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Some remarks on an arbitrary-order SIR model constructed with Mittag-Leffler distribution

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Abstract. Our recent works discuss the meaning of an arbitrary-order SIR model. We claim that arbitrary-order derivatives can be obtained through special power-laws in the infectivity and removal functions. This work intends to summarize previous ideas and show new results on a meaningful model constructed with Mittag-Leffler functions. We emphasize the tricky idea to deal with equilibria, the nonlocality of the model and the non-intuitive behavior near the lower terminal.

Keywords: Fractional Calculus. Epidemiological model. Mittag-Leffler functions. Nonlocality. Non-intuitive behaviors.

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1 Introduction

"Mathematics is biology's next microscope, only better; biology is mathematics' next physics, only better" [1]. The quote by biomathematician

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Joel E. Cohen portrays the current development of mathematical biology, a branch that holds the attention of several researchers and has been on the agenda every day during the current COVID-19 pandemic. The wide use of mathematical models in situations related to biology does not exhaust their study. Contrariwise, some mathematical tools need a revision to be used properly.

A great tool for problem modeling is Arbitrary-Order Calculus, known as Fractional Calculus. In the most used definitions, there is the possibility of explicitly considering the dependence of previous stages of the phenomenon studied, through the nonlocality of the operators. This is generally related to the "memory effect" [2]. The recent explosion of publications in Fractional Calculus highlights its immense applicability in numerous areas (see, for instance, the data collected in [2]). However, the basis is still not unified and coherent.

Compartmental models, for example, have been widely studied with arbitrary orders. Generally, they are obtained by replacing an integer derivative with an arbitrary-order one. Throughout our research, we publish some works about meaning difficulty, loss of properties and the lack of the construction of fractional SIR type models ([3], [4], [5]). In this context, we study a model proposed by Angstmann, Henry and McGann [6] in which the arbitrary-order derivatives are obtained by construction, considering Mittag-Leffler functions and generalizing the infectivity and remotion functions. We seek to extend some analytical and numerical results of the model in [4], [7], [8], [9].

Here, one of the aims is, after some preliminary presentations, discuss the idea around how to find equilibria in the proposed model, since equating the right side of the system to zero is no longer a viable strategy. The discussed strategy is somewhat trickier, using Laplace transform techniques. After that, the main propose deals with two points that were not discussed yet: as one would expect, the model is nonlocal and, moreover, presents a non-intuitive behavior in the lower terminal.

2 A brief note about nonlocality and the Fractional Calculus

The impetus theory, studied by names as Leonardo Da Vinci, deals with the concept of "impression". According to Da Vinci, the impression is maintained during a certain time in its sensitive object. This impression (memory) characterized by a long time produces more lasting effects, while a short memory produces effects that occur in shorter times [10]. Although this reasoning was lost in the context of the Newtonian physics, in the last century the quantum theory and the modern string theory allowed a revival of nonlocal theories. Within that context, the Fractional Calculus can be seen as a continuation or a sublimation of nonlocal concepts [11].

Broadly speaking, the Fractional Calculus, parallel to the delay differential equations, has been shown to be a very useful tool in capturing the dynamics of the physical process of several scientific objects. Probably, it was born in 1695, when l'Hôpital asked Leibniz about the meaning of a derivative of order $1/2$. Over the subsequent centuries, important advances were made by Liouville, Riemann, Grünwald, Caputo, and many others. However, it was only after the first International Conference on Fractional Calculus and Applications, in 1974, that the number of researchers in Fractional Calculus showed great growth. The reader may refer to the reference [12] for a detailed chronology of publications in Fractional Calculus until 2019, as well as for general results.

Nonlocal operators can be constructed in different ways, depending on the bias worked on. Here, before introducing the arbitrary-order integral, we recall the concept of the integer-order integral, sometimes called the multiple or iterated integral:

Definition 2.1 (Integer-order iterated integral)**.** The integral of order $n \in \mathbb{N}$ is defined by the expression

$$I^n f(t) = \int_0^t \int_0^{t_1} \int_0^{t_2} \cdots \int_0^{t_{n-2}} \int_0^{t_{n-1}} f(t_n) dt_n dt_{n-1} \cdots dt_3 dt_2 dt_1. \tag{2.1}$$

By definition, $I^0 f(t) = f(t)$.

The next result, using the Laplace convolution, is a starting point for the generalization of the concept of an integral of order n. For that, we define:

Definition 2.2 (Gel'fand-Shilov function)**.** Let $\alpha \in \mathbb{R}$, $\alpha > 0$. The Gel'fand-Shilov function is defined as

$$\phi_\alpha(t) = \begin{cases} \dfrac{t^{\alpha-1}}{\Gamma(\alpha)} & \text{if } t \geq 0, \\ 0 & \text{if } t < 0, \end{cases} \tag{2.2}$$

where Γ represents the gamma function.

Theorem 2.3. *Let $n \in \mathbb{N}$, $0 < t < \infty$ and $f(t)$ be an integrable function. Then,*

$$I^n f(t) = \phi_n(t) \star f(t) = \int_0^t \frac{(t-\tau)^{n-1}}{(n-1)!} f(\tau) d\tau, \tag{2.3}$$

where $\star$ indicates convolution [13].

Thus, it is to be expected that the definition of an integral of arbitrary order α is given by $I^\alpha f(t) = \phi_\alpha(t) \star f(t)$. Below, we consider $[a, b]$ a finite real interval, and α a real number such that $0 \leq n - 1 < \alpha < n$, with n integer:

Definition 2.4 (Riemann-Liouville integral in finite intervals)**.** The Riemann-Liouville integral of an arbitrary order α is set to $t \in [a, b]$ by

$$I^\alpha_{a+} f(t) = \frac{1}{\Gamma(\alpha)} \int_a^t (t-\theta)^{\alpha-1} f(\theta) d\theta. \tag{2.4}$$

After introducing the arbitrary-order integral, it is natural to search for the definition of the corresponding derivative. There are several definitions of these kind of derivatives, each one constructed with a particular viewpoint. In this work, we use the Riemann-Liouville's one:

Definition 2.5 (Riemann-Liouville derivative in finite intervals)**.** The Riemann-Liouville derivative of an arbitrary order α is set to $t \in [a, b]$ by

$$D^\alpha_{a+} f(t) = D^n [I^{n-\alpha}_{a+} f(t)] = \frac{1}{\Gamma(n-\alpha)} \left(\frac{d^n}{dt^n}\right) \int_a^t (t-\theta)^{n-\alpha-1} f(\theta) d\theta\,, \tag{2.5}$$

with D^n representing the integer-order derivative.

Finally, we present the Mittag-Leffler functions with one, two, and three parameters. The classic Mittag-Leffler function, due to its importance in several arbitrary-order differential equations, was nicknamed the "queen of special functions" of the Fractional Calculus. Its importance for Fractional Calculus is analogous to the significance of the exponential function for classical Calculus. We present the following definition [12]:

Definition 2.6 (Mittag-Leffler function with one, two, and three parameters)**.** Let z be a complex number, and three parameters α, β complex, and ρ real, such that $Re(\alpha) > 0, Re(\beta) > 0, \rho > 0$. We define the Mittag-Leffler function with three parameters through the power series

$$E^{\rho}_{\alpha,\beta}(z) = \sum_{k=0}^{\infty} \frac{(\rho)_k}{\Gamma(\alpha k + \beta)} \frac{z^k}{k!}, \tag{2.6}$$

where $(\rho)_k$ is the Pochhammer symbol, defined by $(\rho)_k = \Gamma(\rho + k)/\Gamma(\rho)$.

The three-parameter Mittag-Leffler function is also called Prabhakar function. Particularly, when $\rho = 1$, we have $(\rho)_k = k!$. In this case, the definition recovers the two-parameter Mittag–Leffler function, denoted simply by $E^1_{\alpha,\beta}(t) = E_{\alpha,\beta}(t)$. When $\rho = \beta = 1$, we obtain the classic Mittag-Leffler function, denoted by $E^1_{\alpha,1}(t) = E_{\alpha,1}(t) = E_{\alpha}(t)$. Finally, we recover the exponential function when $\alpha = \beta = \rho = 1$.

3 The model

We present in [4] a physical derivation following the steps of Angstmann, Henry & McGann [6], which use the probabilistic language of Continuous Time Random Walks (CTRW), and Mittag-Leffler functions. As we can see with more detail in the references, the first idea is to consider an individual infected since the time t'. If there are $S(t)$ susceptible in time t, this infected person has a probability $S(t)/N$ that his contact is susceptible, considering the population homogeneous. Therefore, in the period of t to $t + \Delta T$, the expected number of new infections per infected

individual is given by $\sigma(t,t')S(t)\Delta T/N$. The transmission rate per infectious individual $\sigma(t,t')$ depends on both the age of the infection, $t-t'$, and the present time, t. The probability that an individual infected at the moment t' is still infected at the moment t is given by the survival function $\Phi(t,t')$. Therefore, the flux of individuals to the I compartment at a time t is recursively given by

$$q^+(I,t) = \int_{-\infty}^{t} \sigma(t,t')\frac{S(t)}{N}\Phi(t,t')q^+(I,t')dt'. \tag{3.1}$$

To deal with the individuals infected at the time 0, we consider the time in which each individual has become infected. This is given by the function $i(-t',0)$ which represents the number of individuals who are still infectious at time 0 and who were originally infected at some point earlier $t'<0$. Then, $q^+(I,t') = i(-t',0)/\Phi(0,t')$ for $t'<0$. For simplicity, we consider $i(-t,0) = i_0\delta(-t)$, where $\delta(t)$ is the Dirac delta function. So,

$$q^+(I,t) = \int_{0}^{t} \sigma(t,t')\frac{S(t)}{N}\Phi(t,t')q^+(I,t')dt' + i_0\sigma(t,0)\frac{S(t)}{N}\Phi(t,0). \tag{3.2}$$

As said, the infection rate $\sigma(t,t')$ is assumed to be a function of both the current time (due, for example, to containment measures), having an extrinsic infectivity, ω, and the age of infection, $t-t'$, having an intrinsic infectivity, ρ. So, we can write

$$\sigma(t,t') = \omega(t)\rho(t-t'). \tag{3.3}$$

Assuming that the natural death and the removal of an infected individual are independent processes, we can write the survival function as

$$\Phi(t,t') = \phi(t-t')\theta(t,t'), \tag{3.4}$$

where $\phi(t-t')$ is the probability that an individual infected since t' has not yet recovered or been killed by the disease at time t. Also, $\theta(t,t')$ is the probability that an infected individual since t' has not yet died of natural death (that is, independent of the disease) until time t. The θ function is given by $\theta(t,t') = e^{-\int_{t'}^{t}\gamma(u)du}$, where γ is the death rate.

We define infectivity and recovery memory kernels

$$K_I(t) = \mathcal{L}^{-1}\left\{\frac{\mathcal{L}\{\rho(t)\phi(t)\}}{\mathcal{L}\{\phi(t)\}}\right\} \quad , \quad K_R(t) = \mathcal{L}^{-1}\left\{\frac{\mathcal{L}\{\psi(t)\}}{\mathcal{L}\{\phi(t)\}}\right\}, \tag{3.5}$$

where $\psi(t) = -d\phi(t)/dt$. This ψ has an important relationship with the continuous random variable X that provides the time of removal of the individual from the infectious compartment. The cumulative distribution of X, namely F defined by $F(t) = P(X \leq t)$, is such that $F(t) = 1 - \phi(t)$. Therefore, the probability density function of X is $\psi(t) = -d\phi(t)/dt$. We can state the set of equations for the SIR model in a similar manner to that written originally by Kermack and McKendrick [14]:

$$\frac{dS(t)}{dt} = \gamma(t)N - \omega(t)\frac{S(t)}{N}\theta(t,0)\int_0^t K_I(t-t')\frac{I(t')}{\theta(t',0)}dt' - \gamma(t)S(t), \tag{3.6}$$

$$\frac{dI(t)}{dt} = \omega(t)\frac{S(t)}{N}\theta(t,0)\int_0^t K_I(t-t')\frac{I(t')}{\theta(t',0)}dt' - \theta(t,0)\int_0^t K_R(t-t')\frac{I(t')}{\theta(t',0)}dt' - \gamma(t)I(t), \tag{3.7}$$

$$\frac{dR(t)}{dt} = \theta(t,0)\int_0^t K_R(t-t')\frac{I(t')}{\theta(t',0)}dt' - \gamma(t)R(t), \tag{3.8}$$

where we consider the same rate $\gamma(t)$ of natural mortality in each compartment, with the birth rate equal to that. The population remains constant.

We choose $\psi(t)$ and $\rho(t)$ using Mittag-Leffler functions, in order to generalize the exponential distribution of the random variable X and allow a variable intrinsic infectivity:

$$\phi(t) = E_{\alpha,1}\left(-\left(\frac{t}{\tau}\right)^{\alpha}\right) \quad , \quad \rho(t) = \frac{1}{\phi(t)}\frac{t^{\beta-1}}{\tau^{\beta}}E_{\alpha,\beta}\left(-\left(\frac{t}{\tau}\right)^{\alpha}\right). \tag{3.9}$$

Using Laplace transform techniques, the Riemann-Liouville derivatives arise along the construction and the SIR model, with $1 \geq \beta \geq \alpha > 0$, is given by

$$\frac{dS(t)}{dt} = \gamma(t)N - \frac{\omega(t)S(t)\theta(t,0)}{N\tau^{\beta}}D^{1-\beta}\left(\frac{I(t)}{\theta(t,0)}\right) - \gamma(t)S(t), \tag{3.10}$$

$$\frac{dI(t)}{dt} = \frac{\omega(t)S(t)\theta(t,0)}{N\tau^{\beta}}D^{1-\beta}\left(\frac{I(t)}{\theta(t,0)}\right) - \frac{\theta(t,0)}{\tau^{\alpha}}D^{1-\alpha}\left(\frac{I(t)}{\theta(t,0)}\right) - \gamma(t)I(t), \tag{3.11}$$

$$\frac{dR(t)}{dt} = \frac{\theta(t,0)}{\tau^{\alpha}}D^{1-\alpha}\left(\frac{I(t)}{\theta(t,0)}\right) - \gamma(t)R(t), \tag{3.12}$$

Notice that, if $\alpha = \beta = 1$, and $\gamma(t) \equiv \gamma, \omega(t) \equiv \omega$ are considered constant, we get the simple integer-order SIR model with constant coefficients. Moreover, the cumulative distribution of X is a Mittag-Leffler distribution $F(t;\alpha,\tau) = 1 - E_\alpha\left(-(t/\tau)^\alpha\right)$. If $\alpha = \beta = 1$, we have an exponential distribution and the expectation (first moment) of the random variable X exists, with τ being exactly the average recovery time. When $\alpha < 1$, we do not have finite expectation.

Remark 3.1. In epidemics such as COVID-19, reports exhibit the asymmetry of each infectious wave: "while COVID-19 accelerates very fast, it decelerates much more slowly. In other words, the way down is much slower than the way up" [15]. In addition, scientists suggest that infected people are most infectious immediately before they develop symptoms and at the onset of them (e.g. [16]). The two factors presented, that is, the asymmetry of the data, with a heavy right tail effect, and the decrease in infectivity over time since infection, are captured by the arbitrary-order model presented. As started in [4], [17], [18], we have been working on applications of the model to COVID-19.

3.1 Equilibrium

Here, we analyze the equilibrium point (S^*, I^*, R^*) such that $\lim_{t\to\infty}(S, I, R) = (S^*, I^*, R^*)$, where the limit is taken coordinate by coordinate. We consider $\gamma(t) \equiv \gamma$ constant, so $\theta(t, 0) = e^{-\gamma t}$. Taking the limit $t \to \infty$, the model reduces to:

$$0 = \gamma N - \lim_{t\to\infty}\left(\frac{\omega(t)S(t)e^{-\gamma t}}{N\tau^\beta} D^{1-\beta}(I(t)e^{\gamma t})\right) - \gamma S^*, \tag{3.13}$$

$$0 = \lim_{t\to\infty}\left(\frac{\omega(t)S(t)e^{-\gamma t}}{N\tau^\beta} D^{1-\beta}(I(t)e^{\gamma t}) - \frac{e^{-\gamma t}}{\tau^\alpha} D^{1-\alpha}(I(t)e^{\gamma t})\right) - \gamma I^*, \tag{3.14}$$

$$0 = \lim_{t\to\infty}\left(\frac{e^{-\gamma t}}{\tau^\alpha} D^{1-\alpha}(I(t)e^{\gamma t})\right) - \gamma R^*. \tag{3.15}$$

To calculate the limits of the form $\lim_{t\to\infty} e^{-\gamma t} D^{1-\alpha}(I(t)e^{\gamma t})$, considering $\gamma > 0$, we follow [19] and take the Laplace Transform:

$$\mathcal{L}\{e^{-\gamma t} D^{1-\alpha}(I(t)e^{\gamma t})\} = (s+\gamma)^{1-\alpha}\mathcal{L}\{I\}. \tag{3.16}$$

Using a Taylor series expansion, we get

$$(s+\gamma)^{1-\alpha}\mathcal{L}\{I\} = \mathcal{L}\{I\}(\gamma^{1-\alpha} + (1-\alpha)\gamma^{-\alpha}s + O(s^2)). \tag{3.17}$$

As the Laplace Transform is a linear operator, we can invert term by term, obtaining

$$e^{-\gamma t}D^{1-\alpha}(I(t)e^{\gamma t}) = \gamma^{1-\alpha}I(t) + (1-\alpha)\gamma^{-\alpha}\frac{dI}{dt} + \mathcal{L}^{-1}(O(s^2)). \tag{3.18}$$

As we consider $\lim_{t\to\infty} I(t) = I^*$, we have

$$\lim_{t\to\infty} dI/dt = \lim_{t\to\infty} \mathcal{L}^{-1}(O(s^2)) = 0. \tag{3.19}$$

So, it follows that

$$\lim_{t\to\infty} e^{-\gamma t}D^{1-\alpha}(I(t)e^{\gamma t}) = \gamma^{1-\alpha}I^*. \tag{3.20}$$

Substituting these results into Eq. (3.13)-(3.15) and assuming that $\lim_{t\to\infty} \omega(t) = \omega^*$ (possibly $\omega^* = 0$), we are left with

$$0 = \gamma N - \left(\frac{\omega^* S^*}{N\tau^\beta}\gamma^{1-\beta}I^*\right) - \gamma S^*, \tag{3.21}$$

$$0 = \left(\frac{\omega^* S^*}{N\tau^\beta}\gamma^{1-\beta}I^*\right) - \frac{1}{\tau^\alpha}\gamma^{1-\alpha}I^* - \gamma I^*, \tag{3.22}$$

$$0 = \frac{1}{\tau^\alpha}\gamma^{1-\alpha}I^* - \gamma R^*. \tag{3.23}$$

These equations make it possible to obtain a disease-free state:

$$S^* = N, \quad I^* = 0, \quad R^* = 0, \tag{3.24}$$

and, in the case where $\omega^* > 0$, we also obtain an endemic state:

$$S^* = \frac{((\tau\gamma)^{\beta-\alpha} + (\tau\gamma)^\beta)N}{\omega^*}, I^* = \frac{N(\tau\gamma)^\alpha}{1+(\tau\gamma)^\alpha} - \frac{N(\tau\gamma)^\beta}{\omega^*}, R^* = \frac{N}{1+(\tau\gamma)^\alpha} - \frac{N(\tau\gamma)^{\beta-\alpha}}{\omega^*}. \tag{3.25}$$

When $\omega(t) \equiv \omega$, we get $\omega^* = \omega$ and recover the endemic state of the original article [6]. We observe that the endemic state makes physical sense only if we can have $I^* > 0$ and $R^* > 0$, that is, if

$$\omega^* > (\tau\gamma)^{\beta-\alpha} + (\tau\gamma)^\beta. \tag{3.26}$$

We have thus proven that, if there are asymptotically stable equilibria in the case $\gamma > 0$, then they are given by Eq. (3.24)-(3.25). However, we have not really proved that these states are asymptotically stable equilibria. We expect the disease-free state to be an asymptotically stable equilibrium when $\omega^* < (\tau\gamma)^{\beta-\alpha} + (\tau\gamma)^{\beta}$, while the endemic state must be asymptotically stable if $\omega^* > (\tau\gamma)^{\beta-\alpha} + (\tau\gamma)^{\beta}$.

For the case without vital dynamics, that is, $\gamma = 0$, if $\alpha = \beta$ and $\omega(t) \equiv \omega$, we can write $dS/dR = -(\omega S(t)/N\tau)$, as in the original model, also discussed in [4]. Thus, the equilibrium point is the same as in the original case, as we state in [7]. For the case with $\gamma > 0$, there are difficulties to analyze formally the stability. Some advances were also reported in [7].

4 Main remarks

There are several numerical methods that can be applied to arbitrary-order derivatives. For now, we build a numerical $L1$-scheme [20] to discretize the model described in the last Section. The time interval $[a, t]$ is discretized as $a = t_0 < t_1 < \cdots < t_n = t$, where the time steps $\Delta T_i = t_{i+1} - t_i$, for $i \in \{0, \cdots, n-1\}$, have the same size ΔT. Considering $\alpha \in (0, 1]$, we perform the following discretization for the Riemann-Liouville derivative:

$$D_{a+}^{1-\alpha} f(t_j) \simeq \frac{\Delta T^{\alpha-1}}{\Gamma(\alpha+1)} \left[\sum_{k=0}^{j-1} f(t_k)[(j-k+1)^{\alpha} - 2(j-k)^{\alpha} + (j-k-1)^{\alpha}] + f(t_j) \right], \quad (4.1)$$

where $t_i = i\Delta T + t_0$ for all $i \in \{0, 1, \cdots, n\}$. It is important to state that the integer-order case is obtained by taking $\alpha = 1$.

4.1 Nonlocality

We illustrate that the model (3.10)-(3.12) is nonlocal, as expected. For this, we consider $N = 10^6$ and initial conditions $S(0) = N - 1$, $I(0) = 1$, $R(0) = 0$. At time $t = 90$, the numerical solution gives $S(90) = 205890$, $I(90) = 267840$, and $R(90) = 526270$. We now consider this initial condition and run the model again.

The Figure 4.1 illustrates the change of the solution, where the dashed line corresponds to the solution from the time $t = 90$. In Figure 4.2, we have the equivalent trajectories for a maximum time $T = 3000$. The equilibrium is maintained.

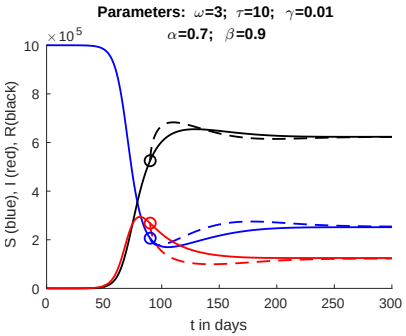

Figure 4.1: Change in the solution.

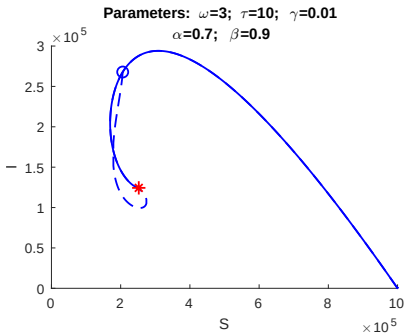

Figure 4.2: Change in the trajectory.

Remark 4.1. In the integer-order SIR model, the epidemiological meaningful parameters define the epidemic independently of time. Given an initial condition $(S(0), I(0), R(0))$, and the solution $(S(t), I(t), R(t))$ of the classic SIR model, let $t^* > 0$: if we start at time t^*, with initial condition $(S(t^*), I(t^*), R(t^*))$, the solution will continue to be $(S(t), I(t), R(t))$ for $t > t^*$. This property of autonomous dynamical systems is called invariance of solutions [21]. However, this is not valid here, making it difficult to correctly choose initial conditions: what about the past prior to the considered initial point? In this model, to adjust to the infection, the parameters depend on the time series prior to the starting point. In other words, different pasts will lead to different solutions in the future. Starting a modeling in the second month of an epidemic, for example, does not lead to the same result as when we start on the first day. It is worth to mention that, to obtain the Equation (3.2), we used the Dirac delta function to indicate that the disease does not exist before the time 0.

The nonlocality also implies an unexpected behavior of the reproduction number at time t'. It can be understood as the expected number of individuals that are infected by an infectious individual since t', with

the basic reproduction number being the average number of secondary infections that occur when an infectious individual is introduced into a completely susceptible population [22]. In the constructed model, it is not natural to define a formula that provides the reproduction number. Then, in [6] the authors propose a construction for the basic reproduction number through an integral. Here, we extend the proposal of the reproduction number for any time t'. Thus, initially, we consider $\omega(t)$ constant and the definitions

$$\Re_0 = \frac{\omega\gamma^{\alpha-\beta}}{\tau^{\beta}\gamma^{\alpha} + \tau^{\beta-\alpha}}, \tag{4.2}$$

$$\Re(t') = \frac{S(t')\omega}{N\tau^{\beta}} \frac{\gamma^{\alpha-\beta}}{\gamma^{\alpha} + \tau^{-\alpha}} = \Re_0 \cdot \frac{S(t')}{N}. \tag{4.3}$$

This analysis is important, since for this model we do not have parameter tables, nor even prior application to any specific infectious disease. As we can see here, in classical epidemiological models, the peak occurs when $\Re(t) = 1$, but this does not occur in models of arbitrary orders or with time-dependent parameters.

We consider the example below, in Figure 4.3, where the value of $\Re(t')$ given by Eq. (4.3) for the peak point t' is $\Re_{peak} \approx 0.9078 < 1$. If we start modeling at a time before the peak, for which $\Re_{peak} < \Re(t) < 1$, the epidemic will decrease even faster, but after a small rise. Note that $\alpha = \beta$. This will be discussed later. Also, is also possible to have no more peaks if we start in a previous point, as we can see in Figure 4.4. In both examples, we consider $N = 10^6$ and the initial condition is $(N - 1, 1, 0)$.

We also can observe $\Re_{peak} > 1$. In some of these cases, we can take an earlier day, where $\Re(t) > \Re_{peak} > 1$, and the disease starts decreasing even faster, as illustrated in Figure 4.5. In other words, "the point where the epidemic must start to decrease" is really not the peak point anymore! This indicates a difficulty in understanding the $\Re_0$ defined in (4.2): although $\Re_0 > 1$ is the condition of endemic equilibrium viability given in (3.25), $\Re_0 S(0)/N > 1$ does not necessarily indicate that the epidemic will or will not occur, that is, whether or not I will grow.

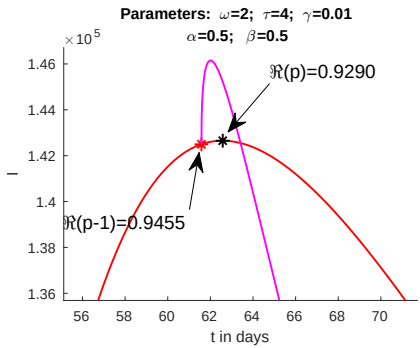

Figure 4.3: Case $\Re(p) < 1$ ($\alpha = \beta$).

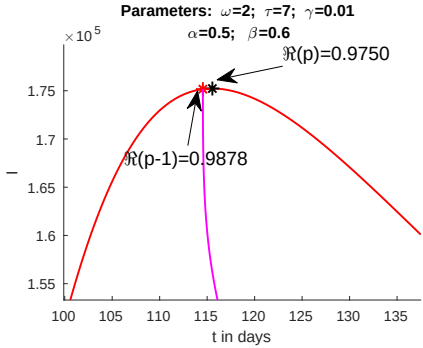

Figure 4.4: Case $\Re(p) < 1$.

In this same Figure 4.5, we consider different starting points along the originally constructed I curve with initial condition $(N-1, 1, 0)$. So, it illustrates the evolution of the I compartment if we start modeling at different starting points along the same curve. The nonlocal behavior of the model is surprising and we see that, in this example, the I compartment only rises if we take a starting point much earlier than the peak. Here, we also start to consider the proposal that we presented in [7] for a S-variable reproduction number:

$$\Re^S(t') = \int_{t'}^{\infty} \frac{S(t)}{N\tau^\beta}\omega(t)e^{-\gamma(t-t')}(t-t')^{\beta-1}E_{\alpha,\beta}\left(-\left(\frac{(t-t')}{\tau}\right)^\alpha\right)dt. \tag{4.4}$$

In this proposal, the basic reproduction number is given by

$$\Re_0^S = \int_0^{\infty} \frac{S(t)}{N\tau^\beta}\omega(t)e^{-\gamma t}t^{\beta-1}E_{\alpha,\beta}\left(-\left(\frac{t}{\tau}\right)^\alpha\right)dt. \tag{4.5}$$

In the Figure table 4.6, we display the $\Re_0 S(0)/N$ and the $\Re_0^S S(0)/N$ for each curve. In classical models, the disease declines since the beginning if and only if $\Re_0 S(0)/N < 1$. If $\Re_0 S(0)/N > 1$, it must grow for a while. As we can see, this does not happen with this model.

However, the equilibrium I^* given in (3.25) is the same for any initial condition. So, broadly speaking, two simulations of the same disease with different starting points lead to different solutions, but to the same end, what is really important to study, for example, how many people will be infected in total. Here we pointed out that, as will be explored in next

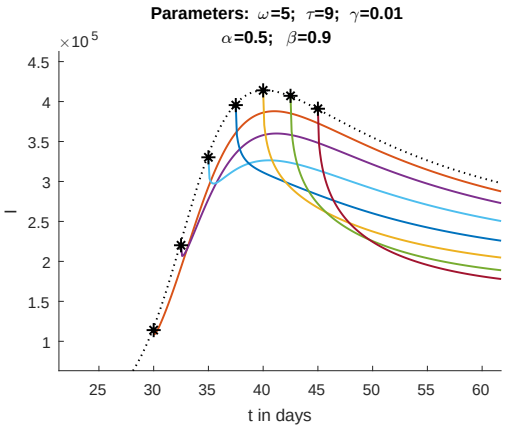

Figure 4.5: Modifying the start point.

	$R_0\, S(0)/N$	$R_0^S\, S(0)/N$
	8.2953	2.1882
	6.5125	1.7069
	4.3990	1.3420
	2.7169	1.1218
	1.6966	1.0103
	1.1490	0.9611
	0.8664	0.9442

Figure 4.6: $\Re_0$ and $\Re_0^S$.

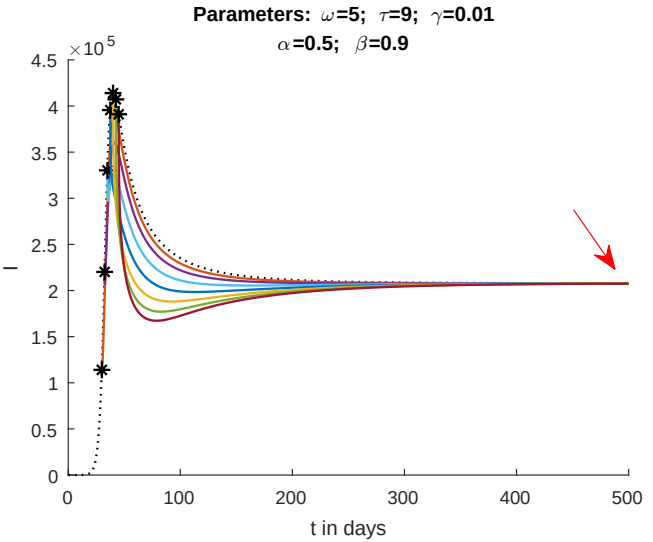

Figure 4.7: The equilibrium remains the same.

works, the $S-$variable reproduction number seems to provide something closer to what we expect, in terms of plausible biological arguments.

4.2 Behavior near lower terminal

Finally, we exhibit an unusual feature observed in the initial instants of the model simulations. One can easily see in the Figure 4.5 that the

blue and purple curves have a small depression before a rise. This type of behavior does not appear in the integer-order model.

In fact, this can be explained given the asymptotic behavior of the Riemann-Liouville derivatives near the lower terminal [23]:

$$D^{\alpha}_{a+}f(t) \approx \frac{f(a)}{\Gamma(1-\alpha)}(t-a)^{-\alpha}, \qquad (t \to a+). \tag{4.6}$$

So, in the model (3.10)-(3.12), if $\beta > \alpha$, we have $dI/dt < 0$ for t small enough, as illustrated in the Figures 4.8-4.9.

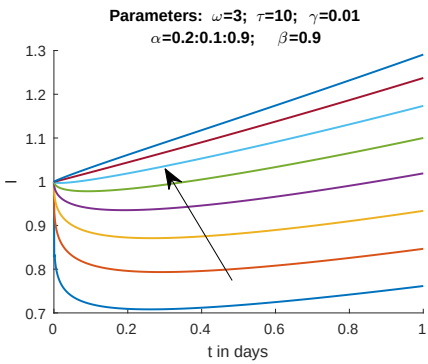

Figure 4.8: Lower terminal - Ex.1.

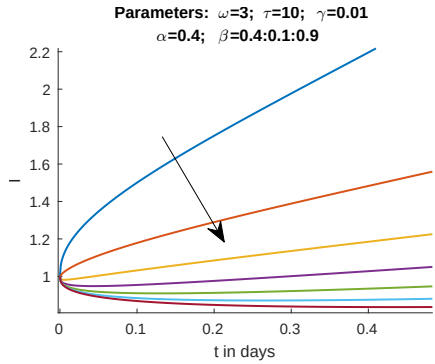

Figure 4.9: Lower terminal - Ex.2.

When $\alpha = \beta$, this behavior is not observed. For instance, we can see in the pink curve of the Figure 4.3 that the behavior in the lower terminal is the traditional movement of rising to a peak and decreasing after that.

Here, we also emphasize that the model assumes that there was no disease before time 0, and then, at that point, an impulse caused the disease to start. So, the historic is theoretically considered completely, avoiding dubiousness about the starting point. However, the solution is not C^1, as it is not derivable at the origin. These remarks aim to strengthening our intuition about the model and provide insights about its application.

5 Final considerations

So far, we have not been able to find a physical structure that allows us to change the order of the original derivatives, even if the units are

adjusted and the new orders are the same in all compartments. Therefore, we seek the possibility of physically modeling a system, with a formalism similar to that of Kermack and McKendrick, the creators of the SIR model. In this sense, we have been discussing the almost unknown model of [6].

Here, we propagate the technique to work with equilibria in this type of model and present important considerations, not previously explored: the model is nonlocal, presents a difficult regarding the supposed relation between the course of the epidemic and the reproduction number and, finally, there is an unexpected behavior at the initial point. Particularly, start the simulation of an epidemic in its beginning or be able to say about the past is a trick question. We aim that the deeper study of the equilibrium points and trajectories are fundamental predicting important features of the model's application. By other hand, the mathematics of the Fractional Calculus' models is still a black box of surprises that the assembling between analytical and numerical studies can help to investigate.

Future works intend to close some results not fully explored, such as the stability of equilibrium points in the general case and the use of an extrinsic infectivity arising from Mittag-Leffler functions. The proposal of a variable extrinsic infectivity was first illustrated in [4] and made it possible to fine-tune the COVID-19 data, which leads us to new opportunities.

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