Research Article

Novel Numerical Estimates of the Pneumonia and Meningitis Epidemic Model via the Nonsingular Kernel with Optimal Analysis

Saima Rashid,1 Bushra Kanwal,2 Abdulaziz Garba Ahmad,3 Ebenezer Bonyah,4 and S.K. Elagan5,6

1Department of Mathematics, Government College University, Faisalabad 38000, Pakistan
2Department of Mathematics, COMSATS University, Islamabad, Pakistan
3Department of Mathematics, National Mathematical Centre Abuja, Abuja 900211, Nigeria
4Department of Mathematics Education, University of Education, Winneba, Kumasi Campus, Ghana
5Department of Mathematics and Computer Sciences, Faculty of Science Menoufia University, Shebin, Elkom 32511, Egypt
6Department of Mathematics and Statistics, College of Science, Taif University, P. O. Box 11099, Taif 21944, Saudi Arabia

Correspondence should be addressed to Ebenezer Bonyah; ebonyah@aamusted.edu.gh

Received 10 April 2022; Accepted 30 May 2022; Published 31 July 2022

Academic Editor: Fathalla A. Rihan

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In this article, we investigated a deterministic model of pneumonia-meningitis coinfection. Employing the Atangana–Baleanu fractional derivative operator in the Caputo framework, we analyze a seven-component approach based on ordinary differential equations (ODEs). Furthermore, the invariant domain, disease-free as well as endemic equilibria, and the validity of the model’s potential results are all investigated. According to controller design evaluation and modelling, the modulation technique devised is effective in diminishing the proportion of incidences in various compartments. A fundamental reproducing value is generated by exploiting the next generation matrix to assess the properties of the equilibrium. The system’s reliability is further evaluated. Sensitivity analysis is used to classify the impact of each component on the spread or prevention of illness. Using simulation studies, the impacts of providing therapy have been determined. Additionally, modelling the appropriate configuration demonstrated that lowering the fractional order from 1 necessitates a rapid initiation of the specified control technique at the largest intensity achievable and retaining it for the bulk of the pandemic’s duration.

1. Introduction

Fractional calculus has gained popularity over the years for representing a plethora of new challenges in fields such as computational virology, quantum theory, technology, and numerous others, wherein fractional-order (FO) operators are either singular (Caputo derivative and Riemann–Liouville (RL) fractional derivatives) or nonsingular (Atangana–Baleanu and Caputo–Fabrizio derivatives) [1–4].

However, the variation between integer-order and FO derivatives is that the integer-order derivative depicts the functionality of a complex nonlinear network for the entirety of the period, whereas the FO derivative operator represents a characteristic of a logistic scheme for the enormous moment. Furthermore, the integer-order derivative reflects a dynamic state’s spatial information, while the FO derivative formulation of a complex process encompasses the project process domain [5–8]. On the other hand, in modelling specific cases, implementing derivation operators via non-integer values is critical for articulating generational requirements and the reliability of memories as a key component of various systems [9, 10].

Therefore, the advent of multiple meanings of a fractional derivative is fascinating and creates an incentive to identify the intricacies of natural surroundings in the context that certain challenges in existence pursue the index law for the RL fractional operator, some also implement the Mittag-Leffler (ML) rules for the Atangana–Baleanu fractional
derivative operator, and many others try to emulate the exponentially decaying law for the Caputo–Fabrizio fractional operator or an amalgamation of such regulations [11, 12].

Furthermore, the fact that many nonlinear mechanisms in connection to complex processes are discovered to be nonlocal with protracted recollection in time, and that inherently fractional derivation operators can characterize such kinds of mechanisms more appropriately than integer derivatives, has resulted in a rapid boost in the description of various nonlinear dynamic structures utilizing fractional-order derivatives [13]. In other respects, fractional-order operators are the ideal way to characterize or reveal crucial characteristics of several complex processes.

Pneumonia, which is classified as an infectious agent, is responsible for the loss of individual lives worldwide due to the inhalation of potential pathogens, primarily Streptococcus pyogenes [14]. Other disorders susceptible to infection include meningitis, respiratory ailments, and nasal congestion, among others (see Figure 1).

Infections can occur in adults, including infants to adults, and pneumonia turns severe whenever the immune system is weakened, as it is in vulnerable populations, as well as when it is co-infected with several other infections, such as meningitis [15]. Meningitis is a contagious bacterial ailment of the membrane that surrounds the cerebrum and spinal cord [16] (see Figures 2(a) and 2(b)).

Meningitis is caused by viral illness in 80 percent of cases, which is caused by pneumococcal pneumonia, streptococci, and Neisseria meningitidis. A majority of the research was carried out to determine the governing strategies of contagious ailments in a population (see [17–19]). In order to analyze the evolution of contagious infections in a population, various researchers have designed simulations to examine the evolution of various contagious infections. The authors [20, 21] also mentioned the co-dynamics of pneumonia and水borne illnesses [22], as well as economic evaluation and optimal monitoring. The findings of this investigation demonstrated that among the suggested techniques, preventing infectious disease and treating pneumococcal meningitis seem to be the most cost-effective. Many researchers have examined co-infection of communicable ailments with HIV and pneumococcal co-infection [23], while Akinyi et al. [24] looked into bacterial meningitis and plasmodium co-infection.

To the best of our knowledge, since its invention in 2016 by Atangana and Baleanu [2], the innovative fractional derivative operator has been significantly employed in a multitude of disciplines of scientific innovation [25, 26]. For a short period of time, modelling using the nonsingular fractional derivatives culminates in a stochastic, deterministic, and physical process [27–29]. Atangana [30] presented the extension of rate of change concept from local to nonlocal operators with applications. Baleanu et al. [31] constructed a fractional framework for a malignant cell's ability to use information and assessed how chemotherapeutics influenced the system. As a consequence of the observations, the optimum modulation technique was found to be effective. Sene [32] studied the formulation of the governing equations of a fractional diffusion equation in the setting of the fractional operator with a Rabotnov fractional exponential kernel. Zhao [33] presented the fuzzy-based strategy to suppress the novel coronavirus (2019-NCOV) massive outbreak.

To approach the ABC fractional derivative, Owolabi [34] adopted a two-step family of Adams–Bashforth procedures to perform evaluation and modelling techniques on a problem. Demirci et al. [35] took into account an SEIR fractional model and its experimental validation. In [36], the SEIRA mathematical model is examined using the ABC fractional derivative operator with the ML kernel. Thus, we can conclude from the above-mentioned research that fractional derivatives have several implications in numerical techniques and in the study of real-world processes. The newly formed Atangana–Baleanu fractional operator, notably, has gained appreciation and acceptance as a result of its variety of uses in ecological, chemical, and biomedical sciences, as well as a variety of other complex studies.

In this research, we investigate a pneumonia and meningitis (P) mathematical formulation with a dominating interaction incidence, which is inspired by the aforesaid considerations. The fractional derivative formulation of the system is generated, and its analytical simulation is estimated by applying the newly reported Atangana–Baleanu fractional derivative and Toufik–Atangana numerical solutions [37]. The P epidemiological theory utilizing the Atangana–Baleanu fractional derivative has not previously been examined, to the best of the researchers’ expertise. The researchers further claim that in the relevant research, robust regulation characterization of mathematical formulae in the context of Atangana–Baleanu fractional operators is infrequent in the earlier research. As a response, we examine the sensitivity characterization of the P model in this article.

2. Depiction of the Model

In this part, the ABC fractional derivative form of the P epidemic mathematical systems is introduced. Let us just continue with a review of the ML kernels’ notions and their concerning consequences.

Definition 1 (see [2]). For $\beta \in [0, 1]$, $c < d$ and $\mathcal{F}_1 \in H^1(c, d)$; then, the ABC derivative of fractional-order for $\mathcal{F}_1$ is presented as

$$
\text{ABC} D_{c+}^{\beta} \mathcal{F}_1 (\xi) = \Psi (\beta) \frac{\mathcal{M} (\beta)}{(1 - \beta)} \int_c^\xi \frac{\mathcal{F}_1 (\eta)}{\xi^{\beta - 1}} \left( \frac{\eta}{\beta} \right)^{\beta - 1} d\eta,
$$

where $\Psi (\beta)$ is a normalization mapping that satisfying $\Psi (0) = \Psi (1) = 1$ and the Mittag–Leffler function is denoted by $E_{\beta} (z_1)$ defined as

$$
E_{\beta} (z_1) = \sum_{y=0}^{\infty} \frac{z_1^y}{\Gamma (\beta y + 1)}, \beta, z_1 \in \mathbb{C}, \Re (\beta) > 0.
$$
Definition 2 (see [2]). For $\beta \in [0, 1]$, $c < d$ and $F_1 \in H^1(c, d)$, then, the AB fractional integral of $F_1$ is presented as

$$\frac{AB}{c} \int_c \beta F_1(\zeta) = \frac{(1 - \beta)}{M(\beta)} F_1(\zeta) + \frac{\beta}{\Gamma(\beta)M(\beta)} \int_c \frac{F_1(\theta)}{(\zeta - \theta)^{\beta-1}} d\theta.$$  

(3)

Lemma 1 (see [38]) (Newton–Leibniz identity). For $F_1 \in C^1(c, d)$, the ABC fractional derivative and integral for $F_1$ hold:

$$\frac{AB}{c} \int_c \beta F_1(\zeta) = F_1(\zeta) - F_1(c).$$  

(4)

Lemma 2 (see [39]). For $c < d$, $F_1, F_2 \in H^1(c, d)$, then the ABC fractional derivative holds for the subsequent variant:

$$\frac{AB}{c} D_\zeta^\beta F_1(\zeta) = F_1(\zeta) - F_1(c).$$

(5)

Our next result is the generalized mean-value theorem, which is mainly due to [40].

Lemma 3 (see [40]). Let there be a function $h_1(\varrho) \in C[c, d]$ and also suppose $\frac{AB}{c} D_\zeta^\beta h(\varrho) \in C[c, q_2], \beta \in (0, 1)$. Then,

$$h_1(\varrho) = h_1(c) + 1/\Gamma(\beta) \frac{AB}{c} D_\zeta^\beta h_1(\theta)(\varrho - c)^{\beta - 1}, \theta \in [0, \varrho].$$

Followed by Lemma 3, for $\beta \in (0, 1)$, if $h_1(\varrho) \in [0, d]$, $\frac{AB}{c} D_\zeta^\beta h_1(\varrho) \in (0, d]$, and $\frac{AB}{c} D_\zeta^\beta h_1(\varrho) \geq 0$ for all $\varrho \in (0, d]$, then the mapping $h_1(\varrho)$ is increasing. Otherwise, $h_1(\varrho)$ is said to be decreasing for all $\varrho \in [0, d]$.

We shall now continue on to the system’s development. The numerical framework for this...
investigation has been developed utilizing the sequence chart below (Figure 3).

The numerical approach with numerical approximation employed in this work is represented by the governing  
formulae, which are predicated on the workflow.

\[
\begin{align*}
S(\zeta) &= \bar{\pi} + \rho_1 R_p + \phi_2 R_m + \phi_3 R_{pm} - (g_1 + g_2 + \rho) S, \\
I_p(\zeta) &= g_1 S - (g_2 + \rho + \rho_1 + \eta_1) I_p, \\
I_m(\zeta) &= g_2 S - (g_1 + \rho + \rho_2 + \eta_2) I_m, \\
I_{pm}(\zeta) &= g_2 I_p + g_1 I_m - (\rho + \rho + \eta_1 + \eta_2) I_{pm}, \\
R_p(\zeta) &= \rho_1 I_p + \epsilon \rho I_{pm} - (\phi_1 + \phi) R_p, \\
R_m(\zeta) &= \rho_2 I_m + \epsilon \rho h_1 (1 - \epsilon) I_{pm} - (\phi_2 + \phi) R_m, \\
R_{pm}(\zeta) &= \rho (1 - h_1) (1 - \epsilon) I_{pm} - (\phi_3 + \phi) R_{pm}.
\end{align*}
\]

Here, we examine a community that is diversified in this scenario. In this framework, we explore the probabilistic seven-dimensional global species. In this framework, we explore the probabilistic seven dimensional global species such as sensitive group ($S$), pneumonia virulent ($I_p$), meningitis epidemic ($I_m$), pneumonia and meningitis co-infectious ($I_{pm}$), pneumonia healed ($R_p$), meningitis regained ($R_m$), and pneumonia-meningitis co-infectious retrieved are the numerous subgroups. ($R_{pm}$). Susceptible are recruited with rate of $\bar{\pi}$ through birth or immigration, and their number increases from individuals that come from subclasses of pneumonia recovered, meningitis recovered, and coinfectious recovered by losing their temporary immunity with rate of $\phi_1, \phi_2,$ and $\phi_3,$ respectively. In the entire susceptible population, individuals can get pneumonia with contact rate of $q_1$ from a pneumonia-only infected or coinfectious person with force of infection of pneumonia $g_1 = q_1 (I_p + I_{pm})/N$ and join $I_p$ compartment. In a similar way, individuals can get meningitis by a contact rate of $q_2$ from a meningitis-only infected or coinfectious person with force of infection of meningitis $g_2 = q_2 (I_m + I_{pm})/N$ and join $I_m$ compartment. Pneumonia-only infected individuals also can get an additional meningitis infection with a force of infection $g_2$ and join coinfectious compartment ($I_{pm}$). Participants emerge from meningitis merely affected by a segment when attacked by pneumonia using $q_1$ a high intensity of transmission to boost the post-operative subsystem. Patients who have only been afflicted with pneumonia may recuperate at a frequency of $\rho_1$ approximately and can represent the pneumonia only cured category ($I_p$). As a result, meningitis-only people who are contaminated recover at a rate of $\rho_2$ and are assigned to the meningitis-only healed group ($I_{pm}$). People in the immune-compromised class also retrieve at a speed $\rho,$ but they either heal just from pneumonia ailment and enter pneumonia only retrieved storage area, with the possibility of $\rho (1 - \epsilon),$ or revive just from meningitis ailment and participate in meningitis

\begin{figure}
\centering
\includegraphics[width=\textwidth]{PM_co-infection_model.png}
\caption{Flowchart for $PM$ co-infection model.}
\end{figure}
only retrieved zone with the plausibility of \( \rho h_1 (1 - e) \), or restore from all these maladies and participate in co-infected recapture zone with a likely hood of \( \rho (1 - e) (1 - h_1) \). Furthermore, \( q \) represents the natural death rate. Furthermore, \( \eta_1 \) is the relative risk induced entirely by pneumonia, and \( \eta_2 \) is the number of fatalities due to meningitis.

2.1. Model Configuration. In this part, we investigate the framework’s descriptive characteristics. We segmented the comprehensive framework into various DEs as presented in (5), which are estimates for pneumonia and meningitis, to make the process easier.

The combination of DEs describes formal system (6) that incorporates the hypotheses, the saturate interaction frequency, and the flowchart (Figure 3) and analyzes model (6) by ABC fractional derivative.

\[
\begin{align*}
\Theta_1 (\zeta, S) &= \pi + \phi_1 R_p + \phi_2 R_m + \phi_3 R_{pm} - (g_1 + g_2 + q) S, \\
\Theta_2 (\zeta, I_p) &= g_1 S - (g_2 + q + \rho_1 + \eta_1) I_p, \\
\Theta_3 (\zeta, I_m) &= g_2 S - (g_1 + q + \rho_2 + \eta_1) I_m, \\
\Theta_4 (\zeta, I_{pm}) &= g_3 I_p + g_4 I_m - (\rho + q + \eta_1 + \eta_2) I_{pm}, \\
\Theta_5 (\zeta, R_p) &= \rho_1 I_p + \epsilon p I_{pm} - (\phi_4 + q) R_p, \\
\Theta_6 (\zeta, R_m) &= \rho_2 I_m + \epsilon p h_1 (1 - e) I_{pm} - (\phi_3 + q) R_m, \\
\Theta_7 (\zeta, R_{pm}) &= \rho (1 - h_1) (1 - e) I_{pm} - (\phi_3 + q) R_{pm}, \\
\end{align*}
\]

subject to the nonnegative initial values \( S(0) = S_0, I_p(0) = I_{p0}, I_m(0) = I_{m0}, I_{pm}(0) = I_{pm0}, R_p(0) = R_{p0}, R_m(0) = R_{m0}, R_{pm}(0) = R_{pm0} \).

Thus, in the absence of disease, the differential equation of the total population size is \( d N / d \zeta = \pi - qN - \eta_1 (I_p + I_{pm}) - \eta_2 (I_m + I_{pm}) \). Table 1 summarizes the characteristics that were considered in the analysis (7).

2.2. Consequences on the Existence-Uniqueness. Here, employing the Banach fixed point \( f_p \) theorem for contraction mapping, the existence-uniqueness of the result for the ABC fractional model suggested in (7) is demonstrated. Before actually moving on, it is important to remember the two additional theorems. For further details, see [41] and the references cited therein.

We continue as follows to demonstrate the method’s existence-uniqueness. We acquire framework (7) while we implement the AB fractional integral.

\[
\begin{align*}
S(\zeta) - S(0) &= \frac{1 - \beta}{\eta(\beta)} \Theta_1 (\zeta, S) + \frac{\beta}{\eta(\beta) \Gamma(\beta)} \int_0^\zeta \Theta_1 (\zeta, S)(\zeta - c)^{\beta - 1} dc, \\
I_p(\zeta) - I_p(0) &= \frac{1 - \beta}{\eta(\beta)} \Theta_2 (\zeta, I_p) + \frac{\beta}{\eta(\beta) \Gamma(\beta)} \int_0^\zeta \Theta_2 (\zeta, I_p)(\zeta - c)^{\beta - 1} dc, \\
I_m(\zeta) - I_m(0) &= \frac{1 - \beta}{\eta(\beta)} \Theta_3 (\zeta, I_m) + \frac{\beta}{\eta(\beta) \Gamma(\beta)} \int_0^\zeta \Theta_3 (\zeta, I_m)(\zeta - c)^{\beta - 1} dc, \\
I_{pm}(\zeta) - I_{pm}(0) &= \frac{1 - \beta}{\eta(\beta)} \Theta_4 (\zeta, I_{pm}) + \frac{\beta}{\eta(\beta) \Gamma(\beta)} \int_0^\zeta \Theta_4 (\zeta, I_{pm})(\zeta - c)^{\beta - 1} dc, \\
R_p(\zeta) - R_p(0) &= \frac{1 - \beta}{\eta(\beta)} \Theta_5 (\zeta, R_p) + \frac{\beta}{\eta(\beta) \Gamma(\beta)} \int_0^\zeta \Theta_5 (\zeta, R_p)(\zeta - c)^{\beta - 1} dc, \\
R_m(\zeta) - R_m(0) &= \frac{1 - \beta}{\eta(\beta)} \Theta_6 (\zeta, R_m) + \frac{\beta}{\eta(\beta) \Gamma(\beta)} \int_0^\zeta \Theta_6 (\zeta, R_m)(\zeta - c)^{\beta - 1} dc, \\
R_{pm}(\zeta) - R_{pm}(0) &= \frac{1 - \beta}{\eta(\beta)} \Theta_7 (\zeta, R_{pm}) + \frac{\beta}{\eta(\beta) \Gamma(\beta)} \int_0^\zeta \Theta_7 (\zeta, R_{pm})(\zeta - c)^{\beta - 1} dc.
\end{align*}
\]
Table 1: Explanation of attributed values assumed in the model.

| Symbols | Value         | References |
|---------|---------------|------------|
| $\phi_1$ | 0.003–0.1     | [20]       |
| $\phi_2$ | 0.00904–0.99  | [16]       |
| $\phi_3$ | 0.01          | Supposed   |
| $q_1$    | 0.007–0.6     | [22]       |
| $q_2$    | 0.9           | [16]       |
| $\eta_1$ | 0.006–0.5     | Estimated  |
| $\eta_2$ | 0.002–0.2     | Estimated  |
| $\rho$   | 0.1           | [20]       |
| $h_1$    | 0.5–1.0       | [22]       |
| $\epsilon$ | 0.5–1.0     | Supposed   |
| $\rho_1$ | 0.01          | Supposed   |
| $\rho_2$ | 0.8           | Supposed   |

Surmise that the collection $\mathcal{B} = \mathcal{A}(\mathcal{F}) \times \mathcal{A}(\mathcal{F}) \times \mathcal{A}(\mathcal{F}) \times \mathcal{A}(\mathcal{F}) \times \mathcal{A}(\mathcal{F}) \times \mathcal{A}(\mathcal{F})$, where $\mathcal{A}(\mathcal{F}) = \mathbb{C}[0, T]$ indicates real-valued continuous mappings for the Banach space on $\mathcal{F} = [0, T]$ considering the norm presented as $\|S| + \|I_1\| + \|I_2\| + \|I_m\| + \|R_p\| + \|R_m\| + \|R_{pm}\|$, where $\|S\| = \sup_{t \in [0, T]}|S(\zeta)|$, $\|I_1\| = \sup_{t \in [0, T]}|I_1(\zeta)|$, $\|I_2\| = \sup_{t \in [0, T]}|I_2(\zeta)|$, $\|I_m\| = \sup_{t \in [0, T]}|I_m(\zeta)|$, $\|R_p\| = \sup_{t \in [0, T]}|R_p(\zeta)|$, $\|R_m\| = \sup_{t \in [0, T]}|R_m(\zeta)|$, and $\|R_{pm}\| = \sup_{t \in [0, T]}|R_{pm}(\zeta)|$.

The contraction and the Lipschitz hypothesis are the foundations of our following theorem.

**Theorem 1.** For the following kernels $\Theta_\ell, \ell = 1, 2, \ldots, 7$ in (7), there exists $L_\ell > 0, \ell = 1, 2, \ldots, 7$, such that

$$
\begin{align*}
\|\Theta_1(\zeta, S) - \Theta_1(\zeta, S_1)\| &\leq L_1\|S - S_1\|, \\
\|\Theta_2(\zeta, I_1) - \Theta_2(\zeta, I_{1,1})\| &\leq L_2\|I_1(\zeta) - I_{1,1}(\zeta)\|, \\
\|\Theta_3(\zeta, I_m) - \Theta_3(\zeta, I_{m,1})\| &\leq L_3\|I_m(\zeta) - I_{m,1}(\zeta)\|, \\
\|\Theta_4(\zeta, I_{pm}) - \Theta_4(\zeta, I_{pm,1})\| &\leq L_4\|I_{pm}(\zeta) - I_{pm,1}(\zeta)\|, \\
\|\Theta_5(\zeta, R_p) - \Theta_5(\zeta, R_{p,1})\| &\leq L_5\|R_p(\zeta) - R_{p,1}(\zeta)\|, \\
\|\Theta_6(\zeta, R_m) - \Theta_6(\zeta, R_{m,1})\| &\leq L_6\|R_m(\zeta) - R_{m,1}(\zeta)\|, \\
\|\Theta_7(\zeta, R_{pm}) - \Theta_7(\zeta, R_{pm,1})\| &\leq L_7\|R_{pm}(\zeta) - R_{pm,1}(\zeta)\|.
\end{align*}
$$

are contractions for $L_\ell \in [0, 1], \ell = 1, 2, \ldots, 7$.  

**Proof.** In order to satisfy Lipschitz assumptions, we have

$$
\begin{align*}
\|\Theta_1(\zeta, S) - \Theta_1(\zeta, S_1)\| = \| \pi + \phi_1R_p + \phi_2R_m + \phi_3R_{pm} - (g_1 + g_2 + \varrho)S \| - (\pi + \phi_1R_p + \phi_2R_m + \phi_3R_{pm} - (g_1 + g_2 + \varrho)S_1) | \| \
\leq \| (g_1 + g_2 + \varrho)(S - S_1) \| \leq (g_1 + g_2 + \varrho)\|S - S_1\| \leq L_1\|S - S_1\|, 
\end{align*}
$$

where $L_1 = q_1(\mathcal{A}(\mathcal{F}) \times \mathcal{A}(\mathcal{F}))$, $\|S\| = \sup_{t \in [0, T]}|S(\zeta)| \leq \mathcal{K}_1$, $\|I_1\| = \sup_{t \in [0, T]}|I_1(\zeta)| \leq \mathcal{K}_2$, $\|I_m\| = \sup_{t \in [0, T]}|I_m(\zeta)| = \mathcal{K}_3$, $\|I_{pm}\| = \sup_{t \in [0, T]}|I_{pm}(\zeta)| = \mathcal{K}_4$, $\|R_p\| = \sup_{t \in [0, T]}|R_p(\zeta)| = \mathcal{K}_5$, $\|R_m\| = \sup_{t \in [0, T]}|R_m(\zeta)| = \mathcal{K}_6$, and $\|R_{pm}\| = \sup_{t \in [0, T]}|R_{pm}(\zeta)| = \mathcal{K}_7$.

It is worth noting that $\Theta_1(\zeta, S_1)$ holds the Lipschitz assumption containing Lipschitz constant $L_1 = q_1(\mathcal{A}(\mathcal{F}) \times \mathcal{A}(\mathcal{F}))$. Furthermore, if $L_1 \in [0, 1)$, then $\Theta_1(\zeta, S_1)$ is a contraction.

Analogously, we can analyze the existence consequences of $L_\ell \in [0, 1]$ and the contraction technique for
\[ \Theta_1(\zeta, S_{n-1}) + \Theta_2(\zeta, I_{n-1}) + \Theta_3(\zeta, I_{pmn}) + \Theta_4(\zeta, R_{pn}) + \Theta_5(\zeta, R_{mn}) + \Theta_6(\zeta, R_{m}) + \Theta_7(\zeta, R_{pmn}) \text{ for } L_\ell \in [0, 1], \ell = 2, 3, \ldots, 7. \]

At \( \zeta = \zeta_n, n = 1, 2, \ldots \), introduce the subsequent recursive version of (9):

\[
\begin{align*}
S_n(\zeta) &= \frac{1 - \beta}{M(\beta)} \Theta_1(\zeta, S_{n-1}) + \frac{\beta}{M(\beta)\Gamma(\beta)} \int_0^\zeta \Theta_1(\zeta, S_{n-1})(\zeta - \zeta)^{\beta - 1} d\zeta, \\
I_{p_n}(\zeta) &= \frac{1 - \beta}{M(\beta)} \Theta_2(\zeta, I_{p_{n-1}}) + \frac{\beta}{M(\beta)\Gamma(\beta)} \int_0^\zeta \Theta_2(\zeta, I_{p_{n-1}})(\zeta - \zeta)^{\beta - 1} d\zeta, \\
I_{m_n}(\zeta) &= \frac{1 - \beta}{M(\beta)} \Theta_3(\zeta, I_{m_{n-1}}) + \frac{\beta}{M(\beta)\Gamma(\beta)} \int_0^\zeta \Theta_3(\zeta, I_{m_{n-1}})(\zeta - \zeta)^{\beta - 1} d\zeta, \\
I_{pmn}(\zeta) &= \frac{1 - \beta}{M(\beta)} \Theta_4(\zeta, I_{pmn_{n-1}}) + \frac{\beta}{M(\beta)\Gamma(\beta)} \int_0^\zeta \Theta_4(\zeta, I_{pmn_{n-1}})(\zeta - \zeta)^{\beta - 1} d\zeta, \\
R_{p_n}(\zeta) &= \frac{1 - \beta}{M(\beta)} \Theta_5(\zeta, R_{p_{n-1}}) + \frac{\beta}{M(\beta)\Gamma(\beta)} \int_0^\zeta \Theta_5(\zeta, R_{p_{n-1}})(\zeta - \zeta)^{\beta - 1} d\zeta, \\
R_{m_n}(\zeta) &= \frac{1 - \beta}{M(\beta)} \Theta_6(\zeta, R_{m_{n-1}}) + \frac{\beta}{M(\beta)\Gamma(\beta)} \int_0^\zeta \Theta_6(\zeta, R_{m_{n-1}})(\zeta - \zeta)^{\beta - 1} d\zeta, \\
R_{pmn}(\zeta) &= \frac{1 - \beta}{M(\beta)} \Theta_7(\zeta, R_{pmn_{n-1}}) + \frac{\beta}{M(\beta)\Gamma(\beta)} \int_0^\zeta \Theta_7(\zeta, R_{pmn_{n-1}})(\zeta - \zeta)^{\beta - 1} d\zeta.
\end{align*}
\]

subject to ICs \( S(0) = S_0, I_p(0) = I_{p0}, I_m(0) = I_{m0}, I_{pm}(0) = I_{pm0}, R_p(0) = R_{p0}, R_m(0) = R_{m0}, R_{pm}(0) = R_{pm0} \).

In (12), the variations of successive components are written in the following form:

\[
\begin{align*}
\Xi_{n}(\zeta) &= S_n(\zeta) - S_{n-1}(\zeta) = \frac{1 - \beta}{M(\beta)} \Theta_1(\zeta, S_{n-1}) - \Theta_1(\zeta, S_{n-2}) + \frac{\beta}{M(\beta)\Gamma(\beta)} \int_0^\zeta \Theta_1(\zeta, S_{n-1}) - \Theta_1(\zeta, S_{n-2})(\zeta - \zeta)^{\beta - 1} d\zeta, \\
\Xi_{2n}(\zeta) &= I_{p_n}(\zeta) - I_{p_{n-1}}(\zeta) = \frac{1 - \beta}{M(\beta)} \Theta_2(\zeta, I_{p_{n-1}}) - \Theta_2(\zeta, I_{p_{n-2}}) + \frac{\beta}{M(\beta)\Gamma(\beta)} \int_0^\zeta \Theta_2(\zeta, I_{p_{n-1}}) - \Theta_2(\zeta, I_{p_{n-2}})(\zeta - \zeta)^{\beta - 1} d\zeta, \\
\Xi_{3n}(\zeta) &= I_{m_n}(\zeta) - I_{m_{n-1}}(\zeta) = \frac{1 - \beta}{M(\beta)} \Theta_3(\zeta, I_{m_{n-1}}) - \Theta_3(\zeta, I_{m_{n-2}}) + \frac{\beta}{M(\beta)\Gamma(\beta)} \int_0^\zeta \Theta_3(\zeta, I_{m_{n-1}}) - \Theta_3(\zeta, I_{m_{n-2}})(\zeta - \zeta)^{\beta - 1} d\zeta, \\
\Xi_{4n}(\zeta) &= I_{pmn}(\zeta) - I_{pmn_{n-1}}(\zeta) = \frac{1 - \beta}{M(\beta)} \Theta_4(\zeta, I_{pmn_{n-1}}) - \Theta_4(\zeta, I_{pmn_{n-2}}) + \frac{\beta}{M(\beta)\Gamma(\beta)} \int_0^\zeta \Theta_4(\zeta, I_{pmn_{n-1}}) - \Theta_4(\zeta, I_{pmn_{n-2}})(\zeta - \zeta)^{\beta - 1} d\zeta, \\
\Xi_{5n}(\zeta) &= R_{p_n}(\zeta) - R_{p_{n-1}}(\zeta) = \frac{1 - \beta}{M(\beta)} \Theta_5(\zeta, R_{p_{n-1}}) - \Theta_5(\zeta, R_{p_{n-2}}) + \frac{\beta}{M(\beta)\Gamma(\beta)} \int_0^\zeta \Theta_5(\zeta, R_{p_{n-1}}) - \Theta_5(\zeta, R_{p_{n-2}})(\zeta - \zeta)^{\beta - 1} d\zeta, \\
\Xi_{6n}(\zeta) &= R_{m_n}(\zeta) - R_{m_{n-1}}(\zeta) = \frac{1 - \beta}{M(\beta)} \Theta_6(\zeta, R_{m_{n-1}}) - \Theta_6(\zeta, R_{m_{n-2}}) + \frac{\beta}{M(\beta)\Gamma(\beta)} \int_0^\zeta \Theta_6(\zeta, R_{m_{n-1}}) - \Theta_6(\zeta, R_{m_{n-2}})(\zeta - \zeta)^{\beta - 1} d\zeta, \\
\Xi_{7n}(\zeta) &= R_{pmn}(\zeta) - R_{pmn_{n-1}}(\zeta) = \frac{1 - \beta}{M(\beta)} \Theta_7(\zeta, R_{pmn_{n-1}}) - \Theta_7(\zeta, R_{pmn_{n-2}}) + \frac{\beta}{M(\beta)\Gamma(\beta)} \int_0^\zeta \Theta_7(\zeta, R_{pmn_{n-1}}) - \Theta_7(\zeta, R_{pmn_{n-2}})(\zeta - \zeta)^{\beta - 1} d\zeta.
\end{align*}
\]

Implementing the norm on the aforementioned system (13), we have
Repeating the same procedure, the subsequent terms of (14) can be simplified to the following:

\[ \left\| \Xi_{1n} (\zeta) \right\| \leq L_1 \left\| \Xi_{1(n-1)} (\zeta) \right\| \text{ with } \frac{1 - \beta}{M(\beta)} \leq L_1 \leq \frac{1 - \beta}{M(\beta)} + \frac{\zeta^\beta}{M(\beta)}. \quad (16) \]
\[ \| \Xi_{2n}(\zeta) \| \leq L_{2n} \| \Xi_{2(n-1)}(\zeta) \| \left( 1 - \frac{\beta}{M(\beta)} + \frac{\zeta^{\beta}}{M(\beta)} \right) \]

(17)

Theorem 2. The fractional \( P \) model presented in (7) has a solution if \( \mathcal{U}_0 \) holds the variant

\[ \left( 1 - \frac{\beta}{M(\beta)} + \frac{\mathcal{U}_0^{\beta}}{M(\beta)\Gamma(\beta)} \right) \leq 1, < 1, \ell = 1, 2, \ldots, 7. \]

(18)

Proof. By means of (16) and (19), we have

\[ \| \Xi_{1n}(\zeta) \| \leq \| S(0) \| \left( 1 - \frac{\beta}{M(\beta)} + \frac{\zeta^{\beta}}{M(\beta)} \right)^n, \]

\[ \| \Xi_{2n}(\zeta) \| \leq \| I_p(0) \| \left( 1 - \frac{\beta}{M(\beta)} + \frac{\zeta^{\beta}}{M(\beta)} \right)^n, \]

\[ \| \Xi_{3n}(\zeta) \| \leq \| I_m(0) \| \left( 1 - \frac{\beta}{M(\beta)} + \frac{\zeta^{\beta}}{M(\beta)} \right)^n, \]

\[ \| \Xi_{4n}(\zeta) \| \leq \| I_{pm}(0) \| \left( 1 - \frac{\beta}{M(\beta)} + \frac{\zeta^{\beta}}{M(\beta)} \right)^n, \]

\[ \| \Xi_{5n}(\zeta) \| \leq \| R_p(0) \| \left( 1 - \frac{\beta}{M(\beta)} + \frac{\zeta^{\beta}}{M(\beta)} \right)^n, \]

\[ \| \Xi_{6n}(\zeta) \| \leq \| R_m(0) \| \left( 1 - \frac{\beta}{M(\beta)} + \frac{\zeta^{\beta}}{M(\beta)} \right)^n. \]

(19)

Utilizing the fact of (20), we find

\[ \| B_{in}(\zeta) \| \leq \frac{1 - \frac{\beta}{M(\beta)}}{\Gamma(\beta)(\beta)M(\beta)} \| S(0) \| \leq \| S(0) \| \| S_{n-1} \| \| S_n \|. \]

(21)

Iteratively performing the technique yields

\[ \| B_{in}(\zeta) \| \leq \left( 1 - \frac{\beta}{M(\beta)} + \frac{\zeta^{\beta}}{M(\beta)\Gamma(\beta)} \right)^n \| S_n - S_{n-1} \|^n. \]

(22)

Theorem 3. Let there be a unique solution of the fractional \( P \) model (7) given that

\[ \left( 1 - \frac{\beta}{M(\beta)} + \frac{\zeta^{\beta}}{\Gamma(\beta)M(\beta)} \right) \leq 1, < 1, \ell = 1, 2, \ldots, 7. \]

(23)

Proof. Suppose that \( S_1, I_p, I_m, I_{pm}, R_p, R_m, \) and \( R_{pm} \) are another solution to fractional \( P \) model (7). Then,
Employing the norm to the aforesaid equation, we have
\[
\|S(\zeta) - S_1(\zeta)\| \leq \frac{1 - \beta}{\mathcal{M}(\beta)} \|S - S_1\|_1 + \frac{\zeta^\beta}{\mathcal{M}(\beta)\Gamma(\beta)} \int_0^\zeta \left(\Theta_1(\zeta, S) - \Theta_1(\zeta, S_1)\right) (\zeta - c)^{\beta - 1} dc.
\]
(26)

3. Qualitative Analysis

Several key aspects of the developed framework, including boundedness, the existence of equilibria, and fundamental reproductive quantity, will be outlined and discussed.

3.1. Positively Invariant and Boundedness. In order to find the invariant domain, we surmise the overall population \( \mathcal{N} = S + I_p + I_m + I_{pm} + R_p + R_m + R_{pm} \).

Let the domain of the fractional \( \mathcal{P} \) model (7) that is epidemiologically sustainable determined by

\[
\mathcal{O} := \left\{ (S, I_p, I_m, I_{pm}, R_p, R_m, R_{pm}) \in \mathbb{R}_+^7 : 0 \leq S + I_p + I_m + I_{pm} + R_p + R_m + R_{pm} \leq \mathcal{N} \right\}.
\]
(28)

In order to prove that the collection \( \mathcal{O} \) is positively invariant, apply Lemma 1, and we have

\[
\begin{align*}
\mathcal{O}^\beta S(\zeta)|_{S = 0} & = \pi \geq 0, \\
\mathcal{O}^\beta S(\zeta)|_{S = 0} & = \pi \geq 0, \\
\mathcal{O}^\beta S(\zeta)|_{S = 0} & = \pi \geq 0, \\
\mathcal{O}^\beta S(\zeta)|_{S = 0} & = \pi \geq 0, \\
\mathcal{O}^\beta S(\zeta)|_{S = 0} & = \pi \geq 0, \\
\mathcal{O}^\beta S(\zeta)|_{S = 0} & = \pi \geq 0, \\
\end{align*}
\]
(29)

It is clear from (29) that every outcome of (7) is positive and will be in \( \mathbb{R}_+^7 \). Therefore, the set \( \mathcal{O} \) presented in (28) is positively invariant for fractional \( \mathcal{P} \) model (7).

Additionally, to prove the boundedness of the solutions of the fractional \( \mathcal{P} \) framework, we proceed by aggregating all of the system’s components, offering

\[
\mathcal{L}_0^{ABC} \mathcal{O}^\beta \mathcal{N}(\zeta) + -\omega \mathcal{N} \leq \mathcal{L}_0^{ABC}(\pi) \mathcal{L}(\mathcal{N}) \left( (1 - \lambda)s_1^\beta - \frac{\lambda \beta}{1 - \beta} \right) - \omega^\beta \mathcal{N}(0) \leq \frac{1 - \beta}{\mathcal{M}(\beta)} \left( \omega^\beta + \frac{\beta}{1 - \beta} \right) \frac{\pi}{\omega}
\]
(31)

Using the Laplace transform, we have

\[
\left( 1 - \frac{\lambda \beta}{(1 - \lambda)(1 - \beta)\omega^\beta} \right) \left( 1 - \frac{\beta}{(1 - \lambda)\mathcal{M}(\beta)} \right) \left( 1 + \frac{\beta}{(1 - \beta)\omega^\beta} \right) \frac{\mathcal{N}(0)}{(1 - \lambda)\omega}.
\]
where $\lambda = (\beta - 1)q/\beta (\beta)$. In view of inverse Laplace transform, we have

$$\mathcal{N}(\zeta) = \pi \varrho (1-\lambda) d\zeta \int_0^\zeta E_\beta \left( \frac{\beta (\zeta - x_1)\beta}{(1-\lambda)(1-\beta)} \right) dx_1 + \frac{1}{1-\lambda} E_\beta \left( \frac{\beta \zeta\beta}{(1-\lambda)(1-\beta)} \right) \mathcal{N}(0),$$

where $E_{\beta,\gamma}$ presents the ML function. Based on the assumption that the ML function exhibits asymptotic nature,

$$E_{\beta,\gamma}(z) = \frac{\sum_{\gamma=1}^\infty \beta^{-\gamma} \eta}{\Gamma (\beta - \gamma z) + \sigma ([z] - 1)}, \frac{\eta}{\varrho} < \text{arg}(z) \leq \pi.$$

It is not really hard to perceive that $\mathcal{N}(\zeta) \rightarrow \pi/\varrho$, and this concludes that system (7) is biologically sustainable in the domain.

### 3.2. Disease-Free Equilibrium (DFE).

Set $I_p = 0, I_m = 0$, and $I_p = 0$ in (6); then, $DFE$ reduces to $E_0 = (\pi/\varrho, 0, 0, 0, 0, 0)$.

$$\mathcal{F} = \begin{bmatrix} q_1 \varrho & 0 & q_1 \varrho \\ \varrho & 0 & \varrho \\ q_2 \varrho & q_2 \varrho & 0 \\ 0 & \varrho & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

and $\mathcal{V} = \begin{bmatrix} \rho_1 + \eta_1 + \varrho & 0 & 0 \\ 0 & \rho_2 + \eta_2 + \varrho & 0 \\ 0 & 0 & \rho + \eta_1 + \eta_2 + \varrho \end{bmatrix}$.

$$\mathcal{F} \mathcal{V}^{-1} = \begin{bmatrix} q_1 \varrho \left( \rho_1 + \eta_1 + \varrho \right) & 0 & q_1 \varrho \left( \rho_1 + \eta_1 + \eta_2 + \varrho \right) \\ 0 & q_2 \varrho \left( \rho_2 + \eta_2 + \varrho \right) & q_2 \varrho \left( \rho + \eta_1 + \eta_2 + \varrho \right) \\ 0 & 0 & 0 \end{bmatrix}.$$

The eigenvalues of the system are $\lambda_1 = 0$, $\lambda_2 = q_1 \varrho (\rho + \eta + \varrho) = \mathcal{R}_{op}$, $\lambda_3 = q_2 \varrho (\rho_2 + \eta_2 + \varrho) = \mathcal{R}_{om}$. Clearly, we see that $\mathcal{R}_0 = \max\{\mathcal{R}_{op}, \mathcal{R}_{om}\}$ is the fundamental reproductive number.

### 3.3. Fundamental Reproductive Number.

Assume that the infected component of system (6) is

$$I_p(\zeta) = g_1S - (g_2 + \varrho + \rho_1 + \eta_1)I_p,$$

$$I_m(\zeta) = g_2S - (g_1 + \varrho + \rho_2 + \eta_2)I_m,$$

$$I_{pm}(\zeta) = g_1I_p + g_1I_m - (\varrho + \rho + \eta_1 + \eta_2)I_{pm}.$$

As in [42], it is constructed employing the next generation matrix. For this, we construct the matrix $\mathcal{F}$ and $\mathcal{V}$ as follows:

### 3.4. Local Stability of DFE

**Theorem 4.** Let there be a $DSE$ point which is locally asymptotically stable if $\mathcal{R}_0 < 1$. Also, if $\mathcal{R}_0 > 1$, then it is unstable.
Proof. For $DFE$, the Jacobian matrix of $P$ model (6) is presented as

$$J_{E_0} = \begin{bmatrix} R_{3\times3} & \partial_{3\times4} \\ \mathcal{R}_{4\times3} & \mathcal{S}_{4\times4} \end{bmatrix},$$

where

$$R_{3\times3} = \begin{bmatrix} -\varrho, & -\frac{q_1\pi}{\varrho}, & 0, \\ 0, & -\frac{q_1\pi}{\varrho - (\rho_1 + \eta_1 + \varrho)}, & 0, \\ 0, & 0, & -\frac{q_2\pi}{\varrho - (\rho_2 + \eta_2 + \varrho)} \end{bmatrix},$$

$$\partial_{3\times4} = \begin{bmatrix} \frac{-q_2\pi}{\varrho} & \phi_1 & \phi_2 & \phi_3 \\ -\frac{q_2\pi}{\varrho} & 0 & 0 & 0, \\ 0 & 0 & 0 & 0 \\ -(\rho + \eta_1 + \eta_2 + \varrho) & 0 & 0 & 0 \end{bmatrix},$$

$$\mathcal{R}_{4\times3} = \begin{bmatrix} 0 & 0 & 0 \\ 0 & \phi_1 & 0 \\ 0 & 0 & \phi_2 \\ 0 & 0 & 0 \end{bmatrix}, \mathcal{S}_{4\times4} = \begin{bmatrix} \rho e & -\varrho & 0 & 0 \\ \rho \eta_1 & 0 & 0 & -\varrho \\ \rho (1 - h_1)(1 - \epsilon) & 0 & 0 & -\varrho + \phi_3 \end{bmatrix}.$$

By the virtue of (36), one can acquire the subsequent characteristic polynomial as

$$D(\lambda) = -(\varrho + \lambda)(\phi_1 + \varrho + \lambda)(\phi_2 + \varrho + \lambda)(\rho + \eta_1 + \eta_2 + \varrho + \lambda)(\phi_3 + \varrho + \lambda)\left(\frac{-q_1\pi}{\varrho + \rho_1 + \eta_1 + \varrho + \lambda}\right) - \frac{-q_2\pi}{\varrho + \rho_2 + \eta_2 + \varrho + \lambda}.$$

After simplification, we get

$$\lambda_1 = -\lambda_2 = -(\phi_1 + \phi_3), \lambda_3 = -(\phi_2 + \phi_4), \lambda_4 = -(\rho + \eta_1 + \eta_2 + \phi_5), \lambda_5 = -\phi_6,$$

$$\lambda_6 = -(\rho + \eta_1 + \eta_2 + \varrho), \lambda_7 = -(\rho + \eta_1 + \eta_1 + \varrho).$$
Since $\lambda_0, \lambda_7 < 0$, that is, $\rho_1 + \eta_1 + \varrho < q_1 \pi / \varrho$ and $\rho_2 + \eta_2 + \varrho < q_2 \pi / \varrho$, this yields that $\Re_0 < 1$. Analogously, $\rho_2 + \eta_2 + \varrho < q_2 \pi / \varrho$ shows that $\Re_{wm} < 1$. This concludes that $\Re_0 = \max\{\Re_{up}, \Re_{wm}\} < 1$ which shows that $\mathcal{F}$ is locally asymptotically stable. \hfill $\square$

3.5. Global Stability of $\mathcal{F}$. Here, we employ the mechanism described in [22] to examine the global stability of $\mathcal{F}$. To begin, rewrite $\mathcal{P}$ model (6) as

$$
\begin{align*}
\dot{\chi}_1 (\zeta) &= \mathcal{Y}_1 (\chi_1, \chi_2), \\
\dot{\chi}_2 (\zeta) &= \mathcal{Y}_2 (\chi_1, \chi_2),
\end{align*}
$$

subject to $\mathcal{Y}_2 (\chi_1, 0) = 0$. It is worth mentioning that $\chi_1 = \{S, R_p, R_m, R_{pm}\}$ represents the uncontaminated population while $\chi_2 = \{I_p, I_m, I_{pm}\}$ indicate the contaminated population, respectively. Furthermore, the $\mathcal{F}$ of the $\mathcal{P}$ model is presented by $\mathcal{F} = (\Re, 0)$. This demonstrates that $\mathcal{F} = (\Re, 0)$ is globally asymptotically stable equilibrium for $\mathcal{P}$ framework given that $\Re < 1$ (by Theorem 1) and the subsequent assumptions hold:

(i) For $\mathcal{N}_1 (\zeta) = \mathcal{Y}_1 (\chi_1, 0)$, $\Re$ is globally asymptotically stable.

(ii) $\mathcal{Y}_2 (\chi_1, \chi_2) = \mathcal{A} \chi_2 - \mathcal{F}_2 (\chi_1, \chi_2), \mathcal{F}_2 (\chi_1, \chi_2) \geq 0$ for $(\chi_1, \chi_2) \in \mathcal{F}$.

The preceding hypothesis applies if $\mathcal{P}$ model (6) satisfies the aforementioned requirements.

Theorem 5. If (i) and (ii) hold, then $\mathcal{F} = (\Re, 0)$ is globally asymptotically stable equilibrium given that $\Re < 1$.

Proof. By means of $\mathcal{P}$ model, we can write

$$
\begin{align*}
\mathcal{Y}_1 (\chi_1, \chi_2) &= \begin{bmatrix}
\pi + \phi_1 R_p + \phi_2 R_m + \phi_3 R_{pm} - (g_1 + g_2 + \varrho) S \\
\rho_1 I_p + \varrho I_{pm} - (\phi_1 + \varrho) R_p \\
\rho_2 I_m + \varrho I_{pm} - (\phi_2 + \varrho) R_m \\
\rho (1 - h_1) (1 - \varpsilon) I_{pm} - (\phi_3 + \varrho) R_{pm}
\end{bmatrix} \mathcal{F},
\end{align*}
$$

and

$$
\begin{align*}
\mathcal{Y}_2 (\chi_1, \chi_2) &= \begin{bmatrix}
g_1 S - (g_2 + \varrho + \rho_1 + \eta_1) I_p \\
g_2 S - (g_1 + \varrho + \rho_2 + \eta_2) I_m \\
g_2 I_p + g_1 I_m - (\rho + \varrho + \eta_1 + \eta_2) I_{pm}
\end{bmatrix} \mathcal{F},
\end{align*}
$$

which illustrates that $\mathcal{F}_2 (\chi_1, \chi_2) < 0$, and this suggests that the secondary assumption (ii) is not provided, and $\mathcal{F} = (\Re, 0)$ may not be globally asymptotically stable when $\Re < 1$.

3.6. Sensitivity Analysis. Through sensitivity analysis, the most important components for the development and regulation of transmission in the population are identified. We employ the strategies provided in [43] to accomplish this. With consideration to a factor, assume $x_1$, the responsiveness factor of $\Re_0$, is determined by $\psi_{x_1} = \partial \Re_0 / \partial x_1$. As $\Re_0 = \max\{\Re_{up}, \Re_{wm}\}$, the sensitivity evaluation of $\Re_{up}$ and $\Re_{wm}$ is conducted individually as follows:
\[
\Psi_{q_1} = \frac{\partial \Psi_0}{\partial q_1} \frac{\partial q_1}{\partial \Psi_0} q_1 \eta_1 (\rho_1 + \beta_1 + \varrho) = 1 > 0,
\]
\[
\Psi_{\eta_1} = \frac{\partial \Psi_0}{\partial \eta_1} \frac{\partial \eta_1}{\partial \Psi_0} = \frac{-\eta_1}{(\rho_1 + \beta_1 + \varrho)} < 0,
\]
\[
\Psi_{\rho_1} = \frac{\partial \Psi_0}{\partial \rho_1} \frac{\partial \rho_1}{\partial \Psi_0} = \frac{-\rho_1}{(\rho_1 + \beta_1 + \varrho)} < 0,
\]
\[
\Psi_{\eta_2} = \frac{\partial \Psi_0}{\partial \eta_2} \frac{\partial \eta_2}{\partial \Psi_0} = \frac{-\eta_2}{(\rho_2 + \beta_2 + \varrho)} < 0,
\]
\[
\Psi_{\rho_2} = \frac{\partial \Psi_0}{\partial \rho_2} \frac{\partial \rho_2}{\partial \Psi_0} = \frac{-\rho_2}{(\rho_2 + \beta_2 + \varrho)} < 0,
\]
\[
\Psi_{\eta_2} = \frac{\partial \Psi_0}{\partial \eta_2} \frac{\partial \eta_2}{\partial \Psi_0} = \frac{-\eta_2}{(\rho_2 + \beta_2 + \varrho)} < 0.
\]

The analysis shows that characteristics with highly accurate index values, especially \(q_1\) and \(q_2\), have a huge promise for spreading bacterial viruses, meningitis, and their co-infections in the congregation even though they boost their corresponding propagation quantity, which seems to be the mean value of supplementary illnesses. If the estimated quantities containing a negative sensitivity value are improved while the quantities of the other characteristics remain stable, the factors having a negative sensitivity level contribute significantly to reducing the spread of \(P\) in the population.

### 3.7. Numerical Approaches of \(P\) Model

Here, we leverage the Toufik–Atangana [37] approach to generate a systematic formula for scheme (7) in this part.

In consideration of the first component of (7), we find

\[
\begin{aligned}
&\delta_{ABC} D^\alpha_\zeta \mathbf{S}(\zeta) = \nabla_1 (\zeta, \mathbf{S}(\zeta)), \\
&\mathbf{S}(0) = \mathbf{S}_0.
\end{aligned}
\]

Observing (9), one can calculate for (48) in the problem described in the following:

\[
\begin{aligned}
\mathbf{S}(\zeta) &= \mathbf{S}(0) + \frac{1 - \beta}{\ln(\beta)} \nabla_1 (\zeta, \mathbf{S}(\zeta)) + \frac{\beta}{\ln(\beta)} \nabla_1 (\zeta, \mathbf{S}(\zeta)) \mathbf{S}(\zeta), \\
&\int_0^\zeta \nabla_1 (\zeta, \mathbf{S}(\zeta)) (\zeta - c)^{\beta-1} dc.
\end{aligned}
\]

Utilizing Lagrange’s interpolating polynomial technique on \([\zeta_{p-1}, \zeta_p]\) to \(\mathbf{V}_1(\zeta, \mathbf{S}(\zeta)) = \pi/ (g_1 + g_2 + \varrho) - (\phi_1 p(y) + \phi_2 R_m(y) + \phi_3 R_pm(y)) / (g_1 + g_2 + \varrho) - \mathbf{S}(y)\), then we have

\[
\begin{aligned}
\mathbf{S}_p &= \frac{1}{h} \left[ (y - \zeta_{p-1}) \mathbf{V}_1(\zeta_{p-1}, \mathbf{S}(\zeta_{p-1}), \mathbf{I}_p(\zeta_{p-1}), \mathbf{I}_{pm}(\zeta_{p-1}), \mathbf{R}_p(\zeta_{p-1}), \mathbf{R}_m(\zeta_{p-1}), \mathbf{R}_{pm}(\zeta_{p-1})) \\
&- (y - \zeta_p) \mathbf{V}_1(\zeta_p, \mathbf{S}(\zeta_p), \mathbf{I}_p(\zeta_p), \mathbf{I}_{pm}(\zeta_p), \mathbf{R}_p(\zeta_p), \mathbf{R}_m(\zeta_p), \mathbf{R}_{pm}(\zeta_p)) \right],
\end{aligned}
\]

where \(h = \zeta_p - \zeta_{p-1}\). Inserting (49) into (50), we have
\[ S(\zeta_{n+1}) = S(0) + \frac{1 - \beta}{h} \mathcal{V}_1(\zeta_n, S(\zeta_n), I_p(\zeta_n), I_m(\zeta_n), I_{pm}(\zeta_n), R_p(\zeta_n), R_m(\zeta_n), R_{pm}(\zeta_n)) \]

\[
\begin{aligned}
&+ \frac{\beta}{h^{d}(\beta)} \sum_{k=1}^{n} \left[ \mathcal{V}_1(\zeta_k, S(\zeta_k), I_p(\zeta_k), I_m(\zeta_k), I_{pm}(\zeta_k), R_p(\zeta_k), R_m(\zeta_k), R_{pm}(\zeta_k)) \right] \\
&\quad \times \int_{\zeta_k}^{\zeta_{k+1}} (y - \zeta_{k-1})(\zeta_{n+1} - y)^{\beta-1} dy \right] \frac{S(\zeta_{k-1}), I_p(\zeta_{k-1}), I_m(\zeta_{k-1}), I_{pm}(\zeta_{k-1}), R_p(\zeta_{k-1}), R_m(\zeta_{k-1}), R_{pm}(\zeta_{k-1}))}{h} \\
&\quad \times \int_{\zeta_k}^{\zeta_{k+1}} (y - \zeta_{k-1})(\zeta_{n+1} - y)^{\beta-1} dy.
\end{aligned}
\]

(51)

where

\[
\mathcal{Z}_{k-1} = \int_{\zeta_k}^{\zeta_{k+1}} (y - \zeta_{k-1})(\zeta_{n+1} - y)^{\beta-1} dy
\]

\[
= \frac{1}{(\beta + 1)} \left[ (\zeta_{n+1} - \zeta_{k-1})^{\beta} - (\zeta_{n+1} - \zeta_{k})^{\beta} \right] - \frac{1}{\beta + 1} \left( (\zeta_{n+1} - \zeta_{k})^{\beta+1} - (\zeta_{n+1} - \zeta_{k})^{\beta} - (\zeta_{n+1} - \zeta_{k})^{\beta+1} \right),
\]

(52)

and

\[
\mathcal{Z}_k = \int_{\zeta_k}^{\zeta_{k+1}} (y - \zeta_{k-1})(\zeta_{n+1} - y)^{\beta-1} dy
\]

\[
= \frac{1}{\beta} \left[ (\zeta_{n+1} - \zeta_{k-1})^{\beta} - (\zeta_{n+1} - \zeta_{k})^{\beta} \right] \quad \text{as follows:}
\]

(53)

And

\[
\mathcal{Z}_{n+1} = \frac{h^{\beta+1}}{\beta(\beta + 1)} \left( (n + 1 - \kappa)\left( (n + \kappa + 2 + \beta) - (n - \kappa)\right) \right),
\]

(54)

and

\[
\mathcal{Z}_k = \frac{h^{\beta+1}}{\beta(\beta + 1)} \left( (n + 1 - \kappa)\left( (n + \kappa + 1 + \beta) \right) \right). \quad \text{(55)}
\]

Eventually, we can write (56) in the form of (54) and (55) as follows:
\[ S(\zeta_{n+1}) = S(\zeta_0) + \frac{1 - \beta}{M(\beta)} \nabla_1(\zeta_n, S(\zeta_n), I_p(\zeta_n), I_m(\zeta_n), I_{pm}(\zeta_n), R_p(\zeta_n), R_m(\zeta_n), R_{pm}(\zeta_n)), \]

\[ + \frac{\beta}{M(\beta)\Gamma(\beta)} \sum_{k=1}^{n} \left\{ \begin{array}{c} \frac{\nabla_1(\zeta_k, S(\zeta_k), I_p(\zeta_k), I_m(\zeta_k), I_{pm}(\zeta_k), R_p(\zeta_k), R_m(\zeta_k), R_{pm}(\zeta_k))}{\Gamma(\beta + 2)} \\
\times h^\beta \left\{ (n + 1 - \kappa)^\beta (n - \kappa + 2 + \beta) - (n - \kappa)^\beta (n - \kappa + 2 + 2\beta) \right\} \\
\frac{\nabla_1(\zeta_{k-1}, S(\zeta_{k-1}), I_p(\zeta_{k-1}), I_m(\zeta_{k-1}), I_{pm}(\zeta_{k-1}), R_p(\zeta_{k-1}), R_m(\zeta_{k-1}), R_{pm}(\zeta_{k-1}))}{h} \\
\times h^\beta \left\{ (n + 1 - \kappa)^\beta (n - \kappa + 2 + \beta) - (n - \kappa)^\beta (n - \kappa + 2 + 2\beta) \right\} \end{array} \right\} \right\} \] (56)

Additionally, the formulations for the remaining model factors are as follows:

\[ I_p(\zeta_{n+1}) = I_p(\zeta_n) + \frac{1 - \beta}{M(\beta)} \nabla_1(\zeta_n, S(\zeta_n), I_p(\zeta_n), I_m(\zeta_n), I_{pm}(\zeta_n), R_p(\zeta_n), R_m(\zeta_n), R_{pm}(\zeta_n)) \]

\[ + \frac{\beta}{M(\beta)\Gamma(\beta)} \sum_{k=1}^{n} \left\{ \begin{array}{c} \frac{\nabla_1(\zeta_k, S(\zeta_k), I_p(\zeta_k), I_m(\zeta_k), I_{pm}(\zeta_k), R_p(\zeta_k), R_m(\zeta_k), R_{pm}(\zeta_k))}{\Gamma(\beta + 2)} \\
\times h^\beta \left\{ (n + 1 - \kappa)^\beta (n - \kappa + 2 + \beta) - (n - \kappa)^\beta (n - \kappa + 2 + 2\beta) \right\} \\
\frac{\nabla_1(\zeta_{k-1}, S(\zeta_{k-1}), I_p(\zeta_{k-1}), I_m(\zeta_{k-1}), I_{pm}(\zeta_{k-1}), R_p(\zeta_{k-1}), R_m(\zeta_{k-1}), R_{pm}(\zeta_{k-1}))}{h} \\
\times h^\beta \left\{ (n + 1 - \kappa)^\beta (n - \kappa + 2 + \beta) - (n - \kappa)^\beta (n - \kappa + 2 + 2\beta) \right\} \end{array} \right\} \right\} \] (57)

\[ I_m(\zeta_{n+1}) = I_m(\zeta_n) + \frac{1 - \beta}{M(\beta)} \nabla_1(\zeta_n, S(\zeta_n), I_p(\zeta_n), I_m(\zeta_n), I_{pm}(\zeta_n), R_p(\zeta_n), R_m(\zeta_n), R_{pm}(\zeta_n)) \]

\[ + \frac{\beta}{M(\beta)\Gamma(\beta)} \sum_{k=1}^{n} \left\{ \begin{array}{c} \frac{\nabla_1(\zeta_k, S(\zeta_k), I_p(\zeta_k), I_m(\zeta_k), I_{pm}(\zeta_k), R_p(\zeta_k), R_m(\zeta_k), R_{pm}(\zeta_k))}{\Gamma(\beta + 2)} \\
\times h^\beta \left\{ (n + 1 - \kappa)^\beta (n - \kappa + 2 + \beta) - (n - \kappa)^\beta (n - \kappa + 2 + 2\beta) \right\} \\
\frac{\nabla_1(\zeta_{k-1}, S(\zeta_{k-1}), I_p(\zeta_{k-1}), I_m(\zeta_{k-1}), I_{pm}(\zeta_{k-1}), R_p(\zeta_{k-1}), R_m(\zeta_{k-1}), R_{pm}(\zeta_{k-1}))}{h} \\
\times h^\beta \left\{ (n + 1 - \kappa)^\beta (n - \kappa + 2 + \beta) - (n - \kappa)^\beta (n - \kappa + 2 + 2\beta) \right\} \end{array} \right\} \right\} \] (58)

\[ I_{pm}(\zeta_{n+1}) = I_{pm}(\zeta_n) + \frac{1 - \beta}{M(\beta)} \nabla_1(\zeta_n, S(\zeta_n), I_p(\zeta_n), I_m(\zeta_n), I_{pm}(\zeta_n), R_p(\zeta_n), R_m(\zeta_n), R_{pm}(\zeta_n)) \]

\[ + \frac{\beta}{M(\beta)\Gamma(\beta)} \sum_{k=1}^{n} \left\{ \begin{array}{c} \frac{\nabla_1(\zeta_k, S(\zeta_k), I_p(\zeta_k), I_m(\zeta_k), I_{pm}(\zeta_k), R_p(\zeta_k), R_m(\zeta_k), R_{pm}(\zeta_k))}{\Gamma(\beta + 2)} \\
\times h^\beta \left\{ (n + 1 - \kappa)^\beta (n - \kappa + 2 + \beta) - (n - \kappa)^\beta (n - \kappa + 2 + 2\beta) \right\} \\
\frac{\nabla_1(\zeta_{k-1}, S(\zeta_{k-1}), I_p(\zeta_{k-1}), I_m(\zeta_{k-1}), I_{pm}(\zeta_{k-1}), R_p(\zeta_{k-1}), R_m(\zeta_{k-1}), R_{pm}(\zeta_{k-1}))}{h} \\
\times h^\beta \left\{ (n + 1 - \kappa)^\beta (n - \kappa + 2 + \beta) - (n - \kappa)^\beta (n - \kappa + 2 + 2\beta) \right\} \end{array} \right\} \right\} \] (59)
\[ R_p(\zeta_{n+1}) = R_p(\zeta_0) + \frac{1 - \beta}{\eta(\beta)} \sum_{n=1}^{\infty} \left\{ \begin{array}{l} \frac{V_0(\zeta_n, S(\zeta_n), I_p(\zeta_n), I_m(\zeta_n), I_{pm}(\zeta_n), R_p(\zeta_n), R_m(\zeta_n), R_{pm}(\zeta_n))}{\Gamma(\beta + 2)} \\
\times h^\beta \left\{ (n + 1 - \kappa)^\beta (n + \kappa + 2 + \beta) - (n + \kappa - 2 + 2\beta) \right\} \\
\frac{V_0(\zeta_{n-1}, S(\zeta_{n-1}), I_p(\zeta_{n-1}), I_m(\zeta_{n-1}), I_{pm}(\zeta_{n-1}), R_p(\zeta_{n-1}), R_m(\zeta_{n-1}), R_{pm}(\zeta_{n-1}))}{h} \\
\times h^\beta \left\{ (n + 1 - \kappa)^\beta (n - \kappa + 2 + \beta) - (n - \kappa - 2 + 2\beta) \right\} \\
\end{array} \right\} \\
\]}

\[ R_m(\zeta_{n+1}) = R_m(\zeta_0) + \frac{1 - \beta}{\eta(\beta)} \sum_{n=1}^{\infty} \left\{ \begin{array}{l} \frac{V_0(\zeta_n, S(\zeta_n), I_p(\zeta_n), I_m(\zeta_n), I_{pm}(\zeta_n), R_p(\zeta_n), R_m(\zeta_n), R_{pm}(\zeta_n))}{\Gamma(\beta + 2)} \\
\times h^\beta \left\{ (n + 1 - \kappa)^\beta (n - \kappa + 2 + \beta) - (n - \kappa - 2 + 2\beta) \right\} \\
\frac{V_0(\zeta_{n-1}, S(\zeta_{n-1}), I_p(\zeta_{n-1}), I_m(\zeta_{n-1}), I_{pm}(\zeta_{n-1}), R_p(\zeta_{n-1}), R_m(\zeta_{n-1}), R_{pm}(\zeta_{n-1}))}{h} \\
\times h^\beta \left\{ (n + 1 - \kappa)^\beta (n - \kappa + 2 + \beta) - (n - \kappa - 2 + 2\beta) \right\} \\
\end{array} \right\} \\
\]}

\[ R_{pm}(\zeta_{n+1}) = R_{pm}(\zeta_0) + \frac{1 - \beta}{\eta(\beta)} \sum_{n=1}^{\infty} \left\{ \begin{array}{l} \frac{V_0(\zeta_n, S(\zeta_n), I_p(\zeta_n), I_m(\zeta_n), I_{pm}(\zeta_n), R_p(\zeta_n), R_m(\zeta_n), R_{pm}(\zeta_n))}{\Gamma(\beta + 2)} \\
\times h^\beta \left\{ (n + 1 - \kappa)^\beta (n + \kappa + 2 + \beta) - (n + \kappa - 2 + 2\beta) \right\} \\
\frac{V_0(\zeta_{n-1}, S(\zeta_{n-1}), I_p(\zeta_{n-1}), I_m(\zeta_{n-1}), I_{pm}(\zeta_{n-1}), R_p(\zeta_{n-1}), R_m(\zeta_{n-1}), R_{pm}(\zeta_{n-1}))}{h} \\
\times h^\beta \left\{ (n + 1 - \kappa)^\beta (n - \kappa + 2 + \beta) - (n - \kappa - 2 + 2\beta) \right\} \\
\end{array} \right\} \\
\]}

3.8. Results and Discussion. In this section, simulation studies for the resulting structure (P co-infection model) are carried out, taking into consideration the ABC derivative fractional operator having ML kernel. We employed MATLAB 2022 to assess the influence of several factors in the proliferation as well as to prevent P co-infection. For modelling purposes, the model parameters in Table 1 are considered.

In Figure 4, by maintaining the interaction rate stable, \( q_1 = 0.9 \), we evaluated the influence of \( \rho_1 \) in reducing the amount of pneumonia exclusively infected people. Figures 5 and 6 show that as the quantity of \( \rho_1 \) increases, the proportion of pneumonia exclusively susceptible people decreases. Figure 5, 6, and 7 indicate that the proportion of instances in categories \( I_p, I_m, I_{pm}, R_p, R_m, R_{pm} \) decreased dramatically when contrasted to Figures 4–7, which were replicated lacking the control approach. For various orders of the fractional derivative \( \beta \), the trajectories have varying asymptotic behaviour. As a result, authorities and regulators should focus on maximizing the levels of the survival rate, either by addressing sick populations or by increasing specific susceptibility to the pneumonia virus.

In Figure 8, we can observe that \( \rho_2 \) is essential to minimize the meningitis development. The proportion of
Figure 4: Two-dimensional illustration of the susceptible class $S$ and pneumonia infectious $I_p$ when $q_1 = 0.9$ with multiple fractional orders $\beta \in [0, 1]$.

Figure 5: Two-dimensional illustration of the meningitis infectious $I_m$ and PM co-infection $I_{pm}$ when $q_1 = 0.9$ with multiple fractional orders $\beta \in [0, 1]$. 
contagious community owing to meningitis decreases as the level of $\rho_2$ increases from 0.1 to 0.9, but the interaction rate stays unchanged at $q_2$.

Figures 9, 10, and 11 indicate that the proportion of instances in categories $I_p$, $I_m$, $I_{pm}$, $R_p$, $R_m$, $R_{pm}$ decreased dramatically when contrasted to Figures 8–11, which were replicated inducing the control approach. Therefore, the outbreak spreads gradually as the fractional order diminishes from 1, and the majority of
patients at the apex drops significantly (Figures 8–11). Consequently, normal individuals or the administration must pay special consideration to healing the afflicted individuals in their locality when combating the meningitis infection.

In Figure 12, the meningitis connection incidence $q_2$ and the success percentage of the co-infectious community $\rho$ are both assumed unchanged. Figure 13 demonstrates that as the interaction frequency of pneumonia improves, the co-infectious numbers boost, implying that the proliferation of
pneumonia and meningitis co-infection will expand as well. According to Figure 14, it is critical to reduce the incidence and prevalence of pneumonia in order to prevent co-infection. As a result, organizations should aim to minimize the interaction risk of pneumonia by quarantining sick individuals or implementing an effective mitigation technique to limit the spread of co-infection in the population. The influence of the survival incidence of the co-infectious community was investigated. According to the scenario characterization in Section 2, co-infectious

\[
\begin{align*}
\beta = 0.75 & \\
\beta = 0.8 & \\
\beta = 0.85 & \\
\beta = 0.9 & \\
\beta = 0.95 & \\
\beta = 1 & 
\end{align*}
\]

Figure 10: Two-dimensional illustration of the pneumonia recovered \( R_p \) and meningitis recovered \( R_m \) when \( q_2 = 0.06, \rho_2 \in [0.1, 0.9] \) with multiple fractional orders \( \beta \in [0, 1] \).

\[
\begin{align*}
\beta = 0.75 & \\
\beta = 0.8 & \\
\beta = 0.85 & \\
\beta = 0.9 & \\
\beta = 0.95 & \\
\beta = 1 & 
\end{align*}
\]

Figure 11: Two-dimensional illustration of the \( R_{pm} \) co-infectious recovered \( R_{pm} \) when \( q_2 = 0.06, \rho_2 \in [0.1, 0.9] \) with multiple fractional orders \( \beta \in [0, 1] \).
populations generally heal from pneumonia solely or resume the corresponding healed section, alleviating the symptoms or additional processes. As a result, Figure 15 indicates that boosting the co-infectious majority’s survival intensity has a significant impact on eliminating both infections in the region.

Figure 12: Two-dimensional illustration of the susceptible class $S$ and pneumonia infectious $I_p$ with varying contact rate $q_2$ and recovery rate co-infection $\rho$ for multiple fractional orders $\beta \in [0, 1]$.

Figure 13: Two-dimensional illustration of the meningitis infection $I_m$ and PM infectious $I_{pm}$ with varying contact rate $q_2$ and recovery rate co-infection $\rho$ for multiple fractional orders $\beta \in [0, 1]$.
4. Conclusion

This research examines seven-dimensional pneumonia and meningitis. The Atangana–Baleanu fractional derivative is applied to describe the integer-order framework, and the Banach contraction hypothesis is employed to assess the presence of systems in the fractional formulation of the numerical method. The Atangana–Baleanu fractional operator featuring generational characteristics is responsible for this beneficial outcome. The findings of this article argue that formal frameworks utilizing the Atangana–Baleanu fractional operator can effectively disclose the underlying or realistic features of real-world situations. This hypothesis can be supported by continuing research into the effects of various fractional operators, including such fractal-fractional derivatives, and reporting the performance of the Atangana–Baleanu fractional operator outcome on the relatively similar system or additional relevant epidemiological concepts. The paucity of a comparison of the present findings for the SEAIR paradigm employing the Atangana–Baleanu fractional operator versus findings acquired for the equivalent system assuming alternative fractional operators could potentially represent a deficit for ongoing studies.

Data Availability

No data were used to support this study.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Authors’ Contributions

S. Rashid provided the main ideas of the article, constructed the main results, and submitted the article. B. Kanwal drafted the manuscript and provided the qualitative analysis. A. G. Ahmad presented the existence and uniqueness analysis with their illustration. E. Bonyah provided the solution of example and completed the final revision. S. K. Elagan presented the qualitative analysis of the proposed model. All authors read and approved the final manuscript.
References

[1] M. Caputo and M. Fabrizio, “A new definition of fractional derivative without singular kernel,” *Prog. Fract. Differ. Appl.* vol. 73, no. 13, 2015.

[2] A. Atangana and D. Baleanu, “New fractional derivatives with nonlocal and non-singular kernel: theory and application to heat transfer model,” *Thermal Science*, vol. 20, no. 2, pp. 763–769, 2016.

[3] R. Scherer, S. L. Kalla, Y. Tang, and J. Huang, “The Grünwald-Letnikov method for fractional differential equations,” *Computers & Mathematics with Applications*, vol. 62, no. 3, pp. 902–917, 2011.

[4] C. Li, d. Qian, and Y. Q. Chen, “On riemann-liouville and Caputo derivatives,” *Discrete Dynamics in Nature and Society*, vol. 2011, Article ID 562494, 15 pages, 2011.

[5] T. Li and Y. Wang, “Stability of a class of fractional-order nonlinear systems,” *Discrete Dynamics in Nature and Society*, vol. 2014, Article ID 724270, 14 pages, 2014.

[6] H. Durur, A. Yokus, and K. A. Abro, “A non-linear analysis and fractionalized dynamics of Langmuir waves and ion sound as an application to acoustic waves,” *International Journal of Modelling and Simulation*, pp. 1–7, 2020.

[7] A. Yokus, H. Durur, D. Kaya, H. Ahmad, and T. A. Nofoal, “Numerical comparison of Caputo and Conformable derivatives of time fractional Burgers-Fisher equation,” *Results in Physics*, vol. 25, Article ID 104247, 2021.

[8] H. Durur, O. Tasbozan, and A. Kurt, “New analytical solutions of conformable time fractional bad and good modified Boussinesq equations,” *Applied Mathematics and Nonlinear Sciences*, vol. 5, no. 1, pp. 447–454, 2020.

[9] H. Khan, J. F. Gómez-Aguilar, A. Alkhazzan, and A. Khan, “A fractional order HIV-TB coinfection model with nonsingular Mittag-Leffler Law,” *Mathematical Methods in the Applied Sciences*, vol. 43, no. 6, pp. 3786–3806, 2020.

[10] C. T. Deressa and G. F. Duressa, “Analysis of Atangana-Baleanu fractional-order SEAIR epidemic model with optimal control,” *Advances in Difference Equations*, vol. 2021, no. 1, 2021.

[11] A. Atangana and J. F. Gómez-Aguilar, “Hyperchaotic behaviour obtained via a nonlocal operator with exponential decay and Mittag-Leffler laws,” *Chaos, Solitons & Fractals*, vol. 102, pp. 285–294, 2017.

[12] A. Atangana and K. M. Owolabi, “New numerical approach for fractional differential equations,” *Mathematical Modelling of Natural Phenomena*, vol. 13, no. 1, p. 3, 2018.

[13] T. Zdzisław, “Matlab solutions of chaotic fractional order circuits,” *Matlab Solutions of Chaotic Fractional Order Circuits. Engineering Education and Research Using MATLAB. Engineering Education and Research Using MATLAB*, Intech Open, 2011.

[14] A. McLuckie, *Respiratory Disease and its Management*, p. 51, Springer, Berlin, 2009.

[15] World Health Organization, *Programme of acute respiratory infections*, Technical Base for the WHO Recommendations on the Management of Pneumonia in Children at First-Level Health Facilities, World Health Organization, Geneva, Switzerland, 1991, https://apps.who.int/iris/handle/10665/61199.

[16] M. J. F. Martínez, E. G. Merino, E. G. Sánchez, J. E. G. Sánchez, A. M. d. Rey, and G. R. Sánchez, “A mathematical model to study the meningococcal meningitis,” *Procedia Computer Science*, vol. 18, pp. 2492–2495, 2013.

[17] E. Joseph, *Mathematical Analysis of Prevention and Control Strategies of Pneumonia in- Adults and Children*, Unpublished MSc Dissertation. University of Dar-es-Salaam, Tanzania, 2012.

[18] G. T. Tilahun, O. D. Makinde, and D. Malonza, “Modelling and optimal control of typhoid fever disease with cost-effective strategies,” *Computational and Mathematical Methods in Medicine*, vol. 2017, Article ID 2324518, 16 pages, 2017.

[19] D. Pessa, *Modelling the Dynamics of streptococcus Pneumonia Transmission in Children*, Master’s thesis, University of De Lisboa, Lisbon, Portugal, 2010.

[20] G. T. Tilahun, O. D. Makinde, and D. Malonza, “Modelling and optimal control of pneumonia disease with cost-effective strategies,” *Journal of Biological Dynamics*, vol. 11, Article ID 28613986, 2017.

[21] J. K. K. Asamoah, F. Nyabadza, B. Seidu, M. Chand, H. Dutta, and H. Dutta, “Mathematical modelling of bacterial meningitis transmission dynamics with control measures,” *Computational and Mathematical Methods in Medicine*, vol. 2018, Article ID 2657461, 21 pages, 2018.

[22] G. T. Tilahun, O. D. Makinde, and D. Malonza, “Co-dynamics of pneumonia and typhoid fever diseases with cost effective optimal control analysis,” *Applied Mathematics and Computation*, vol. 316, pp. 438–459, 2018.

[23] D. O. Onyinge, N. O. Ongati, and F. Ondundo, “Mathematical model for co-infection of pneumonia and HIV/AIDS with treatment,” *Int. J. Sci. Eng. Appl. Sci.*, vol. 2, 2016.

[24] O. C. Akinyi, J. Y. Mugisha, A. Manyonge, and C. Ouma, “Modelling the impact of misdiagnosis and treatment on the dynamics of malaria concurrent and co-infection with pneumonia,” *Applied Mathematical Sciences*, 2013.

[25] S. Rashid, E. I. Abouelmagd, A. Khalid, F. B. Farooq, and Y.-M. Chu, “Some recent developments on dynamical h-discrete fractional type inequalities in the frame of non-singular and nonlocal kernels,” *Fractals*, vol. 30, Article ID 224010, 2022.

[26] S. Rashid, S. Sultana, Y. Karaca, A. Khalid, and Y.-M. Chu, “Some further extensions considering discrete proportional fractional operators,” *Fractals*, vol. 30, Article ID 2240026, 2022.

[27] K. Karthikeyan, P. Karthikeyan, H. M. Baskonus, K. Venkatachalam, and Y.-M. Chu, “Almost sectorial operators on Y-Hilfer derivative fractional impulsive integro-differential equations ψ-Hilfer derivative fractional impulsive integro-differential equations,” *Mathematical Methods in the Applied Sciences*, 2021.

[28] S. N. Hajiseyedazizi, M. E. Samei, J. Alzabut, and Y.-M. Chu, “On multi-step methods for singular fractional q-integro-differential equations,” *Open Mathematics*, vol. 19, no. 1, pp. 1378–1405, 2021.

[29] F. Jin, Z.-S. Qian, Z.-S. Qian, Y.-M. Chu, and M. u. Rahman, “On nonlinear evolution model for drinking behavior under Caputo-Fabrizio derivative,” *Journal of Applied Analysis & Computation*, vol. 12, no. 2, pp. 790–806, 2022.

[30] A. Atangana, “Extension of rate of change concept: from local to nonlocal operators with applications,” *Results in Physics*, vol. 19, Article ID 103515, 2020.

[31] D. Baleanu, A. Jajarmi, S. S. Sajjadi, and D. Mozyrska, “A new fractional model and optimal control of a tumor-immune surveillance with non-singular derivative operator,” *Chaos: An Interdisciplinary Journal of Nonlinear Science*, vol. 29, no. 8, Article ID 083127, 2019.

[32] N. Sene, “Fractional diffusion equation with new fractional operator,” *Alexandria Engineering Journal*, vol. 59, no. 5, pp. 2921–2926, 2020.
[33] T.-H. Zhao, O. Castillo, H. Jahanshahi et al., "A fuzzy-based strategy to suppress the novel coronavirus (2019-NCOV) massive outbreak," *Applied and Computational Mathematics*, vol. 20, pp. 160–176, 2021.

[34] K. M. Owolabi, "Analysis and numerical simulation of multicomponent system with Atangana-Baleanu fractional derivative," *Chaos, Solitons & Fractals*, vol. 115, pp. 127–134, 2018.

[35] E. Demirci, A. Unal, and N. Ozalp, "A fractional order SEIR model with density dependent death rate," *Hacet. J. Math. Stat.* vol. 40, pp. 287–295, 2011.

[36] S. Uçar, "Analysis of a basic SEIRA model with Atangana-Baleanu derivative," *AIMS Mathematics*, vol. 5, no. 2, pp. 1411–1424, 2020.

[37] M. Toufik and A. Atangana, "New numerical approximation of fractional derivative with non-local and non-singular kernel: application to chaotic models," *European Physical Journal A: Hadrons and Nuclei*, vol. 132, p. 144, 2017.

[38] T. Abdeljawad and D. Baleanu, "Integration by parts and its applications of a new nonlocal fractional derivative with Mittag-Leffler nonsingular kernel," *The Journal of Nonlinear Science and Applications*, vol. 10, no. 03, pp. 1098–1107, 2017.

[39] J. Singh, D. Kumar, and D. Baleanu, "On the analysis of fractional diabetes model with exponential law," *Advances in Difference Equations*, vol. 2018, no. 1, p. 2018, 2018.

[40] Z. M. Odibat and N. T. Shawagfeh, "Generalized Taylor’s formula," *Applied Mathematics and Computation*, vol. 186, no. 1, pp. 286–293, 2007.

[41] S. K. Panda, "Applying fixed point methods and fractional operators in the modelling of novel coronavirus 2019-nCoV/SARS-CoV-2," *Results in Physics*, vol. 19, Article ID 103433, 2020.

[42] S. M. Lenhart and J. T. Workman, *Optimal Control Applied to Biological Models*, CRC Press, Boca Raton, 2007.

[43] J. P. LaSalle, *The Stability of Dynamical Systems*, SIAM, Philadelphia, 1976.