A Caputo Fractional Epidemiological MSIR Model

Youssef Difaa, Bouchaib Khajji and Hicham Benaissa

***Abstract*—We introduce a fractional MSIR model employing Caputo fractional derivative. We estimate the basic reproduction number and the model equilibrium states. Furthermore, we analyze its local and global asymptotic stability at the disease-free steady. Lastly, utilising Matlab, we handle simulations to support our findings.**

***Index Terms*—fractional calculus, epidemiological process, epidemiological MSIR model, local stability.**

I. Introduction

CONTAGIOUS illnesses, also known as diseases, are conditions triggered by organisms like bacteria or viruses. Certain infectious diseases can spread through insect stings or direct contact between individuals, while others are contracted from exposition to the surroundings micro-organisms or by consumption of contemned water or food. Every year, millions of individuals lose their lives due to diseases like tuberculosis, measles, and Ebola virus. Infectious maladies persist due to several factors, including inadequate sanitation and healthcare in numerous countries, which create conducive environments for the transmission of infectious agents. Furthermore, the emergence of drug-resistant strains among pathogens poses a significant challenge, potentially precipitating new outbreaks despite existing medical interventions.

The primary objective of abstract models in epidemiology is to explain the dynamics of a specific diseases, including aspects like the spread pattern, epidemic duration, and its repercussions on the population. These models play a crucial role in guiding health authorities towards optimal strategies for managing the epidemic. Such strategies may encompass initiatives like large-scale vaccination campaigns, the administration of antiviral medications, pest management, and disinfection protocols, as well as enforcing isolation and quarantine measures.

Mathematical models serve to represent reality, yet they often necessitate simplifications due to the impracticality of managing numerous input parameters. When examining infectious diseases, certain variables, such as weather conditions, individual diets, other illnesses, and specific types of interactions between individuals, are typically disregarded. Therefore, it becomes crucial to pinpoint the primary variables that exert significant influence on the model. This approach aims to streamline computational complexity while maintaining an accurate depiction of the disease's progression. Typically, an epidemiologic model partitions the total population into distinct compartments, each representing a particular health state regarding the infectious agent of interest. This model is time-dependent, capturing changes in the number of individuals in each compartment as their health conditions progress. This characteristic is particularly significant in infectious diseases characterized by short infectious periods, like measles, chickenpox, rubella, or mumps. In such cases, the birth and death dynamics are commonly disregarded due to the brevity of the timeframe.

A fundamental framework in epidemiological modeling categorizes the population into three distinct health states: Susceptible (S) to the contagious agent, Infected (I) by it, and Immune (R) or Recovered. This structure forms the basis of what is known as the SIR model (see [3], [4]). The original formulation of SIR model was addressed by Kermack et al. [17], significantly shaping the mathematical modeling of disease spreading. Subsequent contributions by these researchers extended the model to incorporate demographic dynamics such as birth and death rates [18], [19]. Over time, various extensions and alternative formulations have emerged. For instance, the Greenwood and Reed-Frost models approach the problem using discrete-time steps [1], [4]. Simpler models, like the SI model, assume no recovery once an individual is infected [29]. More sophisticated versions of the SIR model have been proposed as well, some account for stratification within the susceptible population based on differing infection risks [9], [24], while others allow for multiple infection stages, including both sublethal and lethal forms [6]. Additionally, further refinements to these models include the integration of interventions such as vaccination strategies and antiviral treatments [10], [11], [23]. Consequently, the MSIR model classifies individuals according to

- M : individuals who possess temporary immunity acquired from maternal antibodies;
- S : the susceptible group, comprising those who are vulnerable to infection but have not yet encountered the disease;
- I : individuals currently infected and who may spread the illness to others;
- R : individuals who acquired immunity, i.e., recovered.

Manuscript received April 27, 2024; revised April 19, 2025.

Youssef Difaa is a postgraduate student of Polydisciplinary Faculty of Khouribga, Sultan Moulay Slimane University, Khouribga, 25000, Morocco (E-mail: youssefdifaa21998@gmail.com).

Bouchaib Khajji is a professor of Faculty of Sciences Ben M'Sik, Hassan II University, Sidi Othman, Casablanca, Morocco (E-mail: khajji-bouchaib@gmail.com).

Hicham Benaissa is a professor of Polydisciplinary Faculty of Khouribga, Sultan Moulay Slimane University, Khouribga, 25000, Morocco (E-mail: hi.benaissa@gmail.com).

The structure of an epidemiological model varies based on the specific health compartments it includes. For instance, well-known variations include the SIS model [12], the SIRS model [20], SEIR model [21], and MSEIR model [15], among several others. Lately, fractional derivatives were employed in epidemiological modeling, demonstrating superior accuracy in certain scenarios compared to traditional methods. Various definitions exist for fractional derivatives; however, we opt here to utilize the Caputo derivatives. Employing this derivative offers several benefits, including the ability to incorporate classical initial conditions into the problem formulation. Additionally, unlike other fractional derivatives, the Caputo derivative yields zero when applied to a constant, which enhances its practical utility.

This paper addresses a new approach by presenting a fractional MSIR model, which characterizes the disease's transmission through a set of fractional differential nonlinear equations. One should see that fractional derivatives, being nonlocal functionals, offer a more apt framework for modeling systems with memory dependence on past events. Furthermore, the flexibility in choosing any positive real α for the fractional order allows us to tailor the model to fit empirical data accurately. Consequently, this capability enables us to fine-tune the modeling of real world data, enhancing our ability to forecast the disease's progression effectively.

In the paper: Section 2 gives an overview of the standard MSIR model. In Section 3, the model is extended using tools from fractional calculus. This section focuses on demonstrating the well-posedness of the system, examining equilibrium states, and calculating the basic generation number. Finally, Section 4 showcases numerical simulations performed with Matlab.

II. Classical MSIR model

We assume in the study that individual who recover from the infection, acquire permanent immunity. Additionally, we assume a constant and equal birth and death rate, represented by b, which maintains a stable total population over time, i.e.;

$$M + S + I + R = N.$$

The susceptible class S grows at a rate of bS, as only susceptible mothers can give birth to susceptible offspring (those not protected by maternal antibodies). In contrast, individuals from the other compartments, having been exposed to the disease, are immune, so their newborns, amounting to $b(N - S)$ come in the passive immune category M. Transitions between compartments occur at various rates: individuals leave class M at rate δM, class S at rate μS, and recover from infection at rate γI. Let β be the spread rate, representing the likelihood that a susceptible person becomes infected following effective contact with someone infectious. This gives $\mu = \beta I/N$.

The infection flow is governed by the below ordinary differential system (ODS) (see [13]):

$$\begin{aligned} M'(t) &= b\,(N - S(t)) - (b+\delta)\,M(t),\\ S'(t) &= b\,S(t) + \delta\,M(t)\\ &\quad - \frac{\beta}{N}\,I(t)S(t) - b\,S(t),\\ I'(t) &= \frac{\beta}{N}\,S(t)I(t) - (b+\gamma)\,I(t),\\ R'(t) &= \gamma\,I(t) - b\,R(t). \end{aligned} \tag{1}$$

The time t is expressed in units like months, days or hours, relying on how rapidly the contagion spreads. By using the relation $S = N - I - M - R$, the differential expression for S can be removed. Additionally, introducing the normalized variables $m := M/N$, $r := R/N$ and $i := I/N$ allows for a more compact formulation of the model:

$$\begin{aligned} m'(t) &= b\,(r(t) + i(t)) - \delta m(t),\\ i'(t) &= \beta\,i(t)(1 - m(t) - i(t) - r(t))\\ &\quad - (b+\gamma)\,i(t),\\ r'(t) &= \gamma\,i(t) - b\,r(t). \end{aligned} \tag{2}$$

III. Fractional MSIR model

Building on (1), we introduce a fractional version of the MSIR system. This is achieved by replacing each ordinary derivative with a Caputo derivative of order $\alpha \in (0, 1)$. To maintain dimensional consistency across the equations, each parameter denoted by $\star$ is modified to $\star^\alpha$, except for N, which remains dimensionless, as discussed in [7]. As a result, the model is described by the below nonlinear fractional differential system:

$$\begin{aligned} {}^C D^\alpha_{0+} M(t) &= b^\alpha (N - S(t))\\ &\quad - (b^\alpha + \delta^\alpha) M(t),\\ {}^C D^\alpha_{0+} S(t) &= \delta^\alpha M(t) + b^\alpha S(t)\\ &\quad - \frac{\beta^\alpha}{N} S(t) I(t) - b^\alpha S(t),\\ {}^C D^\alpha_{0+} I(t) &= \frac{\beta^\alpha}{N} I(t) S(t)\\ &\quad - (\gamma^\alpha + b^\alpha) I(t),\\ {}^C D^\alpha_{0+} R(t) &= \gamma^\alpha I(t) - b^\alpha R(t). \end{aligned} \tag{3}$$

It is presupposed that the functions M, I, S and R, along with their Caputo derivatives, maintain continuity. Analogous to the classical interpretation, we have ${}^C D^\alpha_{0+} N(t) = 0$, and this yields $N := M + I + S + R$ remains constant. Consequently, the fractional system (3) can be rephrased, yielding subsequent system:

$$\begin{aligned} {}^C D^\alpha_{0+} m(t) &= b^\alpha\,(r(t) + i(t)) - \delta^\alpha\,m(t)\\ {}^C D^\alpha_{0+} i(t) &= \beta^\alpha\,i(t)(1 - m(t) - i(t) - r(t))\\ &\quad - (\gamma^\alpha + b^\alpha)\,i(t)\\ {}^C D^\alpha_{0+} r(t) &= \gamma^\alpha\,i(t) - b^\alpha\,r(t) \end{aligned} \tag{4}$$

with the initial data

$$m(0) = m_0, i(0) = i_0 \text{ and } r(0) = r_0, \tag{5}$$

where $0 \le m_0, i_0, r_0 \le 1$. The susceptible portion of the population can be determined by

$$s(t) = 1 - r(t) - m(t) - i(t).$$

Remark. *An alternative approach to address the dimen-*

sional inconsistencies arising from substituting ordinary derivatives with fractional derivatives in (2) *is proposed in [8], [26]. This method suggests to multiply the left-hand side of any relation by $\tau^{\alpha-1}$, where τ being a time-constant introduced to restore dimensional balance after the substitution of derivative operators. Applying this adjustment leads to the below fractional system:*

$$\begin{aligned}
\tau^{\alpha-1}\,{}^{C}D^{\alpha}_{0+}\, m(t) &= b\,(r(t)+i(t)) - \delta\, m(t)\\
\tau^{\alpha-1}\,{}^{C}D^{\alpha}_{0+}\, i(t) &= \beta i(t)\,(1-m(t)-i(t)-r(t))\\
&\quad -(b+\gamma)\, i(t)\\
\tau^{\alpha-1}\,{}^{C}D^{\alpha}_{0+}\, r(t) &= \gamma\, i(t) - b\, r(t).
\end{aligned}$$

Theorem III.1. *The problem delineated by* (4)-(5) *admits a unique solution belonging to*

$$[R_0^+]^3 := \mathbb{R}_+ \times \mathbb{R}_+ \times \mathbb{R}_+.$$

Proof: The unique global solvability is established in [22, Theorem 3.1] and [22, Remark 3.2]. To demonstrate its non-negativity, one should examine the subsequent auxiliary fractional differential system:

$$\begin{aligned}
{}^{C}D^{\alpha}_{0+} m(t) &= b^{\alpha}(r(t)+i(t)) - \delta m(t) + \frac{1}{k}\\
{}^{C}D^{\alpha}_{0+} i(t) &= \beta^{\alpha} i(t)(1-i(t)-m(t)-r(t))\\
&\quad -(\gamma^{\alpha}+b^{\alpha})\, i(t) + \frac{1}{k}\\
{}^{C}D^{\alpha}_{0+} r(t) &= \gamma^{\alpha} i(t) - b^{\alpha} r(t) + \frac{1}{k} \quad \text{with } k \in \mathbb{N}.
\end{aligned} \tag{6}$$

We next show that for all $t \geq 0$, the solution $(m_k^{\star}(t), i_k^{\star}(t), r_k^{\star}(t))$ of system (5)-(6) lies in $[R_0^+]^3$. To arrive at a contradiction, suppose there is a specific moment when the condition does not hold. Consider

$$t_0 = \inf\left\{\tilde{s} > 0 : (m_k^*(\tilde{s}), i_k^*(\tilde{s}), r_k^*(\tilde{s})) \notin [R_0^+]^3\right\}.$$

So, $(m_k^*(t_0), i_k^*(t_0), r_k^*(t_0)) \in [R_0^+]^3$ and one of $m_k^*(t_0)$, $i_k^*(t_0)$ or $r_k(t_0)$ is zero. Supposing that $m_k(t_0) = 0$, then since we have

$${}^{C}D^{\alpha}_{0+} m_k^*(t) = b^{\alpha}\,(i_k^*(t_0) + r_k^*(t_0)) + \frac{1}{k} > 0,$$

we use the continuity of ${}^{C}D^{\alpha}_{0+} m_k^*$ to conclude that for some $\zeta > 0$, we have

$${}^{C}D^{a}_{0+} m_k^*([t_0, t_0+\zeta[) \subseteq \mathbb{R}^+.$$

Employing Theorem A.1, we get $m_k^*([t_0, t_0+\zeta[)$ lies in $\mathbb{R}_0^+$, and thus m_k^* is non-negative. Similarly, we show that the functions i_k^*, r_k^* are also non-negative, yielding a contradiction. Using Lemma A.4, we infer for $k \to \infty$ that a solution $(m^*(t), i^*(t), r^*(t))$ of (4)-(5) lies in $[R_0^+]^3$, for each $t \geq 0$. ∎

Theorem III.2. *The system described by* (4)-(5) *admits at most two equilibrium states:*

- Disease-free equilibrium at $P_F = (0,1,0,0)$,

- Endemic equilibrium at $P_E = (m^*, s^*, i^*, r^*)$, where

$$r^* = \frac{\gamma^a}{b^{\alpha}} i^*, \quad s^* = 1 - m^* - i^* - r^*,$$

and

$$i^* = \frac{b^{\alpha}\beta^{\alpha}\delta^{\alpha} - \delta^{\alpha} b^{\alpha}(\gamma^{\alpha}+b^{\alpha})}{\gamma^{\alpha}\beta^{\alpha}b^{\alpha} + b^{2\alpha}\beta^{\alpha} + \delta^{\alpha}\beta^{\alpha}b^{\alpha} + \delta^{\alpha}\gamma^{\alpha}\beta^{\alpha}}$$

$$m^* = \frac{\beta^{\alpha}b^{2\alpha} + \beta^{\alpha}\gamma^{\alpha}b^{\alpha} - 2\gamma^{\alpha}b^{2\alpha} - \gamma^{2\alpha}b^{\alpha} - b^{3\alpha}}{\beta^{\alpha}(\gamma^{\alpha}b^{\alpha} + b^{2\alpha} + \delta^{\alpha}b^{\alpha} + \delta^{\alpha}\gamma^{\alpha})}$$

if m^*, i^*, s^* and r^* range in $(0,1)$.

Proof: The equilibrium states are determined by setting right-hand terms in (4) equal to 0. We have

$${}^{C}D^{\alpha}_{0+} m(t) = {}^{C}D^{\alpha}_{0+} i(t) = {}^{C}D^{\alpha}_{0+} r(t) = 0.$$

Subsequently, we find

$$\begin{cases}
b^{\alpha}(r(t)+i(t)) - \delta^{\alpha} m(t) = 0\\
\beta^{\alpha} i(t)(1-m(t)-r(t)-i(t)) - (\gamma^{\alpha}+b^{\alpha})\, i(t) = 0\\
\gamma^{\alpha} i(t) - b^{\alpha} r(t) = 0,
\end{cases}$$

$$\iff \begin{cases}
b^{\alpha}(i+r) - \delta^{\alpha} m = 0\\
i(\beta^{\alpha}(1-m-i-r) - (\gamma^{\alpha}+b^{\alpha})) = 0\\
\gamma^{\alpha} i - b^{\alpha} r = 0,
\end{cases}$$

$$\iff$$

$$\begin{cases}
b^{\alpha}(i+r) - \delta^{\alpha} m = 0\\
i = 0 \text{ or } (\beta^{\alpha}(1-m-i-r) - (\gamma^{\alpha}+b^{\alpha})) = 0\\
r = \frac{\gamma^{\alpha}}{b^{\alpha}} i.
\end{cases}$$

If $i = 0$, then $m = 0$, $r = 0$ and $s = 1$, Thus the, disease-free steady occurs at

$$P_F = (0,1,0,0).$$

If $(\beta^{\alpha}(1-m-i-r) - (\gamma^{\alpha}+b^{\alpha})) = 0$, then

$$1 - m - i - r = \frac{\gamma^{\alpha}+b^{\alpha}}{\beta^{\alpha}} \Leftrightarrow m = 1 - i - r - \frac{\gamma^{\alpha}+b^{\alpha}}{\beta^{\alpha}}.$$

We replace m and $r = \dfrac{\gamma^{\alpha}}{b^{\alpha}} i$ in the equation:

$$b^{\alpha}(i+r) - \delta^{\alpha} m = 0$$

to find

$$\begin{aligned}
& b^{\alpha}(i + \frac{\gamma^{\alpha}}{b^{\alpha}} i) - \delta^{\alpha}(1 - i - r - \frac{\gamma^{\alpha}+b^{\alpha}}{\beta^{\alpha}}) = 0\\
&\iff b^{\alpha} i + \gamma^{\alpha} i - \delta^{\alpha} + \delta^{\alpha} i\\
&\qquad + \frac{\delta^{\alpha}\gamma^{\alpha}}{b^{\alpha}}\, i + \frac{\delta^{\alpha}(\gamma^{\alpha}+b^{\alpha})}{\beta^{\alpha}} = 0\\
&\iff \gamma^{\alpha}\beta^{\alpha}b^{\alpha} i + b^{2\alpha}\beta^{\alpha} i - b^{\alpha}\beta^{\alpha}\delta^{\alpha} + \delta^{\alpha}\beta^{\alpha}b^{\alpha} i\\
&\qquad + \delta^{\alpha}\gamma^{\alpha}\beta^{\alpha} i + \delta^{\alpha} b^{\alpha}(\gamma^{\alpha}+b^{\alpha}) = 0\\
&\iff i(\gamma^{\alpha}\beta^{\alpha}b^{\alpha} + b^{2\alpha}\beta^{\alpha} + \delta^{\alpha}\beta^{\alpha}b^{\alpha} + \delta^{\alpha}\gamma^{\alpha}\beta^{\alpha})\\
&\qquad = b^{\alpha}\beta^{\alpha}\delta^{\alpha} - \delta^{\alpha} b^{\alpha}(\gamma^{\alpha}+b^{\alpha})
\end{aligned}$$

$$\iff i = \frac{b^{\alpha}\beta^{\alpha}\delta^{\alpha} - \delta^{\alpha} b^{\alpha}(\gamma^{\alpha}+b^{\alpha})}{\gamma^{\alpha}\beta^{\alpha}b^{\alpha} + b^{2\alpha}\beta^{\alpha} + \delta^{\alpha}\beta^{\alpha}b^{\alpha} + \delta^{\alpha}\gamma^{\alpha}\beta^{\alpha}}.$$

We have $m = 1 - i - r - \frac{\gamma^\alpha + b^\alpha}{\beta^\alpha}$, then

$$m = 1 - \frac{b^\alpha\beta^\alpha\delta^\alpha - \delta^\alpha b^\alpha(\gamma^\alpha + b^\alpha)}{\gamma^\alpha\beta^\alpha b^\alpha + b^{2\alpha}\beta^\alpha + \delta^\alpha\beta^\alpha b^\alpha + \delta^\alpha\gamma^\alpha\beta^\alpha} - \frac{\gamma^\alpha}{b^\alpha} i - \frac{\gamma^\alpha + b^\alpha}{\beta^\alpha}$$

$$\Longleftrightarrow$$

$$m = 1 - \frac{b^\alpha\beta^\alpha\delta^\alpha - \delta^\alpha b^\alpha(\gamma^\alpha + b^\alpha)}{\gamma^\alpha\beta^\alpha b^\alpha + b^{2\alpha}\beta^\alpha + \delta^\alpha\beta^\alpha b^\alpha + \delta^\alpha\gamma^\alpha\beta^\alpha} - \frac{\gamma^\alpha}{b^\alpha}\frac{b^\alpha\beta^\alpha\delta^\alpha - \delta^\alpha b^\alpha(\gamma^\alpha + b^\alpha)}{\gamma^\alpha\beta^\alpha b^\alpha + b^{2\alpha}\beta^\alpha + \delta^\alpha\beta^\alpha b^\alpha + \delta^\alpha\gamma^\alpha\beta^\alpha} - \frac{\gamma^\alpha + b^\alpha}{\beta^\alpha}$$

$$\Longleftrightarrow$$

$$m = 1 - \frac{b^\alpha\beta^\alpha\delta^\alpha - \delta^\alpha b^\alpha(\gamma^\alpha + b^\alpha)}{\gamma^\alpha\beta^\alpha b^\alpha + b^{2\alpha}\beta^\alpha + \delta^\alpha\beta^\alpha b^\alpha + \delta^\alpha\gamma^\alpha\beta^\alpha} - \gamma^\alpha\frac{\beta^\alpha\delta^\alpha - \delta^\alpha(\gamma^\alpha + b^\alpha)}{\gamma^\alpha\beta^\alpha b^\alpha + b^{2\alpha}\beta^\alpha + \delta^\alpha\beta^\alpha b^\alpha + \delta^\alpha\gamma^\alpha\beta^\alpha} - \frac{\gamma^\alpha + b^\alpha}{\beta^\alpha}.$$

Then,

$$m = \frac{\beta^\alpha b^{2\alpha} + \beta^\alpha\gamma^\alpha b^\alpha - 2\gamma^\alpha b^{2\alpha} - \gamma^{2\alpha} b^\alpha - b^{3\alpha}}{\beta^\alpha(\gamma^\alpha b^\alpha + b^{2\alpha} + \delta^\alpha b^\alpha + \delta^\alpha\gamma^\alpha)}$$

■

IV. Local stability analysis

Theorem IV.1. *The infection-free state P_F of* (4)-(5) *is locally asymptotically stable (shortly denoted "LAS") if*

$$\frac{\beta^\alpha}{b^\alpha + \gamma^\alpha} < 1$$

Proof: The Jacobian matrix of system (4), computed at P_F is

$$\begin{bmatrix} -\delta^\alpha & b^\alpha & b^\alpha \\ 0 & \beta^\alpha - \gamma^\alpha - b^\alpha & 0 \\ 0 & \gamma^\alpha & -b^\alpha \end{bmatrix} \tag{7}$$

We determine the eigenvalues of this matrix by resolving

$$P(\lambda) = \det(J - \lambda I) = \begin{vmatrix} -\delta^\alpha - \lambda & b^\alpha & b^\alpha \\ 0 & \beta^\alpha - \gamma^\alpha - b^\alpha - \lambda & 0 \\ 0 & \gamma^\alpha & -b^\alpha - \lambda \end{vmatrix} = 0$$

or equivalently,

$$(-\delta^\alpha - \lambda)(\beta^\alpha - \gamma^\alpha - b^\alpha - \lambda)(-b^\alpha - \lambda) = 0.$$

Thus, matrix (9) has the below eigenvalues:

$$\lambda = -\delta^\alpha \vee \lambda = -b^\alpha \vee \lambda = \beta^\alpha - \gamma^\alpha - b^\alpha,$$

As proven in [2], [25], the infection-free equilibrium for (4) is *LAS* if the above eigenvalues verify:

$$\alpha\frac{\pi}{2} < |\arg(\lambda_i)|, \tag{8}$$

proving the desired result. ■

Theorem IV.2. *The disease-present equilibrium P_E of system* (4)-(5) *is LAS if*

$$\frac{\beta^\alpha}{b^\alpha + \gamma^\alpha} > 1$$

Proof: Compute a Jacobian matrix of (4) at P_E, is yields

$$\begin{bmatrix} -\delta^\alpha & b^\alpha & b^\alpha \\ -\beta^\alpha i^* & 0 & -\beta^\alpha i^* \\ 0 & \gamma^\alpha & -b^\alpha \end{bmatrix} \tag{9}$$

We determine then their eigenvalues by resolving

$$P(\lambda) = \det(J - \lambda I) = \begin{vmatrix} -\delta^\alpha - \lambda & b^\alpha & b^\alpha \\ -\beta^\alpha i^* & -\lambda & -\beta^\alpha i^* \\ 0 & \gamma^\alpha & -b^\alpha - \lambda \end{vmatrix} = 0$$

or equivalently,

$$\lambda^3 + \lambda^2(\delta^\alpha + b^\alpha) + \lambda(\delta^\alpha b^\alpha + \gamma^\alpha\beta^\alpha i^* + \beta^\alpha b^\alpha) + \delta^\alpha\gamma^\alpha\beta^\alpha i^* + \beta^\alpha b^{2\alpha} i^* + \beta^\alpha b^\alpha\gamma^\alpha i^* = 0$$

where

$$a_1 = \delta^\alpha + b^\alpha > 0,$$

$$\begin{aligned} a_2 &= \delta^\alpha b^\alpha + \gamma^\alpha\beta^\alpha i^* + \beta^\alpha b^\alpha \\ &= \delta^\alpha b^\alpha + \beta^\alpha b^\alpha + \gamma^\alpha\beta^\alpha\frac{b^\alpha\beta^\alpha\delta^\alpha - \delta^\alpha b^\alpha(\gamma^\alpha + b^\alpha)}{\gamma^\alpha\beta^\alpha b^\alpha + b^{2\alpha}\beta^\alpha + \delta^\alpha\beta^\alpha b^\alpha + \delta^\alpha\gamma^\alpha\beta^\alpha} \\ &= \delta^\alpha b^\alpha + \beta^\alpha b^\alpha + \gamma^\alpha\beta^\alpha\frac{b^\alpha\delta^\alpha(\beta^\alpha - (b^\alpha + \gamma^\alpha))}{\gamma^\alpha\beta^\alpha b^\alpha + b^{2\alpha}\beta^\alpha + \delta^\alpha\beta^\alpha b^\alpha + \delta^\alpha\gamma^\alpha\beta^\alpha} \\ &> 0, \end{aligned}$$

$$\begin{aligned} a_3 &= \delta^\alpha\gamma^\alpha\beta^\alpha i^* + \beta^\alpha b^{2\alpha} i^* + \beta^\alpha b^\alpha\gamma^\alpha\, i^* \\ &= (\delta^\alpha\gamma^\alpha\beta^\alpha + \beta^\alpha b^{2\alpha} + \beta^\alpha b^\alpha\gamma^\alpha)\, i^* \\ &= (\delta^\alpha\gamma^\alpha\beta^\alpha + \beta^\alpha b^{2\alpha} + \beta^\alpha b^\alpha\gamma^\alpha) \\ &\quad \times \frac{b^\alpha\delta^\alpha(\beta^\alpha - (b^\alpha + \gamma^\alpha))}{\gamma^\alpha\beta^\alpha b^\alpha + b^{2\alpha}\beta^\alpha + \delta^\alpha\beta^\alpha b^\alpha + \delta^\alpha\gamma^\alpha\beta^\alpha} \\ &> 0. \end{aligned}$$

By Hurwitz-Routh Criterion, system (4) is *LAS* if

$$a_i > 0\,,\ a_1 a_2 > a_3 \quad (i = 1, 2, 3).$$

Thus, the disease present equilibrium P_E of (4) is *LAS* if

$$\beta^\alpha/(\gamma^\alpha + b^\alpha) > 1.$$

■

The generation number R_0, is given by the formula $\beta^\alpha/(\gamma^\alpha + b^\alpha)$. It signifies the mean count of new cases arising from one infection in a fully susceptible host population.

V. Global stability

Theorem V.1. *The disease-free steady P_E of* (4)-(5) *is globally asymptotically stable (shortly denoted GAS) if*

$$R_0 < 1.$$

Proof: Let $V : \Gamma \to \mathbb{R}$, $V(m, i, r) = i$ be a Lyapunov map so that

$$\Gamma = \big\{(m, i, r) \in \Gamma : \ m > 0, i > 0, r > 0\big\}.$$

Hence, the Lyapunov map derivative is as below

$$\begin{aligned}
&{}^{C}D^{\alpha}_{0+}V(m,i,r) = {}^{C}D^{\alpha}_{0+}i \\
&= \beta^{\alpha} i(t)(1 - m(t) - r(t) - i(t)) - (\gamma^{\alpha} + b^{\alpha})\, i(t) \\
&= i(t)(\beta^{\alpha}\,(1 - m(t) - r(t) - i(t)) - (\gamma^{\alpha} + b^{\alpha})) \\
&= i(t)\,\big[R_0\big(\gamma^{\alpha} + b^{\alpha})(1 - m(t) - r(t) - i(t)\big) \\
&\qquad\qquad - (\gamma^{\alpha} + b^{\alpha})\big] \\
&= i(t)(\gamma^{\alpha} + b^{\alpha})(R_0(1 - m(t) - r(t)) - 1 - i(t)\,).
\end{aligned}$$

Thus

$$0 \geq {}^{C}D^{\alpha}_{0+}V(m,i,r) \iff 1 \geq R_0,$$
$${}^{C}D^{\alpha}_{0+}V = 0 \iff i = 0.$$

Using LaSalle principle [31], we find that P_E is *GAS* in Γ. ∎

VI. Sensitivity Analysis of R_0

Sensitivity is often employed to assess the model robustness regarding to variations in parameter values, helping us identify which parameters significantly influence the basic reproduction number R_0. Applying the method outlined by Chitnis et al. [30], we compute the normalized forward sensitivity indices of R_0. Specifically, consider $\Upsilon^{R_0}_{p} = \frac{\partial R_0}{\partial p} * \frac{p}{R_0}$ as the sensitivity index of R_0, relative to p. This yields:

$$R_0 = \frac{\beta^{\alpha}}{\gamma^{\alpha} + b^{\alpha}}, \ \Upsilon^{R_0}_{\beta} = \alpha,$$
$$\Upsilon^{R_0}_{\gamma} = \frac{-\alpha\gamma^{\alpha}}{\gamma^{\alpha} + b^{\alpha}}, \ \Upsilon^{R_0}_{b} = \frac{-\alpha b^{\alpha}}{\gamma^{\alpha} + b^{\alpha}}.$$

From the above discussion we observe that the basic reproduction number R_0 is most sensitive to changes in β . If β will increase R_0 will also increase with the same proportion and if β decreases in same the proportion, γ and b will be an inversely related to R_0.

VII. Numerical simulation

We present several numerical solutions to system (4) for various parameter values. The initial conditions are chosen such that $m+i+r = 50$. We perform and display simulations of (4) to illustrate our results.. By choosing $b = 0.065$ and $\gamma = \delta = 1$, the disease-free steady is $P_F = (0, 1, 0, 0)$, and the generation number is $R_0 = 0.11$, which is less than 1. According to Theorem IV.1, this disease-free equilibrium is *LAS*, see the figures below.

By varying the initial values m_0, i_0 and r_0, we observe from such figures that

1) The population of individuals with passive immunity, safeguarded by maternal antibodies, approaches zero ($m = 0$), see Fig. 1.
2) The infected cases decreases and converges to zero, see Fig. 2.
3) The recovered people with permanent immunity increases at first, then decreases and approaches the value $r = 0.1$, see Fig. 3.

Similarly, by varying the value α, we observe from the obtained figures that

1) The population of individuals with passive immunity, conferred by maternal antibodies, tends towards $m = 0$, see Fig. 4.
2) The infected cases decreases towards zero, see Fig. 5.
3) The population of recovered people with permanent immunity increases at first, then decreases and approaches the value $r = 0$, see Fig. 6.

VIII. Conclusion

Epidemic models play a role globally by offering insights to health authorities for understanding disease transmission and making informed decisions on epidemic control strategies. We have introduced a fractional nonlinear MSIR model which is applicable to any case where individuals gain permanent immunity post-infection by the causing microorganism. We demonstrated the uniqueness and non negativity of our models solution along with proving that it has a maximum of two equilibrium points. Furthermore, we presented a requirement for *LAS* and *GAS* stability of the infection-free equilibrium.

Appendix A
Appendix : Preliminaries

We recall here some useful notations, definitions, and lemmas. Let $f : \mathbb{R}^+ \to \mathbb{R}$. For a given $\alpha \in (0, 1)$ and $t_0 \in \mathbb{R}$, we consider the fractional system:

$${}^{C}D^{\alpha}y(t) = f(t, y(t)) \ \text{ with } \ y\,(t_0) = y_0 \qquad (10)$$

For a global solvability of system (10), we need the following theorem, see [22].

Theorem A.1. *Let $J = [t_0 - \alpha, t_0 + \alpha]$, $B = \overline{B}_{\mathbb{R}^d}(y_0, b)$ and $D = J \times B$. Let $f : D \to \mathbb{R}^d$ verifying*

1) *$f_{t,\cdot} : y \to f(t, y)$ is continuous on B,*
2) *$f_{\cdot,y} : t \to f(t, y)$ is measurable on J,*
3) *$\|f(\cdot, y)\| \leq \omega + \lambda\|y\|$, $\forall\, y \in B$, where ω, $\lambda \geq 0$.*

Thus, there exists a function y solving System (10)*.*

Remark. *Besides assumptions of theorem A.1, $y \mapsto \frac{\partial f(t,y)}{\partial y}$ is further supposed to be continuous on B. Then, a solution $y(t)$ of* (10) *exists and is unique.*

We now highlight the Gamma map, given by

$$\Gamma(n) = \int_0^{\infty} e^{-t} t^{n-1}\, dt.$$

Using the Matlab syntax $[\,] = gamma()$, the function Γ can be directly implemented.

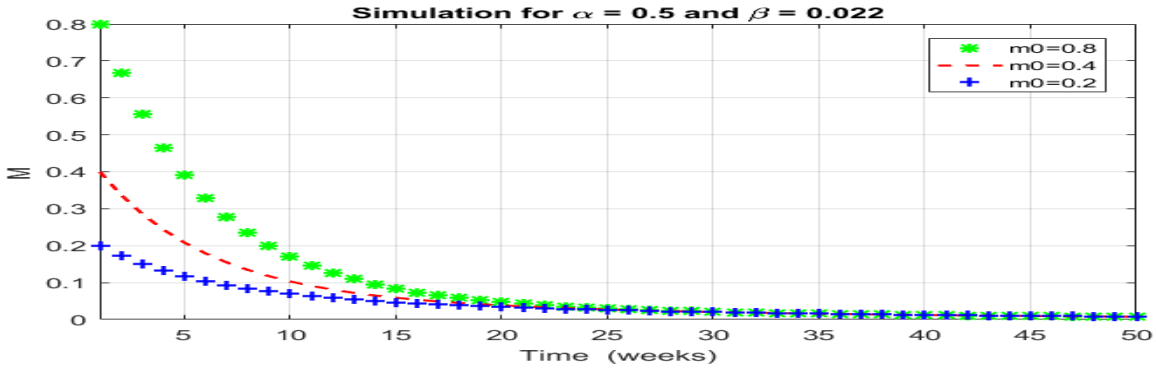

Fig. 1. Evolution of passively immunized population.

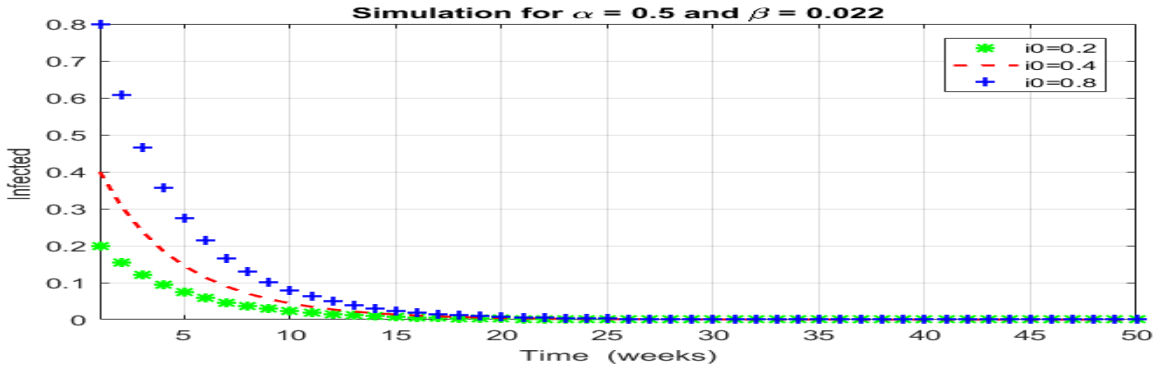

Fig. 2. Evolution of infected population

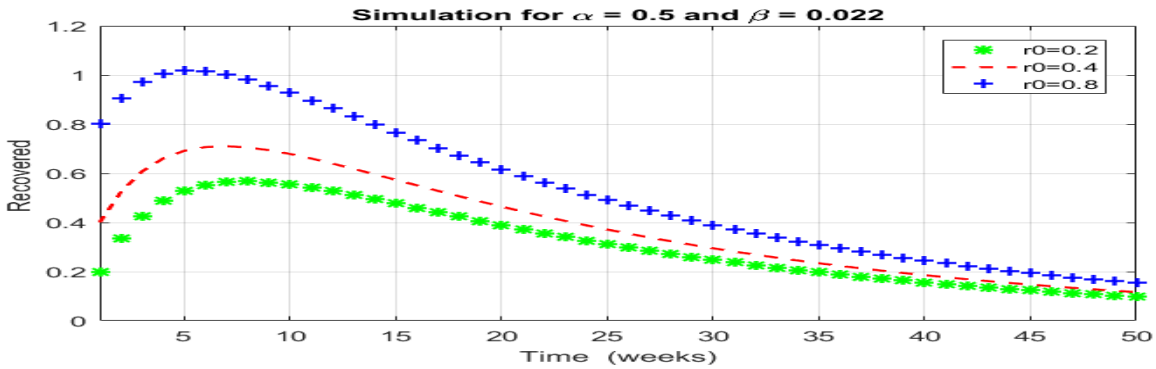

Fig. 3. Evolution of recovered population

Let $\alpha,\ \beta \geq 0$, we highlight the Leffler-Mittag function $E_{\alpha,\beta}$ of parameters α and β is as below

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

Definition A.2. *1. Given $\alpha > 0$ and $y : [a,b] \to \mathbb{R}$ integrable, the α-order fractional integral of y is*

$$I_{a+}^{\alpha} y(t) = \frac{1}{\Gamma(\alpha)} \int_a^t (t-\tau)^{\alpha-1} y(\tau)\, d\tau,\ \forall\, t > a.$$

2. The α-order Caputo fractional derivative is

$${}^{C}D_{a+}^{\alpha} y(t) := \begin{cases} \dfrac{1}{\Gamma(n-\alpha)} \displaystyle\int_a^t (t-\tau)^{n-\alpha-1} y^{(n)}(\tau)\, d\tau \\ \qquad\qquad \text{if } \ \alpha \notin \mathbb{N}\ (n = [\alpha]+1) \\ y^{(\alpha)}(t) \quad \text{if} \quad \alpha \in \mathbb{N}. \end{cases}$$

Theorem A.3. *Let y and ${}^{C}D_{a+}^{\alpha} y$ be continuous functions. For any $t \in (a,b]$, there is $c \in]a,t[$ verifying*

$$y(t) = y(a) + \frac{1}{\Gamma(\alpha+1)}\, {}^{C}D_{a+}^{\alpha} y(c)\, (t-a)^{\alpha}.$$

Remark. *Theorem A.3 implies that if ${}^{C}D_{a+}^{\alpha} y(t) > 0,\ \forall\, t,\ y$*

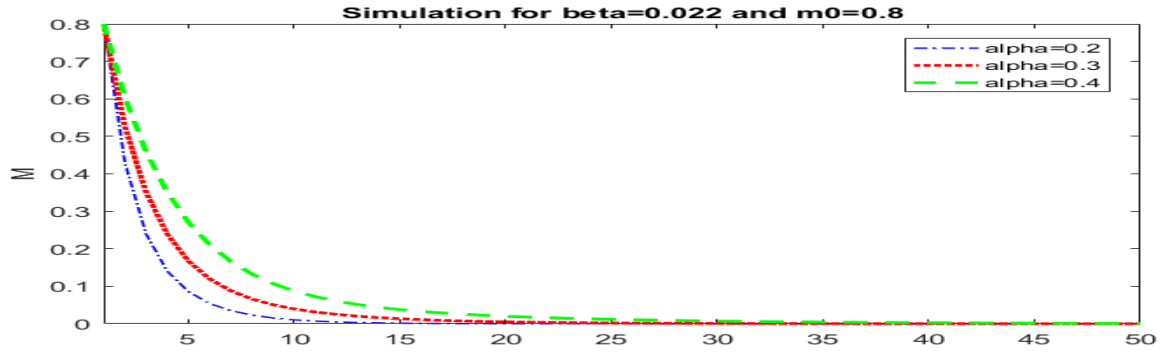

Fig. 4. Evolution of passively immunized population

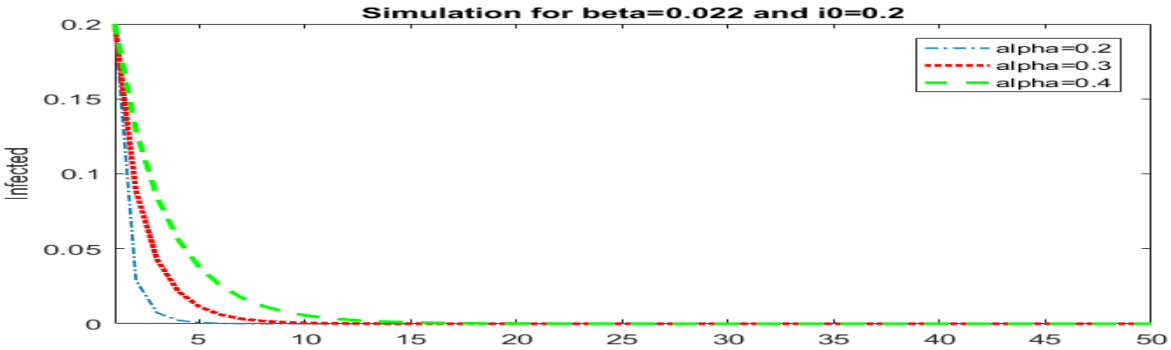

Fig. 5. Evolution of infected population

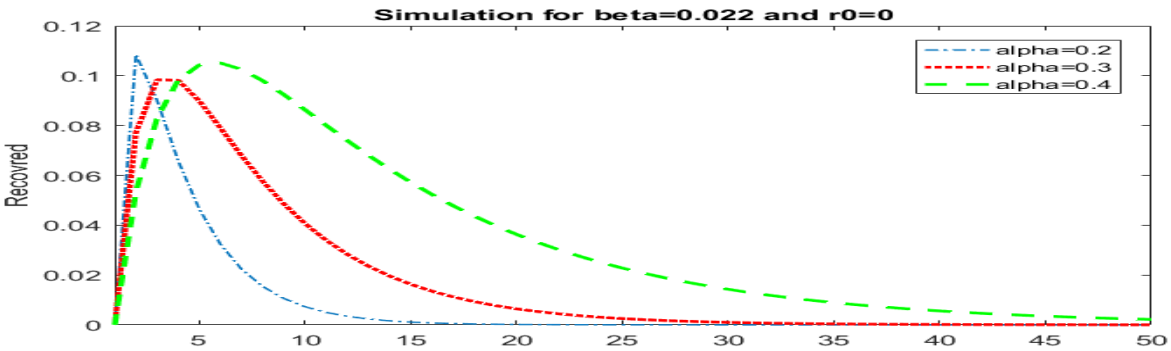

Fig. 6. Evolution of recovered population

increase strictly (y decreases strictly if ${}^{C}D^{\alpha}_{a+}y(t) < 0, \forall\, t.$

Proposition A.1. *1. If* $y : [a,b] \to \mathbb{R}$ *is continuous, then*

$$ {}^{C}D^{\alpha}_{a+} I^{\alpha}_{a+} y(t) = y(t). $$

2. Let $y : [a,b] \to \mathbb{R}$ *be of class* C^n*, then*

$$ I^{\alpha}_{a+}\, {}^{C}D^{\alpha}_{a+} y(t) = y(t) - \sum_{k=0}^{n-1} \frac{y^{(k)}(a)}{k!} (t-a)^k. $$

Lemma A.4. *Let* $f_i : [a,b] \times \mathbb{R}^m \to \mathbb{R}$ *be a continuous functions which are Lipschtiz on* x*, i.e.;*

$$ \exists\, \mathfrak{K}_i > 0\,; \;\; |f_i(t,X) - f_i(t,X')| \leq \mathfrak{K}_i \, \|X - X'\|. $$

Consider $f = (f_1, .., f_m)$*,* $k \in \mathbb{N}^*$ *and* $\alpha \in (0,1)$*. Then, if* $X^{\star}_k = (x_{1,k}, .., x_{m,k})$ *and* $X^{\star} = (x_1, .., x_m)$ *are respectively, solutions of*

$$ \begin{aligned} {}^{C}D^{\alpha}_{a+} Y(t) &= f(t,Y) + \frac{1}{k}, \\ {}^{C}D^{\alpha}_{a+} Y(t) &= f(t,Y), \end{aligned} \tag{11} $$

for the same initial requirement, one has

$$ X^{\star}_k(t) \underset{k\to\infty}{\longrightarrow} X^{\star}(t), \;\; \forall\, t. $$

Proof: By integrating (11) in fractional sens, we find

$$\begin{aligned}
&\|X_k^\star(t) - X^\star(t)\| \\
&\leq \sum_{i=1}^{m} \left|x_{i,k}^\star(t) - x_i^\star(t)\right| \\
&\leq \sum_{i=1}^{m} \Big[\frac{1}{\Gamma(\alpha)} \\
&\int_a^t (t-\tau)^{\alpha-1} \left|f_i\left(\tau, kX^\star(\tau)\right) - f_i\left(\tau, X^\star(\tau)\right)\right| d\tau \\
&\qquad + \frac{(t-a)^\alpha}{\Gamma(\alpha+1)k}\Big] \\
&\leq \sum_{i=1}^{m} \Big[\frac{L_i}{\Gamma(\alpha)} \int_a^t (t-\tau)^{\alpha-1} \left\|kX^\star(\tau) - X^\star(\tau)\right\| d\tau \\
&\qquad + \frac{(t-a)^\alpha}{\Gamma(\alpha+1)k}\Big].
\end{aligned}$$

Then, Gronwall inequality leads to (see [14, Theorem 8])

$$\begin{aligned}
&\|X_k^\star(t) - X^\star(t)\| \\
&\leq \frac{m(t-a)^\alpha}{k\Gamma(\alpha+1)} E_\alpha\Big(\sum_{i=1}^{m} L_i (t-a)^\alpha\Big).
\end{aligned}$$

Therefore, we infer that

$$\|X_k^\star(t) - X^\star(t)\| \underset{k\to\infty}{\longrightarrow} 0.$$

■

REFERENCES

[1] E. Ackerman, L.R. Elveback and J.P. Fox, "Simulation of Infectious Disease Epidemics," *Charles Thomas Publisher*, Springfield 1984.

[2] E. Ahmed, A.M. El-Sayed and H.A. El-Saka, "Equilibrium points, stability and numerical solutions of fractional-order predator-prey and rabies models," *J. Math. Anal. Appl.*, vol. 325, no 1, pp. 542-553, 2007.

[3] R.M. Anderson and R.M. May, "Infectious Diseases of Humans: Dynamics and Control," *Oxford Univ. Press*, London 1991.

[4] N.T Bailey, "The Mathematical Theory of Infectious Diseases and Its Application," *Griffin*, London 1975.

[5] M. Berir, R. Saadeh, M.A. Abdoon, A. Qazza and D. Almutairi, "A Fractional Study for Solving the SIR Model and Chaotic System," *IAENG Inter. Jou. Appl. Mathematics*, vol. 54, no. 2, pp. 212-217, 2024.

[6] M. Boots and R. Norman, "Sublethal infection and the population dynamics of host-microparasite interactions," *J. Animal Ecology*, vol. 69, pp. 517-524, 2000.

[7] K. Diethelm, "A fractional calculus based model for the simulation of an outbreak of dengue fever," *Nonlinear Dyn.*, vol. 71, no. 4, pp. 613-619, 2013.

[8] A. Dokoumetzidis, R. Magin and P. Macheras, "A commentary on fractionalization of multicompartmental models," *J Pharmacokinet Pharmacodyn*, vol, 37, pp. 203-207, 2010.

[9] L. Esteva and C. Vargas, "Analysis of a dengue disease transmission model," *Math. Biosciences*, vol. 150, pp. 131-151, 1998.

[10] Z. Feng, S. Towers and Y. Yang, "Modeling the effects of vaccination and treatment on pandemic influenza," *Am. Assoc. Pharm. Sci. J.*, vol. 13, no. 3, pp. 427-437, 2011.

[11] J. Glasser, D. Taneri, Z. Feng, J.-H. Chuang, P. Tüll, et al, "Evaluation of targeted influenza vaccination strategies via population modeling," *PLoS One*, vol. 5, no. 9, e12777, 2010.

[12] A. Gray, D. Greenhalgh, L. Hu, X. Mao and J. Pan, "A stochastic differential equation SIS epidemic model," *SIAM J. Appl. Math.*, vol. 71, no. 3, pp. 876-902, 2011.

[13] H.W. Hethcote, "The mathematics of infectious diseases," *SIAM Rev.*, vol.42, pp. 599-653, 2000.

[14] R. Hilfer, "Applications of Fractional Calculus in Physics," *World Scientific*, Singapore 2000.

[15] I. Hisashi, "Age-structured homogeneous epidemic systems with application to the MSEIR epidemic model," *J. Math. Biol.*, vol. 54, pp. 101-146, 2007.

[16] J. Huo and H. Zhao, "Dynamical analysis of a fractional SIR model with birth and death on heterogeneous complex networks," *Phys. A*, vol. 448, pp. 41-56, 2016.

[17] W.O. Kermack and A.G. McKendrick, "A contribution to the mathematical theory of epidemics," *Proc. R. Soc. Lond. A*, vol. 115, pp. 700-721, 1927.

[18] W.O. Kermack and A.G. McKendrick, "Contributions to the mathematical theory of epidemics, II - the problem of endemicity," *Proc. R. Soc. Lond. A*, vol. 138, pp. 55-83, 1932.

[19] W.O. Kermack and A.G. McKendrick, "Contributions to the mathematical theory of epidemics, III - further studies of the problem of endemicity," *Proc. R. Soc. Lond. A*, vol. 141, pp. 94-122, 1933.

[20] A. Korobeinikov, "Lyapunov functions and global stability for SIR and SIRS epidemiological models with non-linear transmission," *Bull. Math. Biol.*, vol. 68, no. 3, pp. 615-626, 2006.

[21] A. Korobeinikov and P.K. Maini, "A Lyapunov function and global properties for SIR and SEIR epidemiological models with nonlinear incidence," *Math. Biosci. Eng.*, vol. 1, no. 1, pp. 57-60, 2004.

[22] W. Lin, "Global existence theory and chaos control of fractional differential equations," *J. Math. Anal. Appl.*, vol. 332, no. 1, pp. 709-726, 2007.

[23] M. Lipsitch, T. Cohen, M. Muray and B.R. Levin, "Antiviral resistance and the control of pandemic influenza," *PLoS Med.*, vol. 4, 01110120, 2007.

[24] G. MacDonald, "The Epidemiology and Control of Malaria," *Oxford University Press*, London, 1957.

[25] D. Matignon, "Stability results for fractional differential equations with applications to control processing," *In: Computational Engineering in Systems Applications*, Lille, France 2, 1996.

[26] J.K. Popovic, M.T. Atanackovic, A.S. Pilipovic, M.R. Rapaic and S. Pilipovic, "A newapproach to the compartmental analysis in pharmacokinetics: fractional time evolution of diclofenac," *J Phamacokinet Pharmacodyn*, vol. 37, pp. 119-134, 2010.

[27] S. Side, M. Jannah, A. Saman, M.I. Pratama and W. Sanusi, "Optimal Control and Analysis of the SEIRS Model on the Problem of Online Game Addiction : A Case Study Among Class VIII Students of the State Junior High Schools in Makassar City," *IAENG International Journal of Applied Mathematics*, vol. 54, no. 2, pp. 232-237, 2024.

[28] J. Suksamran, S. Amornsamankul, and Y. Lenbury, "Reaction-Diffusion-Integral System Modeling SARS-CoV-2 Infection-Induced versus Vaccine-Induced Immunity: Analytical Solutions and Stability Analysis," *IAENG International Journal of Applied Mathematics*, vol. 54, no. 2, pp. 223-231, 2024.

[29] R.W. West and J.R. Thompson, "Models for the simple epidemic," *Math. Biosci.*, vol. 141, pp. 29-39, 1997.

[30] N. Chitnis, S. Hyman, and J.M. Cushing, "Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model," *Bulletin of Mathematical Biology*. 70 (5), 1272 (2008).

[31] J.P. La Salle, *The Stability of Dynamical Systems. Society for Industrial and Applied Mathematics, Philadelphia (1976).*