A Fractional Mathematical Model of Influenza: Meningitis Coinfection Using Caputo Derivatives

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Abstract: This study examines a mathematical model involving the influenza infection on the spread of meningitis within a population. This research extends previous studies by formulating the model in Caputo fractional derivative with order α. Based on this model, we determined the equilibrium points of the system and their stability conditions are determined. We also employ the Next Generation Matrix method to calculate the basic reproduction number (R_0). Subsequently, the model solution is addressed through a numerical simulation scheme for the fractional model, specifically the Predictor-Corrector Product Integration Rule (PECE-PI) method. The result of this study showed that Different values of the fractional order indicated varying speeds of reaching a steady state or endemic level while the changes of both influenza and meningitis transmission rate and quarantine rate have an impact to transmission dynamics.

1 INTRODUCTION

Influenza is one of the diseases that can be transmitted through airborne droplets and infect the respiratory tract (Beauchemin & Handel, 2011). The transmission rate of influenza is relatively high, thus a susceptible person should maintain a distance of at least one meter from an infected individual to minimize the risk of infection (Jonnalagadda, 2022). The infection caused by this virus typically lasts around one week and is characterized by symptoms such as fever, headache, pharyngitis, cough, and fatigue. Generally, influenza infection affects the nose, throat, bronchi, and even the lungs (Zhou & Guo, 2012). In recent years, influenza has been found in several different strains. In 2009, the H5N1 strain caused avian flu infections, which were later followed by the H1N1 strain that marked the onset of swine flu (Kharis & Arifudin, 2017).

In addition to influenza, meningitis is a contagious disease caused by the bacterial infection of meningococcus (*Neisseria meningitidis*) (Widyastuti et al., 2023). The infection process of meningitis occurs through the transmission of bacteria via airborne droplets from an infected individual to a susceptible person. Additionally, the use of personal items contaminated with bacteria can also cause meningitis infection. The Neisseria meningitidis bacteria infect the meninges, which are thin layers that provide protection to the brain and spinal cord (Abdullahi Baba et al., 2020; Musa et al., 2020; Sulma et al., 2020; Türkün et al., 2023). After infection, an individual may be asymptomatic or may exhibit symptoms. Symptoms that can appear in an infected individual include high fever, headache, stiff neck, vomiting, and skin rash. Meningitis infection requires prompt and accurate treatment, as untreated meningitis can lead to fluid swelling around the brain and spinal cord, potentially causing disability or death (Bashir et al., 2003; Musa et al., 2020).

In terms of their transmission, both influenza and meningitis spread from person to person through coughing, sneezing, or airborne droplets. Several symptoms caused by these diseases are similar,

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necessitating clinical tests for accurate identification. The similar patterns of spread and symptoms make it possible for an individual infected with meningitis to be influenced by influenza or vice versa (Cartwright et al., 1991; Salomon et al., 2020). Previous research has extensively studied the mathematical models of influenza transmission only (Aulia & Kharis, 2016; Goswami & Shanmukha, 2016; Kanyiri et al., 2018; Wu & Cowling, 2011) and meningitis only (Abdullahi Baba et al., 2020; Asamoah et al., 2018; Buonomo & Della Marca, 2024; Peter et al., 2022). However, there is still a lack of studies examining the transmission dynamics of co-infection models for influenza and meningitis. Therefore, mathematical models are one of the methods that can be used to explore the transmission dynamics of these co-infection models.

A previous study that examined the influenza-meningitis co-infection model is that of Varshney et al., who constructed a mathematical model of the spread of influenza and meningitis co-infection. This model was developed using an integer-order mathematical model to understand the dynamics of the co-infection spread (Varshney & Dwivedi, 2021). Based on this model, the present study develops the influenza-meningitis co-infection model using a fractional-order mathematical model to yield a better understanding of the transmission dynamics of the influenza-meningitis co-infection spread.

2 MODEL FORMULATION AND PROPERTIES

2.1 Model Formulation

The model formulated in this study is divided into six subpopulations: Susceptible $S(t)$, influenza-infective only $I_f(t)$, meningitis-infective only $I_m(t)$, influenza-meningitis coinfectives $I_{fm}(t)$, quarantine of influenza $Q(t)$, and recovered $R(t)$. The $S(t)$ compartment increases by the birth rate Λ. Individuals who come into contact with those infected with influenza or those with influenza-meningitis coinfection will move to the $I_f(t)$ compartment at a contact rate β_1. The proportion of individuals infected with influenza or coinfected with influenza and meningitis moving to the I_f compartment is represented by the force of infection of influenza $f_1 = \frac{\beta_1(I_f+I_{fm})}{N}$, where N is the number of total populations. Similarly, susceptible individuals move to the $I_m(t)$ compartment due to contact with individuals infected with meningitis or those with influenza-meningitis coinfection at a contact rate β_2, with the force of infection of meningitis $f_2 = \frac{\beta_2(I_m+I_{fm})}{N}$. Additionally, the $S(t)$ compartment decreases due to natural death at a rate μ.

The I_f compartment decreases due to several factors: natural death, death caused by influenza infection at a rate d_1, individuals infected with influenza being quarantined at a rate τ_1, individuals who experience coinfection with meningitis (with a force of infection f_2) moving to the I_{fm} compartment, and individuals who recover naturally at a rate γ_1. Similarly, the I_m compartment decreases due to natural death, death caused by meningitis at a rate d_1, secondary influenza infection (with a force of infection f_2) leading to a transition to the I_{fm} compartment, and individuals who recover naturally at a rate γ_3.

Subsequently, the I_{fm} compartment increases due to secondary infection of individuals in the I_f and I_m compartments. This compartment decreases due to natural death, death from coinfection at a rate d_3, and natural recovery at a rate γ_2. The $Q(t)$ compartment increases when individuals infected with influenza are quarantined at a rate τ_1, preventing them from spreading the disease to susceptible individuals. The $Q(t)$ compartment decreases due to natural death, death from influenza infection, and recovery of individuals at a rate ψ. The $R(t)$ compartment increases as individuals recover and decreases due to natural death and loss of immunity at a rate θ, leading to a transition back to the $S(t)$ compartment. The interactions among these compartments are illustrated in Figure 1.

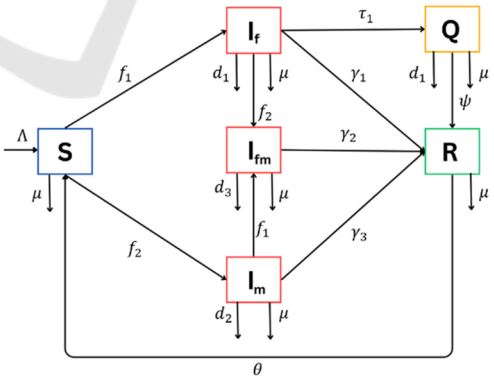

Figure 1. Flowchart of Model.

In this study, we developed the deterministic model of influenza-meningitis coinfection from Varshney & Dwivedi (Varshney & Dwivedi, 2021) into fractional differential system. The model divided into six compartments as follows

$$
\begin{aligned}
&{}_0^C D_t^\alpha S(t) = \Lambda^\alpha + \theta^\alpha R - (f_1 + f_2 + \mu^\alpha)S,\\
&{}_0^C D_t^\alpha I_f(t) = f_1 S - (\mu^\alpha + {d_1}^\alpha + \tau_1^\alpha + \gamma_1^\alpha + \phi f_2) I_f,\\
&{}_0^C D_t^\alpha I_m(t) = f_2 S - (\mu^\alpha + {d_2}^\alpha + \gamma_3^\alpha + \omega f_1) I_m,\\
&{}_0^C D_t^\alpha I_{fm}(t) = \phi f_2 I_f + \omega f_1 I_m - (\mu^\alpha + {d_3}^\alpha + \gamma_2^\alpha) I_{fm},\\
&{}_0^C D_t^\alpha Q(t) = \tau_1^\alpha I_f - (\mu^\alpha + {d_1}^\alpha + \psi^\alpha) Q,\\
&{}_0^C D_t^\alpha R(t) = \gamma_1^\alpha I_f + \gamma_2^\alpha I_{fm} + \gamma_3^\alpha I_m + \psi^\alpha Q - (\mu^\alpha + \theta^\alpha) R.
\end{aligned} \tag{1}
$$

We apply the Caputo fractional derivative in the left-hand side of the model (1) The adjustment to fractional system implies the change of dimension t into $s^{-\alpha}$ dimension for $0 < \alpha \leq 1$ (Barros et al., 2021). Therefore, we set all parameters with power of α to accommodate dimensional changes

2.2 Positivity and Boundedness

Let us consider the closed set Ω defined as $\Omega = \{(S, I_f, I_m, I_{fm}, Q, R) \in R_+^6 | S, I_f, I_m, I_{fm}, Q, R \geq 0\}$ is the biologically feasible region for system (1).

Theorem 1 *The solution of fractional model of Influenza Meningitis coinfection starting in R_+^6 along with initial conditions are positive invariant and bounded for all time $t \geq 0$.*

Proof. We have to show that the set Ω is a positive invariant. From the system (1) we obtained.

$$
\begin{aligned}
&{}_0^C D_t^\alpha S(t) = \Lambda^\alpha + \theta^\alpha R \geq 0\\
&{}_0^C D_t^\alpha I_f(t) = f_1 S \geq 0\\
&{}_0^C D_t^\alpha I_m(t) = f_2 S \geq 0\\
&{}_0^C D_t^\alpha I_{fm}(t) = \phi f_2 I_f + \omega f_1 I_m \geq 0\\
&{}_0^C D_t^\alpha Q(t) = \tau_1^\alpha I_f \geq 0\\
&{}_0^C D_t^\alpha R(t) = \gamma_1^\alpha I_f + \gamma_2^\alpha I_{fm} + \gamma_3^\alpha I_m + \psi^\alpha Q \geq 0
\end{aligned} \tag{2}
$$

The Equation (2) hold for all points in Ω and using Lemma 1 show that the set Ω is positive invariant of model (1).

Next, we derived the boundedness of Ω. If all of the equations in model (1) are added then we obtained the total population as follows

$$
\begin{aligned}
{}_0^C D_t^\alpha N(t) = \Lambda^\alpha - \mu^\alpha \big(S + I_f + I_m + I_{fm} + Q + R\big)\\
- {d_1}^\alpha Q - {d_1}^\alpha I_f - {d_2}^\alpha I_m\\
- {d_3}^\alpha I_{fm}.
\end{aligned}
$$

This gives

$$
{}_0^C D_t^\alpha N(t) \leq \Lambda^\alpha - \mu^\alpha N.
$$

By using Lemma 9 in Choi et al., (2014) we get

$$
N(t) \leq \Lambda^\alpha t^\alpha E_{\alpha,\alpha+1}(-\mu^\alpha t^\alpha) + N_0 E_{\alpha,1}(-\mu^\alpha t^\alpha)
$$

$$
N(t) \leq \frac{\Lambda^\alpha}{\mu^\alpha}\Big(\mu^\alpha t^\alpha E_{\alpha,\alpha+1}(-\mu^\alpha t^\alpha) + E_{\alpha,1}(-\mu^\alpha t^\alpha)\Big)
$$

where $E_{\alpha,\alpha+1}$ is Mittag-Leffler function. Using Theorem 5.1 in Haubold et al., (2011) we obtained

$$
N(t) \leq \frac{\Lambda^\alpha}{\mu^\alpha}\left(\frac{1}{\Gamma(1)} - E_{\alpha,1}(-\mu^\alpha t^\alpha) + E_{\alpha,1}(-\mu^\alpha t^\alpha)\right)
$$

$$
N(t) \leq \frac{\Lambda^\alpha}{\mu^\alpha}\left(\frac{1}{\Gamma(1)}\right) \leq \frac{\Lambda^\alpha}{\mu^\alpha}.
$$

Since the total population is bounded so the subpopulations are also bounded and this complete the proof.

2.3 Normalized Model of Influenza-Meningitis Coinfection Model

By assuming new dimensionless variables, $x_1 = \frac{S}{N}, x_2 = \frac{I_f}{N}, x_3 = \frac{I_m}{N}, x_4 = \frac{I_{fm}}{N}, x_5 = \frac{Q}{N}, x_6 = \frac{R}{N}$, the dimensionless model is obtained as follows

$$
\begin{aligned}
&{}_0^C D_t^\alpha x_1(t) = \mu^\alpha + \theta^\alpha x_6 - (f_1 + f_2 + \mu^\alpha) x_1,\\
&{}_0^C D_t^\alpha x_2(t) = f_1 x_1 - (\mu^\alpha + {d_1}^\alpha + \tau_1^\alpha + \gamma_1^\alpha + \phi f_2) x_2,\\
&{}_0^C D_t^\alpha x_3(t) = f_2 x_1 - (\mu^\alpha + {d_2}^\alpha + \gamma_3^\alpha + \omega f_1) x_3,\\
&{}_0^C D_t^\alpha x_4(t) = \phi f_2 x_2 + \omega f_1 x_3 - (\mu^\alpha + {d_3}^\alpha + \gamma_2^\alpha) x_4,\\
&{}_0^C D_t^\alpha x_5(t) = \tau_1^\alpha x_2 - (\mu^\alpha + {d_1}^\alpha + \psi^\alpha) x_5,\\
&{}_0^C D_t^\alpha x_6(t) = \gamma_1^\alpha x_2 + \gamma_2^\alpha x_4 + \gamma_3^\alpha x_3 + \psi^\alpha x_5 - (\mu^\alpha + \theta^\alpha) x_6.
\end{aligned} \tag{3}
$$

where $f_1 = \beta_1^\alpha (x_2 + x_4)$ and $f_2 = \beta_2^\alpha (x_3 + x_4)$ and the initial values of system (3) is nonnegative $x_1(0) \geq 0, x_2(0) \geq 0, x_3(0) \geq 0, x_4(0) \geq 0, x_5(0) \geq 0, x_6(0) \geq 0$.

3 RESULT AND DISCUSSION

3.1 Equilibrium Point of Influenza-Meningitis Coinfection Fractional Model

We obtained the disease-free equilibrium (DFE) point of the system by setting all the equation equal to zero and providing that $x_2^0 = x_3^0 = x_4^0 = 0$. We denoted the DFE point as follows

$$
X_{fm}^0 = (1,0,0,0,0,0)
$$

On the other side, the endemic equilibrium (EE) point is denoted by ${X_{fm}}^* = (x_1^*, x_2^*, x_3^*, x_4^*, x_5^*, x_6^*)$. The EE point exists when $x_2 = x_3 = x_4 \neq 0$ which means that the disease persists among the community. By making all equations in (3) equal to zero and performing some algebraic manipulation, we obtain EE point for influenza meningitis coinfection model as follows.

$$x_1^* = \frac{\mu^\alpha + \theta^\alpha x_6}{f_1 + f_2 + \mu^\alpha};$$
$$x_2^* = \frac{f_1 x_1}{\mu^\alpha + {d_1}^\alpha + \tau_1^\alpha + \gamma_1^\alpha + \phi f_2};$$
$$x_3^* = \frac{f_2 x_1}{\mu^\alpha + {d_2}^\alpha + \gamma_3^\alpha + \omega f_1};$$
$$x_4^* = \frac{\{\phi(\mu^\alpha + {d_2}^\alpha + \gamma_3^\alpha + \omega f_1) + \omega(\mu^\alpha + {d_1}^\alpha + \tau_1^\alpha + \gamma_1^\alpha + \cdot}{(\mu^\alpha + {d_3}^\alpha + \gamma_2^\alpha)(\mu^\alpha + {d_1}^\alpha + \tau_1^\alpha + \gamma_1^\alpha + \phi f_2)(\mu^\alpha + {d_2}^\alpha} \quad (4)$$
$$x_5^* = \frac{\tau_1^\alpha f_1 x_1}{(\mu^\alpha + {d_1}^\alpha + \psi^\alpha)(\mu^\alpha + {d_1}^\alpha + \tau_1^\alpha + \gamma_1^\alpha}$$
$$x_6^* = \frac{\gamma_1^\alpha x_2 + \gamma_2^\alpha x_4 + \gamma_3^\alpha x_3 + \psi^\alpha x_5}{(\mu^\alpha + \theta^\alpha)};$$

3.2 Basic Reproduction Number

The Next generation Matrix method (Dreessche & Watmough, 2002) is employed to obtain the reproduction number of influenza – meningitis coinfection. First, we consider the disease class $X = (x_2, x_3, x_4, x_5)$ in F and V

$$F = \begin{pmatrix} f_1 x_1 \\ f_2 x_1 \\ 0 \\ 0 \end{pmatrix},$$
$$V = \begin{pmatrix} (\mu^\alpha + {d_1}^\alpha + \tau_1^\alpha + \gamma_1^\alpha + \phi f_2)x_2 \\ (\mu^\alpha + {d_2}^\alpha + \gamma_3^\alpha + \omega f_1)x_3 \\ -\phi f_2 x_2 - \omega f_1 x_3 + (\mu^\alpha + {d_3}^\alpha + \gamma_2^\alpha)x_4 \\ -\tau_1^\alpha x_2 + (\mu^\alpha + {d_1}^\alpha + \psi^\alpha)x_5 \end{pmatrix}$$

We evaluate the matrices F and V at X^0

$$F = \begin{bmatrix} \beta_1^\alpha & 0 & \beta_1^\alpha & 0 \\ 0 & \beta_2^\alpha & \beta_2^\alpha & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad (5)$$

And

$$V = \begin{bmatrix} k_1 & 0 & 0 & 0 \\ 0 & k_2 & 0 & 0 \\ 0 & 0 & k_3 & 0 \\ 0 & 0 & 0 & k_4 \end{bmatrix} \quad (6)$$

$k_1 = \mu^\alpha + {d_1}^\alpha + \tau_1^\alpha + \gamma_1^\alpha$
$k_2 = \mu^\alpha + {d_2}^\alpha + \gamma_3^\alpha$
$k_3 = \mu^\alpha + {d_3}^\alpha + \gamma_2^\alpha$
$k_4 = \mu^\alpha + {d_1}^\alpha + \psi^\alpha$

The matrix FV^{-1} becomes as

$$FV^{-1} = \begin{bmatrix} \frac{\beta_1^\alpha}{k_1} & 0 & \frac{\beta_1^\alpha}{k_3} & 0 \\ 0 & \frac{\beta_2^\alpha}{k_2} & \frac{\beta_2^\alpha}{k_3} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad (7)$$

Then, we obtained the corresponding eigen values of the next matrix generation FV^{-1} are

$$R_1 = \frac{\beta_1^\alpha}{\mu^\alpha + {d_1}^\alpha + \tau_1^\alpha + \gamma_1^\alpha}, R_2 = \frac{\beta_2^\alpha}{\mu^\alpha + {d_2}^\alpha + \gamma_3^\alpha}.$$

Thus, the reproduction number is

$R_0 = max\{R_1, R_2\}$.

3.3 Local Stability Analysis of DFE

We begin the local-stability analysis by forming a Jacobian Matrix respect to X_{fm}^0 as follows.

$$J(E^0) = \begin{bmatrix} -\mu^\alpha & -\beta_1^\alpha & -\beta_2^\alpha & -\beta_1^\alpha - \beta_2^\alpha & 0 & \theta^\alpha \\ 0 & -k_1 & 0 & 0 & 0 & 0 \\ 0 & 0 & -k_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & -k_3 & 0 & 0 \\ 0 & \tau_1^\alpha & 0 & 0 & -k_4 & 0 \\ 0 & \gamma_1^\alpha & \gamma_3^\alpha & \gamma_2^\alpha & \psi^\alpha & -(\mu^\alpha + \theta^\alpha). \end{bmatrix} \quad (8)$$

From Equation (8), we obtained a characteristic polynomial

$$(\lambda + \mu^\alpha)(\lambda + \mu^\alpha + {d_1}^\alpha + \tau_1^\alpha + \gamma_1^\alpha)(\lambda + \mu^\alpha + {d_2}^\alpha + \gamma_3^\alpha)(\lambda + \mu^\alpha + {d_3}^\alpha + \gamma_2^\alpha)(\lambda + \mu^\alpha + {d_1}^\alpha + \psi^\alpha)(\lambda + \mu^\alpha + \theta^\alpha) = 0 \quad (9)$$

Based on Equation (9) we obtained the eigen values

$\lambda_1 = -\mu^\alpha;$
$\lambda_2 = -(\mu^\alpha + {d_1}^\alpha + \tau_1^\alpha + \gamma_1^\alpha);$
$\lambda_3 = -(\mu^\alpha + {d_2}^\alpha + \gamma_3^\alpha);$
$\lambda_4 = -(\mu^\alpha + {d_3}^\alpha + \gamma_2^\alpha)$
$\lambda_5 = -(\mu^\alpha + {d_1}^\alpha + \psi^\alpha)$
$\lambda_6 = -(\mu^\alpha + \theta^\alpha)$

Because we have all parameters $\mu, d_1, d_2, d_3, \tau_1, \gamma_1, \gamma_2, \gamma_3, \psi, \theta > 0$, it implies $\lambda_{i,i=1\ldots6} < 0$ and $|\arg(\lambda_i)| = \pi$. Therefore, it can be guaranteed that $|\arg(\lambda_i)| > \frac{\alpha\pi}{2}$ for all $0 < \alpha \leq 1$ and X_{fm}^0 is local asymptotically stable ■

3.4 Numerical Simulation Findings

This section shows various numerical simulation of Influenza and Meningitis coinfection model to analyse the transmission dynamics. The initial conditions are set to be $x_1(0) = 0.7$, $x_2(0) = 0.1$, $x_3(0) = 0.1$, $x_4(0) = 0.05$, $x_5(0) = 0.001$, $x_6(0) = 0.049$ and the parameter values are provided in Table 1.

Table 1. Parameters of the Model.

Parameters	Description	Value	Source
μ	Natural birth/death rate	0.02	(Varshney & Dwivedi, 2021)
θ	Loss of Immunity	0.00735	(Kotola & Mekonnen, 2022)
β_1	Influenza contact Rate	2.343	(Varshney & Dwivedi, 2021)
β_2	Meningitis Contact rate	0.9	(Kotola & Mekonnen, 2022)
d_1	Influenza only caused death rate	0.001	(Jonnalagadda, 2022)
τ_1	The rate of discovered influenza moved to quarantine	0.5 – 2	(Varshney & Dwivedi, 2021)
γ_1	Natural Recovery rate of Influenza only	0.14	(Jonnalagadda, 2022)
d_2	Meningitis only caused death rate	0.002	(Kotola & Mekonnen, 2022)
d_3	Influenza and Meningitis co infection death rate	0.2	(Varshney & Dwivedi, 2021)
γ_2	Recovery rate of Influenza and Meningitis co infection	0.04	Assumed
γ_3	Natural recovery rate of Meningitis only	0.02	(Kotola & Mekonnen, 2022)
ϕ	Modification parameter	1	Assumed
ω	Modification parameter	1	Assumed
ψ	The average spent in isolation	0.244	(Varshney & Dwivedi, 2021)

We employ the Predictor-corrector (PECE) with Product Integration (PI) rules method developed by Garrappa (Garrappa, 2018) in MATLAB to perform numerical simulation for several values of fractional order α. It aims to analyse the dynamical behaviour of each population. By using PECE-PI method we get numerical expression to solve the system (3) as follows

$$x_{i_n}^{p} = T_{m-1}[x_i; t_0](t_n) + h^{\alpha} \sum_{j=0}^{n-1} b_{n-j-1}^{(\alpha)} g_i\left(t_j, x_{i_j}\right) \quad (10)$$

$$x_{i_n} = T_{m-1}[x_i; t_0](t_n) + h^{\alpha}\left(\tilde{a}_n^{(\alpha)} g_i(0) + \sum_{j=1}^{n-1} a_{n-j}^{(\alpha)} g_i\left(t_j, x_{i_j}\right) + a_0^{(\alpha)} g_i(t_n, x_{i_n}^{p})\right) \quad (11)$$

Where $i = 1,2, \ldots 6$,

$b_n^{(\alpha)} = \frac{((n+1)^{\alpha} - n^{\alpha})}{\Gamma(\alpha+1)}$, $\tilde{a}_n^{(\alpha)} = \frac{(n-1)^{\alpha+1} - n^{\alpha}(n-\alpha-1)}{\Gamma(\alpha+2)}$,

and

$$a_n^{(\alpha)} = \begin{cases} \frac{1}{\Gamma(\alpha+2)}, & n = 0 \\ \frac{(n-1)^{\alpha+1} - 2n^{\alpha+1} + (n+1)^{\alpha+1}}{\Gamma(\alpha+2)}, & n = 1,2, \ldots \end{cases}$$

Figure 2 illustrates the dynamics of the proportion of individuals in the susceptible compartment with various fractional orders. In this simulation, the four alpha values exhibit different dynamics. At the beginning of the simulation, all four show a decrease in proportion. It is evident that the fractional orders closer to 1 have a graph that tends to be more fluctuating before reaching the endemic equilibrium point. Figure 3 demonstrates the dynamics of the compartment of individuals infected with influenza with different fractional orders. The results indicate that during the first 1-5 days, the population infected with influenza shows an increase, followed by a decrease in the number of infections due to individuals recovering, receiving quarantine measures, or acquiring secondary infections and moving to the influenza-meningitis co-infection compartment. The larger the fractional order, the faster the approach to the endemic point.

Figure 4 shows the simulation results for the proportion of individuals infected with meningitis for several different fractional orders. During the first 2-3 days, the proportion of infected individuals increases and then decreases due to individuals acquiring secondary infections or recovering. The different fractional orders exhibit varying behaviours. The fractional order α=0.95 tends to be more fluctuating compared to the other fractional orders.

Figure 5 illustrates the dynamics of the proportion of individuals in the influenza-meningitis co-infection compartment. The simulation results indicate an increase in cases during the first 10 days of the simulation. Subsequently, the proportion of individuals in the co-infection compartment decreases towards the endemic equilibrium point. It is observed that smaller fractional orders have relatively smaller fluctuations compared to other fractional orders, and therefore tend to reach the endemic equilibrium point more slowly.

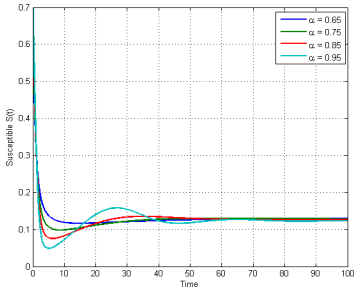

Figure 2: Behaviour of Susceptible Population with different values of α.

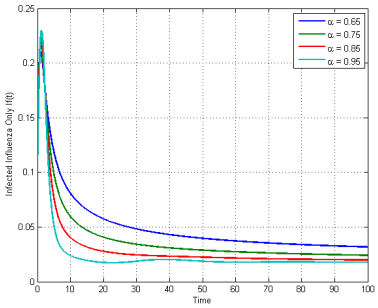

Figure 3: Behaviour of Infected Influenza Only with different values of α.

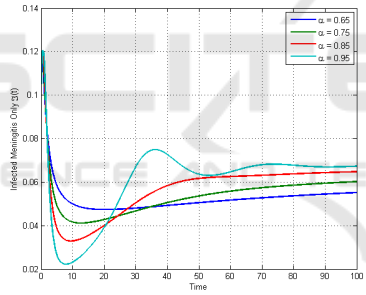

Figure 4: Behaviour of Infected Meningitis Only with different values of α.

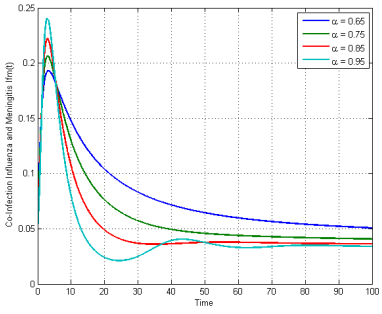

Figure 5: Behaviour of Influenza-Meningitis Coinfection with different values of α.

Figure 6 shows the dynamics of the proportion of individuals with influenza who are quarantined with various fractional orders α. In the initial days, the proportion of quarantined individuals increases in line with the rising number of influenza infections. Subsequently, the proportion of quarantined individuals decreases due to the recovery of individuals or the decreasing number of influenza infections

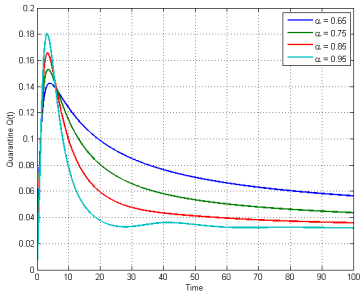

Figure 6: Behaviour of Quarantine Population with different values of α.

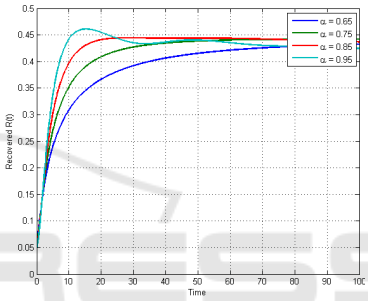

Figure 7: Behaviour of Recovered Population with different values of α.

Figure 7 presents the simulation results depicting the recovered subpopulation with different fractional orders α. In the first 30 days, the number of recoveries increases before reaching a relatively stagnant phase. It is observed that smaller fractional orders α reach a steady state condition relatively faster due to the memory effect of the susceptible population

Figures 8, 9, 10, and 11 illustrate various contact rates for infected influenza (β_1) and infected meningitis (β_2) on the populations infected with influenza only and meningitis only. Figure 8 shows that the larger the contact rate for infected influenza, the higher the proportion of individuals who will be infected with influenza. Figure 9 shows an inverse result: an increase in the contact rate for infected influenza reduces the proportion of individuals infected with meningitis. This is because more individuals progressing to the influenza compartment reduces the proportion of susceptible individuals, thereby lowering the proportion of individuals infected with meningitis. Figures 10 and 11 exhibit similar behavior to the previous cases, where an increase in the contact rate for infected meningitis

increases the proportion of individuals infected with meningitis (Figure 11) but, on the other hand, decreases the proportion of individuals infected with influenza (Figure 10).

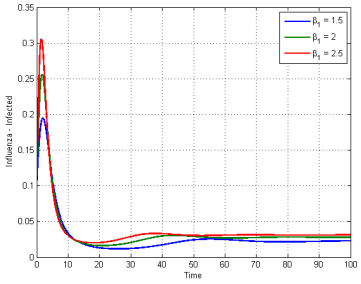

Figure 8: Infected Influenza Only with different contact rate β_1.

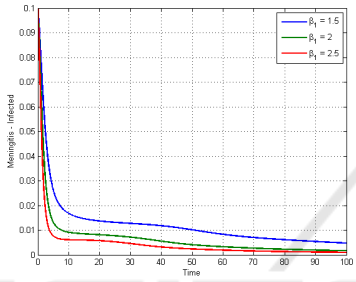

Figure 9: Infected Meningitis Only with different contact rate β_1.

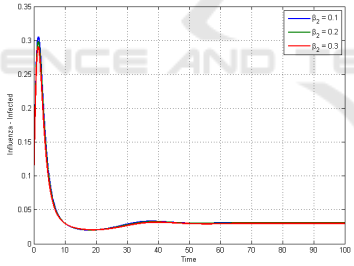

Figure 10: Infected Influenza Only with different contact rate β_2.

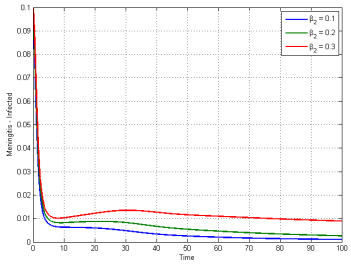

Figure 11:Infected Meningitis Only with different contact rate β_2.

Figures 12, 13, and 14 present simulation results for various quarantine rates. The higher the quarantine rate for influenza, the lower the increase in the proportion of individuals infected with influenza only (Figure 12). This is consistent with the lower increase in the proportion of co-infected individuals when the quarantine rate for influenza-infected individuals rises (Figure 14). However, an increase in the quarantine rate does not reduce the proportion of individuals infected with meningitis. A high quarantine rate for influenza-infected individuals reduces the spread of influenza, which gradually increases the proportion of meningitis infections in the population (Figure 13).

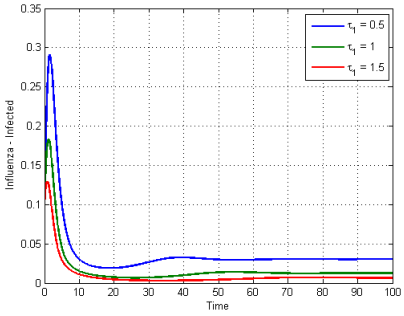

Figure 12: Infected Influenza - Only with different quarantine rate.

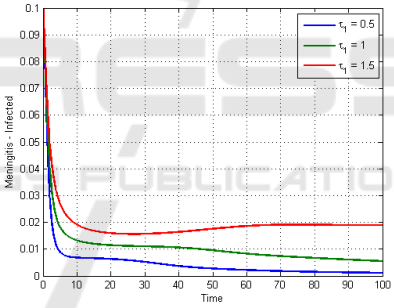

Figure 13: Infected Meningitis - Only with different quarantine rate.

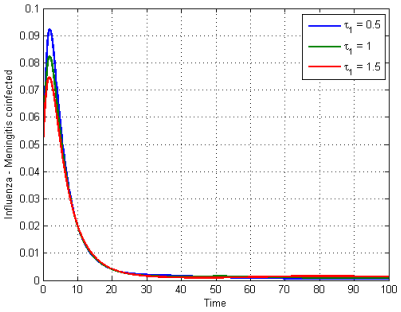

Figure 14: Influenza - Meningitis Coinfection with different quarantine rate.

4 CONCLUSIONS

This research extended the deterministic model of Influenza-Meningitis coinfection transmission dynamics to a generalized Caputo fractional derivative to consider the memory effect of a biological system. We demonstrated the qualitative properties of the model to ensure its biological relevance, specifically focusing on coinfection transmission. Using the next-generation method, we obtained two equilibrium points and computed the basic reproduction number for Influenza-Meningitis coinfection. Furthermore, we analysed the local stability condition of the disease-free equilibrium. Additionally, we performed numerical simulations with several values of fractional order, influenza transmission rate, meningitis transmission rate, and quarantine rate to explore their effects to the transmission. Different values of the fractional order indicated varying speeds of reaching a steady state or endemic level.

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