \frac{1}{\mu^a} \left[ \eta_t - \frac{(\mu^a + \sigma^a + \omega^a)(\mu^a + \delta^a + \gamma^a)}{\sigma^a} \right] \Gamma, \]
\[M^* = \frac{(\mu^a + \delta^a + \gamma^a)}{\sigma^a} \Gamma, \]
\[R^* = \frac{1}{\mu^a} \left[ \gamma^a + \frac{\omega^a (\mu^a + \sigma^a + \omega^a)}{\sigma^a} \right] \Gamma, \]
\[\Gamma = \frac{\eta^a \sigma^a}{(\mu^a + \sigma^a + \omega^a)(\mu^a + \delta^a + \gamma^a)} \frac{\mu^a \sigma^a N^*}{\beta^a \sigma^a \Gamma^*}, \]

where,
\[N^* = \frac{\eta^a R_0 \left( \mu^a + \omega^a \right) \left( \mu^a + \delta^a + \gamma^a \right) + \sigma^a \left( \mu^a + \delta^a \right)}{\left( R_0 - 1 \right) \left( \mu^a + \sigma^a + \omega^a \right) \left( \mu^a + \delta^a + \gamma^a \right) + \left( \mu^a + \omega^a \right) \left( \mu^a + \delta^a + \gamma^a \right) + \sigma^a \left( \mu^a + \delta^a \right)} \]

4.2. Local stability

Consider system (1) then, we have the following Jacobian matrix
\[J = \begin{bmatrix}
-\frac{\beta^a}{N(t)} (l(t) + k^a M(t)) - \mu^a & -\beta^a k^a & -\beta^a k^a & 0 \\
0 & -\mu^a - \sigma^a - \omega^a & 0 & 0 \\
0 & \sigma^a & -\mu^a - \delta^a & 0 \\
0 & 0 & \omega^a & -\mu^a - \gamma^a - \mu^a \\
\end{bmatrix} \]

Theorem 4. The disease free equilibrium \(E^0\) is locally asymptotically stable.
Proof.
\[J_{E^0} = \begin{bmatrix}
-\mu^a & -\beta^a k^a & -\beta^a k^a & 0 \\
0 & -\mu^a - \sigma^a & 0 & 0 \\
0 & \sigma^a & -\mu^a - \delta^a & 0 \\
0 & 0 & \omega^a & -\mu^a - \gamma^a - \mu^a \\
\end{bmatrix} \]

The eigenvalues of the characteristics matrix \(J_{E^0}\) are
\[\lambda_1 = \lambda_2 = -\mu^a, \]
\[\lambda_3, 4 = \frac{1}{2} \left[ -2\mu^a + \sigma^a + \omega^a + \delta^a + \gamma^a - \beta^a k^a \right] \pm \sqrt{\left(2\mu^a + \sigma^a + \omega^a + \delta^a + \gamma^a - \beta^a k^a\right)^2 - 4\left(\mu^a + \delta^a + \gamma^a\right)(\mu^a + \sigma^a + \omega^a - \beta^a k^a) + 4\beta^a \sigma^a}. \]

Clearly, all the eigenvalues have zero imaginary part \(\text{Im}(\lambda_i) = 0, i = 1, 2, 3, 4\).
Hence by theorem 1 above, the disease free equilibrium \(E^0\) is locally asymptotically stable.

4.2.1. Basic Reproduction Number
To obtain basic reproduction number which is a key parameter describing the number of secondary infections generated by a single infectious individual, we consider the eigen values above.

Clearly \(\lambda_1 < 0, \lambda_2 < 0, \lambda_3 < 0\).
For \(\lambda_4 < 0\) implies that
\[\beta^a \sigma^a - (\mu^a + \delta^a + \gamma^a)(\mu^a + \sigma^a + \omega^a - \beta^a k^a) < 0. \]

Or
\[\frac{\beta^a \sigma^a + k^a (\mu^a + \delta^a + \gamma^a)}{(\mu^a + \sigma^a + \omega^a)(\mu^a + \delta^a + \gamma^a)} < 1. \]

This threshold quantity which if less than one disease free equilibrium will be stable and if greater than one it is unstable is what we termed as basic reproduction ratio \(R_0\). Hence we define
\[R_0 = \frac{\beta^a \sigma^a + k^a (\mu^a + \delta^a + \gamma^a)}{(\mu^a + \sigma^a + \omega^a)(\mu^a + \delta^a + \gamma^a)}. \]

Theorem 5. The endemic equilibrium is stable if \(R_0 > 1\).
Proof.

The endemic equilibrium points can be rewritten in the form of

\[ I^* = \frac{\eta_0 \sigma \alpha}{(\mu_0 + \sigma_0 + \alpha_0)(\mu_0 + \delta_0 + \gamma_0)} \]

\[ - \frac{\beta_0 \sigma_0 \mu_0 N_0}{(\mu_0 + \sigma_0 + \alpha_0)(\mu_0 + \delta_0 + \gamma_0)} \]

\[ = \frac{1}{\mu_0} \left[ \eta_0 - \frac{\eta_0 \mu_0 N_0}{R_0} \right] \]

\[ = \frac{1}{\mu_0} \left[ \eta_0 - \frac{\eta_0 \mu_0 N_0}{R_0} \right] \]

\[ = \frac{1}{\mu_0} \left[ \eta_0 - \frac{\eta_0 \mu_0 N_0}{R_0} \right] \]

\[ \lambda_1 = -\mu_0. \]

To determine the nature of the eigenvalues \( \lambda_1, \lambda_2, \lambda_3 \) in

\[ a_0 \lambda_1^3 + a_1 \lambda_2^2 + a_2 \lambda_2 + a_3 = 0, \]

then \( \lambda_2 > 0 \).

Lastly, for \( \lambda_3 = a_3 \), if

\[ R_0 > \frac{\mu_0 N_0}{\eta_0}, \]

then \( \lambda_3 = a_3 > 0 \).

Now since all the conditions depend on the magnitude of \( R_0 \) and this equilibrium solution only exist when \( R_0 > 1 \), then we can conclude that the solution is stable when \( R_0 > 1 \).

4.3. Global Stability

Theorem 8. the positive equilibrium is globally asymptotically stable

Proof.

To derive the Lyapunov candidate function for fractional order as in [24], consider the family of quadratic Lyapunov function

\[ L(x_1, x_2, \ldots, x_n) = \sum_{i=1}^n \frac{C_i}{2} (x_i(t) - x_i)^2. \]

And define the Lyapunov candidate function as

\[ L(S(t), M(t), I(t), R(t)) = \frac{1}{2} (S(t) - S^*)^2 + \frac{1}{2} (M(t) - M^*)^2 \]

\[ + \frac{1}{2} (I(t) - I^*)^2 + \frac{1}{2} (R(t) - R^*)^2. \]

Linearity of Caputo operator gives

\[ \xi D^\alpha_t L(S(t), M(t), I(t), R(t)) = \frac{1}{2} \xi \xi D^\alpha_t L(S(t) - S^*)^2 \]

\[ + \xi D_t^\alpha L(M(t) - M^*)^2 + \xi D_t^\alpha L(I(t) - I^*)^2 + \xi D_t^\alpha L(R(t) - R^*)^2. \]

Applying Lemma 2 [23] above

\[ \xi D_t^\alpha L(S(t), M(t), I(t), R(t)) \leq \xi D_t^\alpha L(S(t) - S^*) \]

\[ + \xi D_t^\alpha L(M(t) - M^*) + \xi D_t^\alpha L(I(t) - I^*) + \xi D_t^\alpha L(R(t) - R^*) \]

\[ = \xi a_0 - \mu_0 (S(t) - S^*) + M(t) - M^* + I(t) - I^* + R(t) - R^* \]

\[ = \xi \xi - \mu_0 (S(t) - S^*) \]

\[ \xi (I(t) - I^*). \]

To obtain a disease-free equilibrium \( (S(t) = S^*, M(t) = M^*, I(t) = I^*, R(t) = R^*) \).

Case 1: Clearly, substituting the disease free equilibrium \( E_0^0 \) into (28) yields

\[ \xi D_t^\alpha L(S(t), M(t), I(t), R(t)) \leq 2 \xi a_0 - \mu_0 N(t) - \delta_0 I(t). \]

Therefore

\[ \xi D_t^\alpha L(S(t), M(t), I(t), R(t)) \leq -W(x(t)), \]

where

\[ W(x(t)) = \mu_0 N(t) + \delta_0 I(t) - 2 \xi a_0. \]

Hence by Theorem 3 above, the disease free equilibrium is globally asymptotically stable.

Case 2: At the endemic (positive) equilibrium, (28) becomes

\[ \xi D_t^\alpha L(S(t), M(t), I(t), R(t)) \leq \xi a_0 + \mu_0 N_0 \]

\[ \xi \delta_0 - \mu_0 N(t) - \delta_0 I(t). \]

Hence by Theorem 3 above, the disease free equilibrium is globally asymptotically stable.
\[
\begin{align*}
\delta^\alpha \sigma - \delta^\alpha \sigma^c < & \frac{(\mu^c + \alpha^c + \omega^c)(\mu^c + \delta^c + \gamma^c)}{\delta^\alpha \sigma + \omega^c + \alpha^c + \delta^c + \gamma^c} < 1. \\
\xi \partial_t^\alpha (S(t), M(t), I(t), R(t)) = 2\eta^c - \mu^c N(t) - \delta^c I(t),
\end{align*}
\]

where \(W(x(t)) = \mu^c N(t) + \delta^c I(t) - 2\eta^c\).

Hence by theorem 3 above, the endemic equilibrium is globally asymptotically stable.

5. Numerical simulation

In this chapter we carry out numerical examples to study the analytic results using parameter values in Table 1. For the variables we use \(S(t) = 1.0 \times 10^7, M(t) = 4 \times 10^3, I(t) = 1 \times 10^3\) and \(R(t) = 1.5 \times 10^2\).

From Fig. 2, we can see the dynamics of the three important populations. In Fig. 3, 4 and 5 each of these populations was treated separately using various values of \(\alpha\). The values of \(\alpha\) used are 0.2, 0.4, 0.6, 0.8 and 1.0. We can see that in Fig. 3, there is no wide difference between the populations of infected individuals for the different values of \(\alpha\). For Fig. 4, there were intersections between the populations of mild individuals. For Fig. 5, there were wide differences between the populations of recovered individuals for the various \(\alpha\) values. Lastly, from Fig. 6, we can see the effect of mild cases on the population of infected individuals. When the rate of infection of the mild cases increases, there is equivalent increase in the overall population of infected individuals. Hence to curtail the spread of the disease there is need to take care of the Mild cases as well.

6. Conclusion

In mostly poor and underdeveloped territories where there is no capacity of rapid diagnostic COVID-19 test due to insufficiency of testing kits, the Mild cases (who usually show no symptoms of the infection due to their strong and active immune system up to their recovery period) play a major role as a route that spread the disease to healthy individuals. Here we build our model by incorporating the population of mild individuals into the compartmental SIR model to become SMIR model in form of system of fractional order differential equations (FODE) in the Caputo sense.

The existence of the solutions of the model was shown by solving the fractional Gronwall’s inequality using the Laplace transform approach. Two equilibrium solutions, disease free and endemic were obtained. Both local and global stability of the equilibria were shown to depend on the magnitude of basic reproduction ratio. Numerical simulations were carried out and dynamics of the populations were shown to vary for different values of \(\alpha\). It was also shown that when the rate of infection of the mild cases increases, there is equivalent increase in the overall population of infected individuals. Hence to curtail the spread of the disease there is need to take care of the Mild cases as well.

Declaration of Competing Interest

We write to declare our interest in publishing our work titled “Fractional Order Model for the Role of Mild Cases in the Transmission of COVID-19” with the journal “Chaos, Solitons and Fractals”.

CRediT authorship contribution statement

I. A. Baba: Visualization, Investigation, Supervision, Software, Validation, Writing - review & editing. Bashir Ahmad Nasidi: Conceptualization, Methodology, Software, Data curation, Writing - original draft.

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