Mathematical Methods in the Applied Sciences

WILEY

RESEARCH ARTICLE

A Fractional Order Model for the Transmission Dynamics of Meningococcal Meningitis With Real Statistical Data

Festus Abiodun Oguntolu[1] | Olumuyiwa James Peter[2,3,4] | Benjamin Idoko Omede[5] | Ghaniyyat Bolanle Balogun[6] | Zainab Olabisi Dere[7] | Sania Qureshi[8,9,10]

[1]Department of Mathematics, Federal University of Technology, Minna, Nigeria | [2]Department of Mathematics, Saveetha School of Engineering, SIMATS, Saveetha University, Chennai, India | [3]Department of Mathematical and Computer Sciences, University of Medical Sciences, Ondo, Nigeria | [4]Department of Epidemiology and Biostatistics, School of Public Health, University of Medical Sciences, Ondo, Nigeria | [5]Department of Mathematical Sciences, Prince Abubakar Audu University (formerly Kogi State University), Anyigba, Nigeria | [6]Department of Computer Science, University of Ilorin, Ilorin, Nigeria | [7]Department of Mathematics, Florida State University, Tallahassee, Florida, USA | [8]Department of Basic Sciences and Related Studies, Mehran University of Engineering & Technology, Jamshoro, Pakistan | [9]Department of Mathematics, Near East University, Mathematics Research Center, Nicosia, Turkey | [10]Research Center of Applied Mathematics, Khazar University, Baku, Azerbaijan

Correspondence: Olumuyiwa James Peter (peterjames4real@gmail.com)

Received: 12 June 2025 | **Revised:** 12 January 2026 | **Accepted:** 14 January 2026

Keywords: basic reproduction number | fractional order | meningococcal meningitis | numerical simulations | stability

ABSTRACT

In this paper, we propose a Caputo-based fractional-order derivative model for the transmission dynamics of meningococcal meningitis (MM), incorporating the environmental concentration of Neisseria meningitidis as well as factors such as vaccination and the hygiene consciousness of susceptible individuals. The existence and uniqueness of solutions to the model are established using Banach's and Schauder's fixed-point theorems. Additionally, we compute the basic reproduction number and examine the local asymptotic stability of the disease-free equilibrium using the Routh–Hurwitz criterion. We analyze the stability of the fractional-order meningitis model using the Ulam–Hyers–Rassias stability method. Furthermore, we fit the model to the cumulative confirmed cases of cerebrospinal meningitis in Nigeria using data obtained from the Nigeria Centre for Disease Control (NCDC) to validate the model. The model demonstrates a good fit with the reported cumulative cases. Numerical simulations are conducted for various values of the fractional order. The results reveal an inverse relationship between the fractional order and the total number of asymptomatic infected individuals (carriers), symptomatic infected individuals, and the environmental concentration of Neisseria meningitidis. This implies that increasing the order of the fractional derivative leads to a decrease in the number of infections and bacterial concentration. Moreover, increasing vaccine uptake and improving hygiene consciousness among susceptible individuals significantly reduce both the number of infections and the environmental concentration of Neisseria meningitidis.

1 | Introduction

Meningitis is a medical condition characterized by the inflammation of the meninges, the protective membranes that encase the brain and spinal cord. These membranes, along with the cerebrospinal fluid, serve to safeguard the central nervous system and supply it with essential nutrients [1]. Meningitis is a medical emergency that can result in severe neurological complications and is typically life-threatening if not promptly treated [2–4]. Meningitis ranks among the four leading global causes of neurological disability-adjusted life years (DALYs) [5]. Meningitis encompasses various types, including bacterial, viral, fungal, parasitic, noninfectious, and aseptic forms [4]. Bacterial meningitis is a significant global public health concern, affecting approximately 1.7 million individuals annually and resulting in an estimated 170,000 deaths worldwide. It is considered one of the most severe forms of meningitis, primarily targeting the central nervous system. The four major pathogens responsible for acute bacterial meningitis are *Neisseria meningitidis*, *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Streptococcus agalactiae* [6]. Meningococcal meningitis (MM) is an acute, respiratory-transmitted infectious disease caused by *Neisseria meningitidis* (*N. meningitidis*), characterized by a high case fatality rate and severe long-term complications among survivors [7, 8].

Neisseria meningitidis (*N. meningitidis*) is a Gram-negative β-proteobacterium belonging to the family *Neisseriaceae*. It is classified into 13 serogroups based on variations in capsular polysaccharide structures; however, only six serogroups-A, B, C, W-135, X, and Y-are responsible for the majority of life-threatening infections [9]. Its pathogenicity depends on several virulence factors, among which the polysaccharide capsule is particularly critical, as it inhibits phagocytosis and facilitates evasion of the host immune response [10, 11]. It is a rapidly progressing infection that necessitates prompt diagnosis and timely, effective antibiotic treatment [11, 12]. However, even with appropriate care, up to 15% of cases remain fatal [11]. *N. meningitidis* is a leading cause of recurrent outbreaks of MM in the African "meningitis belt," a region in Sub-Saharan Africa that extends from Ethiopia to Senegal [13]. Epidemics typically begin at the onset of the dry season and subside rapidly with the arrival of the rains, only to recur when the subsequent dry season commences. While the exact cause of this pattern is not fully understood, environmental factors-such as absolute humidity, dust concentrations, and an increased incidence of respiratory tract infections due to cold nights-have been identified as significant contributors [14]. *Neisseria meningitidis* is known to cause severe clinical manifestations in the form of invasive meningococcal disease (IMD). IMD arises from the ability of *N. meningitidis* to colonize the mucosal surfaces of the nasopharynx and invade sterile tissues, including the meninges, bloodstream, and vital organs. Clinically, IMD often presents as either MM or septicemia, both of which progress rapidly, leading to death in 10%–15% of cases and permanent disabilities in 20% of survivors [15–17].

Meningococcal disease rates are highest in young children due to the decline of protective maternal antibodies and subsequently increase again in adolescents and young adults [14]. Certain lifestyle factors prevalent among adolescents and young adults heighten their risk of meningococcal disease. Everyday activities, such as sharing food, drinks, or utensils, or engaging in behaviors that involve the exchange of respiratory secretions, throat fluids, or saliva, can facilitate the transmission of the disease. Additionally, other lifestyle factors, such as overcrowded living conditions in college dormitories, may further contribute to the increased risk [14]. The symptoms of MM include headache, fever, vomiting, photophobia, neck stiffness, and lethargy. Meningococcal septicemia is characterized by fever, rash, vomiting, headache, flu-like symptoms, and abdominal pain [14]. The typical incubation period for meningococcal disease is 3–4 days (range: 1–10 days), during which the disease is most transmissible. *Neisseria meningitidis* can be detected in the nose and throat for 2–4 days and may persist for up to 24 h after the initiation of antibiotic treatment [18, 19]. The reservoir of infection can include a human case, a carrier, an incubatory contact, a convalescent carrier, or a throat carrier [19, 20]. The carrier is the primary source of infection [19]. Effective antibiotics promptly inhibit the proliferation of *N. meningitidis* [14]. All *Neisseria meningitidis* bacteria in the cerebrospinal fluid are eradicated three to four hours after the initiation of intravenous treatment with an appropriate antibiotic at the correct dosage [14], and the concentration of meningococcal endotoxin in plasma decreases by 50% within 2 h [14]. In Africa, the general treatment recommendations during endemic periods include ceftriaxone (2 g IV once daily for 5 days) in multiple doses to ensure effective treatment. In industrialized countries, the recommended treatment for adults with suspected acute bacterial meningitis typically involves an extended-spectrum third-generation cephalosporin. For example, in Sweden, the treatment regimen may include cefotaxime (3 g every 4 h, IV) or ceftriaxone (2 g twice daily or 4 g once daily, IV), often in combination with ampicillin (3 g every 4 h, IV), or alternatively, monotherapy with meropenem (2 g every 8 h, IV) [14]. In addition to antibiotic prophylaxis for secondary prevention in close contacts, vaccination may also play a crucial role in controlling meningococcal outbreaks [21]. Effective vaccines are available for serogroups A, C, W, and Y, and vaccines for serogroup B (MnB) have recently been approved [21].

Mathematical models have played a crucial role in analyzing the transmission dynamics of various infectious diseases, including malaria, HIV, tuberculosis, and COVID-19. The mathematical modeling of infectious diseases has gained growing importance over time, owing to its capacity to enhance understanding, forecasting, and management of disease transmission. Numerous studies employing integer-order mathematical models have been conducted to investigate the dynamics of MM, bacterial meningitis, viral meningitis, and meningitis more broadly (see [22–28]). Musa et al. [29] developed and analyzed an epidemic model to examine the transmission dynamics of MM, incorporating high- and low-risk susceptible populations. Their model differentiated between these groups based on access to medical resources, which influence the level of infection risk. The findings of their study underscore the importance of ensuring adequate medical resources within communities, particularly in regions endemic to MM, as a key strategy for reducing both incidence and mortality rates. Blyuss [30] introduced deterministic mathematical models to investigate the key factors influencing the dynamics of MM. The study highlighted the significant role of temporary immunity within the population, emphasizing that this factor should be carefully considered in disease surveillance and in evaluating the effectiveness of vaccination strategies. Crankson et al. [31] developed a novel two-strain deterministic mathematical model to study the transmission dynamics of bacterial meningitis, incorporating vaccination as a control measure. Their findings revealed that the spread of bacterial meningitis can be effectively prevented if at least 25% of the population possesses immunity to the disease. Asamoah et al. [32] proposed an optimal control model for bacterial meningitis that incorporates both vaccination and treatment influenced by public health education. Numerical simulations from their study indicated that the most effective strategy for controlling bacterial meningitis involves a combined approach, integrating vaccination with additional interventions such as treatment and public health education. Elmojtaba and Adam [33] formulated an SVCIRS compartmental deterministic model to investigate the impact of vaccination on the transmission of meningitis within a small human population. Their model differentiated between individuals who recovered with disabilities and those who recovered without disabilities. The findings from their study suggested that effective control of the disease can be achieved if the vaccine uptake rate is sufficiently high. Amar et al. [34] developed and conducted a qualitative analysis of a deterministic mathematical model to explore the transmission dynamics of meningitis, incorporating vaccination and screening as intervention strategies. Their findings concluded that enhancing both vaccination and screening efforts significantly reduces the transmission and overall impact of meningitis within the population. Yano and Bitok [35] formulated and analyzed a two-strain mathematical model to study the transmission dynamics of bacterial meningitis, incorporating key interventions such as treatment and vaccination while accounting for the presence of antibiotic resistance. Their model provides insights into how these interventions influence disease dynamics in the context of emerging resistance. Kotola and Mekonnen [36] developed a mathematical model to examine the co-dynamics of meningitis and pneumonia infections, incorporating interventions. Their findings highlighted that vaccination against both meningitis and pneumonia, reducing contact with infected individuals, and providing treatment are all critical factors in significantly reducing the impact of these diseases within communities. Recently, numerous researchers have studied the use of fractional-order and optimal control models to better capture disease dynamics and memory effects. These approaches can give more realistic simulations and successful intervention options [37–39].

Fractional calculus is increasingly recognized as a valuable tool in epidemic modeling, as it enables more precise representations of disease dynamics, particularly by capturing memory effects and nonlocal interactions. The use of non-integer order in mathematical modeling is gaining growing attention among scientists and researchers. Unlike traditional epidemiological models, which are defined by fixed integer-order derivatives, fractional-order derivative models are characterized by flexible, variable orders, offering a more adaptable framework for understanding complex disease transmission processes. The fractional derivative's modeling capabilities are enhanced by its nonfixed order [40]. In the field of fractional calculus, the operators used are differential operators with fractional or non-integer orders, which exhibit memory-related characteristics. These operators allow for the modeling of systems where the current state depends not only on the present conditions but also on the history of the system, providing a more accurate representation of processes with long-term effects or delays [41]. The use of fractional-order models has proven to be highly effective in predicting the dynamics of real-world processes, making mathematical modeling of such phenomena an invaluable tool [41]. Researchers have investigated fractional-order mathematical models for various diseases, including COVID-19 [42, 43], Chlamydia and Gonorrhea [44], and HIV [45]. Few fractional-order models have been developed to study the dynamics of meningitis. Elsonbaty et al. [46] proposed a bi-susceptible model for the transmission dynamics of meningitis using the Caputo fractional-order operator. Furthermore, they conducted an optimal control and cost-effectiveness analysis on the proposed Caputo fractional-order model. Their findings indicated that Peter et al. [47] developed a mathematical model based on the Atangana–Baleanu Caputo (ABC) derivative to explore the transmission dynamics of meningitis. This model incorporates fractional-order derivatives to better capture the complexities of disease spread, offering a more accurate representation of the dynamics involved in meningitis transmission. Rashid et al. [48] proposed a deterministic model for the co-infection of pneumonia and meningitis using the Atangana–Baleanu fractional derivative operator within the Caputo

framework, accompanied by an optimal analysis. Their results highlighted that adjusting the fractional order from 1 to a lower value required the rapid implementation of the specified control strategy at the highest possible intensity, with its sustained application throughout the majority of the pandemic's duration for effective control.

In this study, we propose a Caputo-based fractional-order model to examine the transmission dynamics of MM, incorporating factors such as the concentration of *Neisseria meningitidis* in the environment (since meningitis transmission can occur through direct and indirect contact with contaminated surfaces and objects like door handles, light switches, and furniture), along with vaccination and hygiene consciousness among susceptible individuals. Additionally, we fit the model to the cumulative confirmed cases of cerebrospinal meningitis in Nigeria. The structure of the paper is as follows: Section 2 outlines the materials and methods used; Section 3 presents the results; and Section 4 provides the concluding remarks.

2 | Materials and Methods

2.1 | Preliminaries

Definition 1 ([49]). The Caputo fractional derivative of order $\beta > 0$ of a function $f(t)$ of order $\beta \in \mathbb{R}^+$ is defined by

$$ {}^{c}\mathcal{D}_t^{\beta} f(t) = \frac{d^{\beta} f(t)}{dt^{\beta}} = \frac{1}{\Gamma(n-\beta)} \int_0^t (t-s)^{(n-\beta-1)} f^{(n)}(s) ds, \tag{1} $$

where n is an integer whose definition is $n-1 < \beta \leq n$. Furthermore, the symbol Γ represent Gamma function, and it is given by

$$ \Gamma(\beta) = \int_0^{\infty} \exp(-s) s^{\beta-1} ds, \quad \Gamma(\beta+1) = \beta\Gamma(\beta), \quad Re(\beta) > 0. \tag{2} $$

Definition 2 ([44]). The fractional integral of order $\beta > 0$ of a function $f \in \mathbb{C}^1(0, \mathcal{T})$ is defined by

$$ \mathcal{I}_t^{\beta} f(t) = \frac{1}{\Gamma(\beta)} \int_0^t (t-s)^{(\beta-1)} f(s) ds, \quad t > 0, \tag{3} $$

provided the integral part is integrable in $\mathbb{R}^+$. For simplicity, suppose $f(t) = X$, where X is any constant, we have

$$ \mathcal{I}_t^{\beta} f(t) = \frac{1}{\Gamma(\beta)} \int_0^t (t-s)^{(\beta-1)} (X) ds = X \frac{t^{\beta}}{\Gamma(\beta+1)}. \tag{4} $$

Definition 3 ([49]). The Laplace transform of the Caputo fractional derivative is given by

$$ \mathscr{L}\left\{ {}^{c}\mathcal{D}_t^{\beta} f(t) \right\} = s^{\beta} \tilde{f}(s) - s^{\beta-1} f(0), \quad 0 < \beta \leq 1, \tag{5} $$

where $\mathscr{L}$ is the operator of the Laplace transform.

Definition 4 ([50]). Euler's reflection formula is given by

$$ \Gamma(\beta) = \frac{\pi}{\Gamma(1-\beta)\sin(\pi\beta)} \tag{6} $$

Lemma 1 ([50]). *Given that $\beta \in \mathbb{R}^+$, $\vartheta_1(t)$ and $\vartheta_2(t)$ represents nonnegative functions and $\vartheta_3(t)$ represent both the nonnegative and increasing function for $0 \leq t \leq \mathcal{T}$, $\mathcal{T} > 0$, $\vartheta_3(t) \leq \mathcal{G}$, where $\mathcal{G}$ is a constant. Suppose*

$$ \vartheta_1 \leq \vartheta_2(t) + \vartheta_3(t) \int_0^t (t-s)^{\beta-1} \vartheta_1(t) ds, \tag{7} $$

then

$$\vartheta_1 \leq \vartheta_2 \mathcal{E}_\beta \left(\vartheta_3(t) \frac{\pi}{\Gamma(1-\beta)\sin(\pi\beta)} \mathcal{T}^\beta \right) \tag{8}$$

2.2 | Formulation of the Model

In this section, we shall design a model for MM dynamics structured within the human population and the concentration of *Neisseria Meningitidis* in the environment. At time t, we assume that the total human population is constant and of the size N. Similarly, we assume that the concentration of *Neisseria Meningitidis* in the environment at time t is represented by $Q(t)$. The total human population $N(t)$ is categorized into seven (7) compartments of susceptible individuals $S(t)$, vaccinated individuals $V(t)$, exposed individuals $E(t)$, asymptomatic infected individuals that are carriers of the *Neisseria Meningitidis* bacteria $C(t)$, symptomatic infected individuals $I(t)$, treated individuals $T(t)$, and recovered individuals $R(t)$. With this, we can say

$$N(t) = S(t) + V(t) + E(t) + C(t) + I(t) + T(t) + R(t)$$

The population of the susceptible individuals is generated by the recruitment of individuals into the population either by birth or migration at a constant rate Λ. Susceptible individuals are vaccinated against MM at the rate θ, and ψ is the waning rate of the MM vaccine. Susceptible individuals are exposed to MM when they come in adequate contact with infected individuals or the concentration of the *Neisseria Meningitidis* in the environment at the rate $(1-\varpi)\zeta$; ζ is given by

$$\zeta = \frac{\alpha_h(C+I)}{N} + \frac{\alpha_e Q}{K+Q}.$$

where ϖ is the hygiene consciousness rate of susceptible individuals, α_h is the effective contact rate between susceptible individuals and infected individuals, α_e is the effective contact rate between susceptible individuals and the *Neisseria Meningitidis* bacteria in the environment, and K is the carrying capacity of the *Neisseria Meningitidis* bacteria. The exposed individuals progressed to being infected at the rate φ, where a proportion $(1-q)\varphi$ are symptomatic, and the remaining proportion $q\varphi$ are asymptomatic. Asymptomatic infected individuals (carriers) progressed to being symptomatic at the rate σ. η_C and η_I are the treatment rates for the asymptomatic and symptomatic infected individuals, respectively. The rate δ is the disease-induced death rate of symptomatic infected individuals, and $\varepsilon\delta$ is the disease-induced death rate of treated individuals (where ε is a modification parameter that accounts for the reduction in meningitis-related death while receiving treatment). Treated individuals recover from meningitis at the rate ϕ, and recovered individuals become susceptible again at the rate μ. The human natural death rate is given by γ, and it is the same in all human compartments. The concentration of the *Neisseria Meningitidis* bacteria in the environment is generated by the shedding of the bacteria by asymptomatic and symptomatic infected individuals at the rates τ_1 and τ_2, respectively. The decay rate of bacteria in the environment is given by ξ. The model variables and parameters are presented in Table 1. The model diagram is also presented in Figure 1. Based on the aforementioned assumptions and formulation, the Meningitis model is governed by the following system of nonlinear fractional-order differential equations:

$$\begin{aligned}
{}^c\mathcal{D}_t^\beta S &= \Lambda - (1-\varpi)\zeta S - (\theta+\gamma)S + \mu R + \psi V,\\
{}^c\mathcal{D}_t^\beta V &= \theta S - (\psi+\gamma)V,\\
{}^c\mathcal{D}_t^\beta E &= (1-\varpi)\zeta S - (\varphi+\gamma)E,\\
{}^c\mathcal{D}_t^\beta C &= q\varphi E - \left(\eta_C + \sigma + \gamma\right)C,\\
{}^c\mathcal{D}_t^\beta I &= (1-q)\varphi E + \sigma C - \left(\eta_I + \delta + \gamma\right)I,\\
{}^c\mathcal{D}_t^\beta T &= \eta_C C + \eta_I I - (\phi + \varepsilon\delta + \gamma)T,\\
{}^c\mathcal{D}_t^\beta R &= \phi T - (\mu+\gamma)R,\\
{}^c\mathcal{D}_t^\beta Q &= \tau_1 C + \tau_2 I - \xi Q.
\end{aligned} \tag{9}$$

where

$$\zeta = \frac{\alpha_h(C+I)}{N} + \frac{\alpha_e Q}{K+Q} \tag{10}$$

TABLE 1 | Description of the model variables.

Variable	Description
S	Population of susceptible humans.
V	Population of vaccinated humans against meningococcal meningitis.
E	Population of exposed humans to meningococcal meningitis.
C	Population of asymptomatic infected humans (carriers of the *Neisseria Meningitidis* bacteria).
I	Population of symptomatic infected humans with meningococcal meningitis.
T	Population of treated humans with meningococcal meningitis.
R	Population of recovered humans from meningococcal meningitis.
Q	The concentration of *Neisseria Meningitidis* in the environment.

Parameter	Description
Λ	Recruitment rate for humans.
γ	Natural death rate for humans.
α_h	Effective contact rate between susceptible humans and infected humans with meningococcal meningitis infection.
α_e	Effective contact rate between susceptible humans and the *Neisseria Meningitidis* bacteria in the environment.
ϖ	Hygiene consciousness rate of susceptible individuals.
φ	Progression rate from the exposed class to the infected classes with meningococcal meningitis.
q	Proportion of exposed individuals who progress to being infectious and are asymptomatic (carriers).
μ	Rate at which recovered humans from meningococcal meningitis become susceptible again.
θ	Vaccination rate against meningococcal meningitis.
ψ	Waning-rate of meningococcal meningitis vaccine.
σ	Progression rate from asymptomatic to symptomatic.
δ	Disease-induced death rate.
ϕ	Recovery rate of treated individuals.
ϵ	Modification parameter that accounts for reduction in meningococcal meningitis disease-induced death in the treatment class.
η_C	Treatment rate of asymptomatic infected individuals (carriers).
η_I	Treatment rate of symptomatic infected individuals.
τ_1	Shedding rate of *Neisseria Meningitidis* bacteria in the environment from asymptomatic infected individuals (carriers) with meningococcal meningitis only.
τ_2	Shedding rate of *Neisseria Meningitidis* bacteria in the environment from symptomatic infected individuals with meningococcal meningitis that are exposed to malaria infection.
ξ	Decay rate of *Neisseria Meningitidis* bacteria in the environment.
K	Carrying capacity.

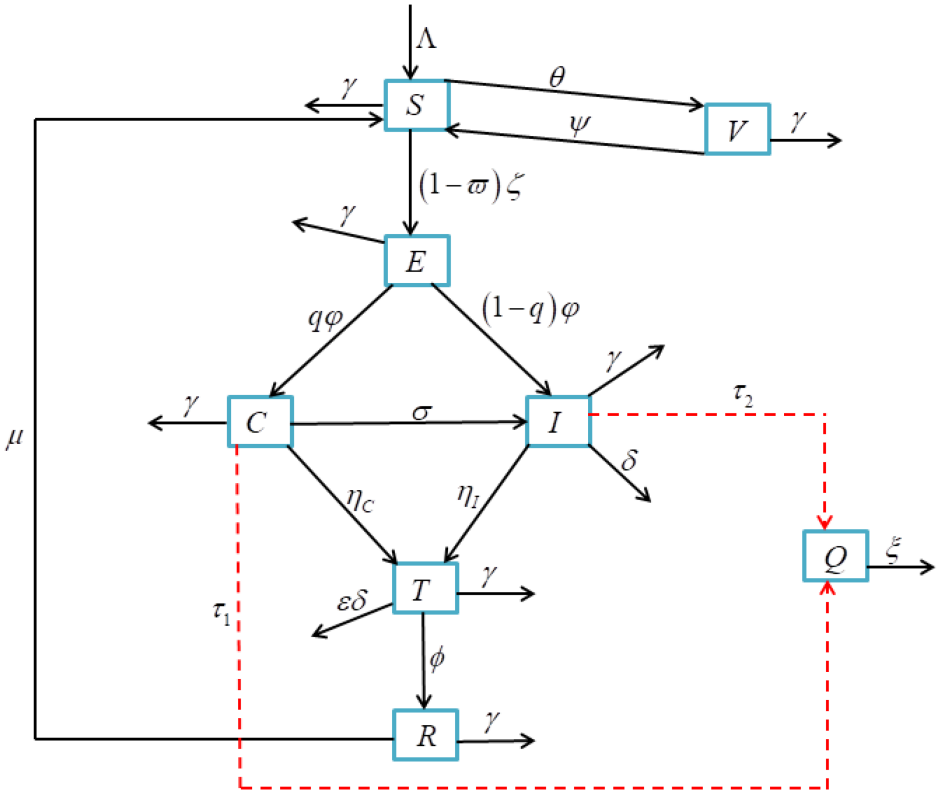

FIGURE 1 | Schematic diagram of the Meningitis model.

With the initial conditions:

$$S(0) \geq 0, V(0) \geq 0, E(0) \geq 0, C(0) \geq 0, I(0) \geq 0, T(0) \geq 0, R(0) \geq 0, \quad \textit{and} \quad Q(0) \geq 0. \tag{11}$$

2.3 | Invariant Domain

Theorem 1. *Given that $S(t)$, $V(t)$, $E(t)$, $C(t)$, $I(t)$, $T(t)$, $R(t)$, are solutions to the model system (9), then the set $\Theta = \Theta_H \cup \Theta_e$, where*

$$\Theta_H = \left\{ (S(t), V(t), E(t), C(t), I(t), T(t), R(t)) \in \mathbb{R}_+^7 : S + V + E + C + I + T + R \leq \frac{\Lambda}{\gamma} \right\} \tag{12}$$

and

$$\Theta_e = \left\{ Q : Q \leq \frac{\tau^*}{\xi}\left(\frac{\Lambda}{\gamma}\right) \right\}, \tag{13}$$

is positively invariant with respect to the proposed model (9).

Proof. Recall that the expression for the total human population at time t is given by

$$N(t) = S(t) + V(t) + E(t) + C(t) + I(t) + T(t) + R(t).$$

Thus, adding the equations for the human compartments gives

$${}^c\mathcal{D}_t^\beta N(t) = {}^c\mathcal{D}_t^\beta S(t) + {}^c\mathcal{D}_t^\beta V(t) + {}^c\mathcal{D}_t^\beta E(t) + {}^c\mathcal{D}_t^\beta C(t) + {}^c\mathcal{D}_t^\beta I(t) + {}^c\mathcal{D}_t^\beta T(t) + {}^c\mathcal{D}_t^\beta R(t)$$

which results in

$${}^c\mathcal{D}_t^\beta N(t) = \Lambda - \gamma(S + V + E + C + I + T + R) - (\delta I + \varepsilon\delta T) \tag{14}$$

We have that

$${}^c\mathcal{D}_t^\beta N(t) \leq \Lambda - \gamma N \tag{15}$$

Applying the Laplace transform to the inequality above, we have

$$s^{\beta}\tilde{N}(s) - s^{\beta-1}N(0) \leq \frac{\Lambda}{s} - \gamma\tilde{N}(s),$$

from which we obtained

$$\tilde{N}(s) \leq \frac{\Lambda}{s\left(s^{\beta}+\gamma\right)} + N(0)\frac{s^{\beta-1}}{\left(s^{\beta}+\gamma\right)} \tag{16}$$

Using the method of partial fraction to decompose the above equation, it can be rewritten as

$$\tilde{N}(s) \leq \frac{\Lambda}{\gamma}\left(\frac{1}{s}\right) - \left(\frac{\Lambda}{\gamma} - N(0)\right)\sum_{k=0}^{\infty}\frac{(-\gamma)^k}{s^{\beta k+1}} \tag{17}$$

The inverse Laplace transform of the above inequality gives

$$N(t) \leq \frac{\Lambda}{\gamma} - \left(\frac{\Lambda}{\gamma} - N(0)\right)\mathbb{E}_{\beta}(-\gamma t^{\beta}), \tag{18}$$

as $t \to \infty$, we have

$$N(t) \leq \frac{\Lambda}{\gamma} \tag{19}$$

Giving the condition for the human compartments in Equations (9–11) to be bounded and mathematically posed within the domain.

Similarly, we recall that the expression for the concentration of *Neisseria Meningitidis* in the environment is given by

$${}^{c}\mathcal{D}_{t}^{\beta}Q = \tau_1 C + \tau_2 I - \xi Q.$$

We have that

$${}^{c}\mathcal{D}_{t}^{\beta}Q \leq \tau^{*}(C+I) - \xi Q, \tag{20}$$

where $\tau^{*} = \max\left(\tau_1, \tau_2\right)$.

We let $C + I \leq \frac{\Lambda}{\gamma}$ since the human population is bounded by $\frac{\Lambda}{\gamma}$, which gives

$${}^{c}\mathcal{D}_{t}^{\beta}Q \leq \tau^{*}\left(\frac{\Lambda}{\gamma}\right) - \xi Q, \tag{21}$$

Applying the Laplace transform to the inequality above, we have

$$s^{\beta}\tilde{Q}(s) - s^{\beta-1}Q(0) \leq \frac{\tau^{*}}{s}\left(\frac{\Lambda}{\gamma}\right) - \xi\tilde{Q}(s), \tag{22}$$

from which we obtain

$$\tilde{Q}(s) \leq \frac{\tau^{*}\Phi}{s\left(s^{\beta}+\xi\right)} + Q(0)\frac{s^{\beta-1}}{\left(s^{\beta}+\xi\right)}, \tag{23}$$

where $\Phi = \frac{\Lambda}{\gamma}$.

Applying the partial fraction technique, the above inequality can be rewritten as

$$\tilde{Q}(s) \leq \frac{\tau^{*}\Phi}{\xi}\left(\frac{1}{s}\right) - \left(\frac{\tau^{*}\Phi}{\xi} - Q(0)\right)\sum_{k=0}^{\infty}\frac{(-\xi)^k}{s^{\beta k+1}} \tag{24}$$

Applying the inverse Laplace transform to the above inequality gives

$$Q(t) \leq \frac{\tau^{*}\Phi}{\xi} - \left(\frac{\tau^{*}\Phi}{\xi} - Q(0)\right)\mathbb{E}_{\beta}(-\xi t^{\beta}), \tag{25}$$

which can be written as

$$Q(t) \leq \frac{\tau^*}{\xi}\left(\frac{\Lambda}{\gamma}\right) - \left(\frac{\tau^*}{\xi}\left(\frac{\Lambda}{\gamma}\right) - Q(0)\right)\mathbb{E}_\beta(-\xi t^\beta), \tag{26}$$

as $t \to \infty$, we have

$$Q(t) \leq \frac{\tau^*}{\xi}\left(\frac{\Lambda}{\gamma}\right) \tag{27}$$

Which is the condition for the concentration of *Neisseria Meningitidis* in the environment to be bounded and well-posed within the domain □

2.4 | Positivity of the Model System Solution

Following the approach described in [44], by contradiction, let us assume that the third equation of the meningitis fractional-order model (9) is not positively invariant.

$$\Delta_1 = \min_{0\leq t\leq t_1}\left\{\frac{(1-\varpi)\zeta S}{E} - (\psi+\gamma)\right\} \tag{28}$$

It follows that

$${}^c\mathcal{D}_t^\beta E(t) - \Delta_1 E(t) > 0 \tag{29}$$

Let λ_1 be a continuous function. We can say that the following equation can be established

$${}^c\mathcal{D}_t^\beta E(t) - \Delta_1 E(t) = -\lambda_1(t) \tag{30}$$

Applying the Laplace transform to the equation above gives

$$s^\beta \tilde{E}(s) - s^{\beta-1}E(0) - \Delta_1 \tilde{E}(s) = -\tilde{\lambda}_1(s), \tag{31}$$

We have that

$$\begin{aligned}\tilde{E}(s) &= E(0)\frac{s^{\beta-1}}{s^\beta-\Delta_1} - \frac{\lambda_1(s)}{s^\beta-\Delta_1} = \frac{E(0)}{s}\left(1-\frac{\Delta_1}{s^\beta}\right)^{-1} - \frac{\lambda_1(s)}{s^\beta}\left(1-\frac{\Delta_1}{s^\beta}\right)^{-1},\\ &= E(0)\sum_{k=0}^{\infty}\frac{\Delta_1^k}{s^{\beta k+1}} - \lambda_1(s)\sum_{k=0}^{\infty}\frac{\Delta_1^k}{s^{\beta k+\beta}}\end{aligned} \tag{32}$$

Employing the Mittag-Leffler function and ignoring the negative term, the inverse Laplace transform gives the solution of (29) satisfying the expression below

$$E(t) > E(0)\sum_{k=0}^{\infty}\frac{\left(\Delta_1 t^\beta\right)^k}{\Gamma(\beta k+1)} = E(0)\mathbb{E}_\beta\left(\Delta_1 t^\beta\right) \tag{33}$$

From which the positivity of E is given by

$$E(t) > E(0)\mathbb{E}_\beta\left(\Delta_1 t^\beta\right) > 0 \tag{34}$$

This contradict $E(t_1) = 0$. In similar reasoning, suppose $C(t_1) = 0$ which implies that $S(t) > 0$, $V(t) > 0$, $E(t) > 0$, $I(t) > 0$, $T(t) > 0$, $R(t) > 0$, and $Q(t) > 0$, for $0 \leq t \leq t_1$. We assume that the following expression exists

$$\Delta_2 = \min_{0\leq t\leq t_1}\left\{\frac{q\varphi E}{C} - (\eta_C+\sigma+\gamma)\right\}, \tag{35}$$

Then it follows that

$${}^c\mathcal{D}_t^\beta C(t) > \Delta_2 C(t) \tag{36}$$

It follows that a continuous function $\lambda_2(t)$ may be ascertained in a way that the following equation is discovered

$${}^c\mathcal{D}_t^\beta C(t) - \Delta_2 C(t) = -\lambda_2(t) \tag{37}$$

Applying the Laplace transform to the above equation yields

$$s^{\beta}\tilde{C}(s) - s^{\beta-1}C(0) - \Delta_2\tilde{C}(s) = -\tilde{\lambda}_2(s), \tag{38}$$

which results in

$$\tilde{C}(s) = C(0)\frac{s^{\beta-1}}{s^{\beta}-\Delta_2} - \frac{\lambda_2(s)}{s^{\beta}-\Delta_2} = C(0)\sum_{k=0}^{\infty}\frac{\Delta_2^k}{s^{\beta k+1}} - \lambda_2(s)\sum_{k=0}^{\infty}\frac{\Delta_2^k}{s^{\beta k+\beta}} \tag{39}$$

Employing the Mittag-Leffler function and ignoring the negative term, the inverse Laplace transform gives the solution of (36) satisfying the expression below

$$C(t) > C(0)\sum_{k=0}^{\infty}\frac{\left(\Delta_2 t^{\beta}\right)^k}{\Gamma(\beta k+1)} = C(0)\mathbb{E}_{\beta}\left(\Delta_2 t^{\beta}\right). \tag{40}$$

Thus, the positivity of C is given by

$$C(t) > C(0)\mathbb{E}_{\beta}\left(\Delta_2 t^{\beta}\right) > 0 \tag{41}$$

This contradict $C(t_1) = 0$. Hence, using the same process, it can be shown that the positivity of the solution S, V, I, T, R, and Q, respectively, is as follows

$$\begin{aligned}
S(t) &> S(0)\mathbb{E}_{\beta}\left(\Delta_3 t^{\beta}\right) > 0,\\
V(t) &> V(0)\mathbb{E}_{\beta}\left(\Delta_4 t^{\beta}\right) > 0,\\
I(t) &> I(0)\mathbb{E}_{\beta}\left(\Delta_5 t^{\beta}\right) > 0,\\
T(t) &> T(0)\mathbb{E}_{\beta}\left(\Delta_6 t^{\beta}\right) > 0,\\
R(t) &> R(0)\mathbb{E}_{\beta}\left(\Delta_7 t^{\beta}\right) > 0,\\
Q(t) &> Q(0)\mathbb{E}_{\beta}\left(\Delta_8 t^{\beta}\right) > 0.
\end{aligned} \tag{42}$$

2.5 | Existence and Uniqueness of Solution of the Meningitis Model

In this section, we will prove the existence and uniqueness of the solution to the Meningitis model using the Banach fixed-point theorem, as demonstrated in [50, 51]. Additionally, we will utilize Schafer's fixed-point theorem to establish the existence of the solution and demonstrate its boundedness.

Applying the fractional integral to the Caputo fractional derivative of the Meningitis model (9) of order $\beta > 0$, along with the appropriate initial conditions (11), we obtain the corresponding Volterra integral equations of the second kind. These integral equations will represent the solution to the fractional-order Meningitis model (9).

$$\begin{aligned}
S(t) - S(0) &= \frac{1}{\Gamma(\beta)}\int_0^t (t-s)^{\beta-1}A(s,S(s))ds,\\
V(t) - V(0) &= \frac{1}{\Gamma(\beta)}\int_0^t (t-s)^{\beta-1}W(s,V(s))ds,\\
E(t) - E(0) &= \frac{1}{\Gamma(\beta)}\int_0^t (t-s)^{\beta-1}U(s,E(s))ds,\\
C(t) - C(0) &= \frac{1}{\Gamma(\beta)}\int_0^t (t-s)^{\beta-1}F(s,C(s))ds,\\
I(t) - I(0) &= \frac{1}{\Gamma(\beta)}\int_0^t (t-s)^{\beta-1}H(s,I(s))ds,\\
T(t) - T(0) &= \frac{1}{\Gamma(\beta)}\int_0^t (t-s)^{\beta-1}P(s,T(s))ds,\\
R(t) - R(0) &= \frac{1}{\Gamma(\beta)}\int_0^t (t-s)^{\beta-1}B(s,R(s))ds,
\end{aligned}$$

$$Q(t) - Q(0) = \frac{1}{\Gamma(\beta)} \int_0^t (t-s)^{\beta-1} Z(s, Q(s)) ds, \tag{43}$$

We assume that the functions $(A, W, U, F, H, P, B, Z) : [0, \mathcal{T}] \times \mathcal{X} \to \mathcal{X}$ are continuous such that $(\mathcal{X}, \|.\|)$ is the Banach space and $\mathbb{H}^1([0, \mathcal{T}])$ is the Banach space of all the continuous functions defined in $[0, \mathcal{T}] \to \mathcal{X}$ shaped with Chebyshev norm.

Subsequently, we demonstrate that the continuous functions A, W, U, F, H, P, B, and Z satisfies the Lipschitz condition if

$$\sup_{0<t\leq\mathcal{T}} \left\| \frac{C}{N} \right\| \leq \Omega_1, \sup_{0<t\leq\mathcal{T}} \left\| \frac{I}{N} \right\| \leq \Omega_2, \quad and \quad \sup_{0<t\leq\mathcal{T}} \left\| \frac{Q}{K+Q} \right\| \leq \Omega_3 \tag{44}$$

First we have

$$\begin{aligned}
\|A(S_1) - A(S_2)\| &= \left\| \Lambda - (1-\varpi)\left(\frac{\alpha_h(C+I)}{N} + \frac{\alpha_e Q}{K+Q} \right) S_1 - (\theta+\gamma) S_1 + \mu R + \psi V \right. \\
&\quad \left. - \left(\Lambda - (1-\varpi)\left(\frac{\alpha_h(C+I)}{N} + \frac{\alpha_e Q}{K+Q} \right) S_2 - (\theta+\gamma) S_2 + \mu R + \psi V \right) \right\| \\
&= \left\| -\frac{(1-\varpi)\alpha_h C}{N}(S_1 - S_2) - \frac{(1-\varpi)\alpha_h I}{N}(S_1 - S_2) - \theta(S_1 - S_2) \right. \\
&\quad \left. - \frac{(1-\varpi)\alpha_e Q}{K+Q}(S_1 - S_2) - \gamma(S_1 - S_2) \right\| \\
&\leq (1-\varpi)\alpha_h \sup_{0<t\leq\mathcal{T}} \left\| \frac{C}{N} \right\| \|S_1 - S_2\| + (1-\varpi)\alpha_h \sup_{0<t\leq\mathcal{T}} \left\| \frac{I}{N} \right\| \|S_1 - S_2\| \\
&\quad + (1-\varpi)\alpha_e \sup_{0<t\leq\mathcal{T}} \left\| \frac{Q}{K+Q} \right\| \|S_1 - S_2\| + \theta \|S_1 - S_2\| + \gamma \|S_1 - S_2\| \\
&\leq \mathscr{M}_A \|S_1 - S_2\|,
\end{aligned} \tag{45}$$

where $\mathscr{M}_A = \big((1-\varpi)\big(\alpha_h\Omega_1 + \alpha_h\Omega_2 + \alpha_e\Omega_3\big) + \theta + \gamma\big) > 0$.

Secondly, we have

$$\begin{aligned}
\|W(V_1) - W(V_2)\| &= \left\| \theta S - (\psi+\gamma) V_1 - \big(\theta S - (\psi+\gamma) V_2\big) \right\| \\
&= \|-\psi(V_1 - V_2) - \gamma(V_1 - V_2)\| \\
&\leq \psi \|V_1 - V_2\| + \gamma \|V_1 - V_2\| \\
&\leq \mathscr{M}_W \|V_1 - V_2\|,
\end{aligned} \tag{46}$$

where $\mathscr{M}_W = (\psi + \gamma) > 0$.

Then we have

$$\begin{aligned}
\|U(E_1) - U(E_2)\| &= \left\| (1-\varpi)\left(\frac{\alpha_h(C+I)}{N} + \frac{\alpha_e Q}{K+Q} \right) S - (\varphi+\gamma) E_1 \right. \\
&\quad \left. - \left((1-\varpi)\left(\frac{\alpha_h(C+I)}{N} + \frac{\alpha_e Q}{K+Q} \right) S - (\varphi+\gamma) E_2 \right) \right\| \\
&= \|-\varphi(E_1 - E_2) - \gamma(E_1 - E_2)\| \\
&\leq \varphi \|E_1 - E_2\| + \gamma \|E_1 - E_2\| \\
&\leq \mathscr{M}_U \|E_1 - E_2\|,
\end{aligned} \tag{47}$$

where $\mathscr{M}_U = (\varphi + \gamma) > 0$.

We have

$$\|F(C_1) - F(C_2)\| = \left\| q\varphi E - (\eta_C + \sigma + \gamma) C_1 - \big(q\varphi E - (\eta_C + \sigma + \gamma) C_2\big) \right\|$$

$$
\begin{aligned}
&= \|-\eta_C(C_1 - C_2) - \sigma(C_1 - C_2) - \gamma(C_1 - C_2)\| \\
&\leq \eta_C\|C_1 - C_2\| + \sigma\|C_1 - C_2\| + \gamma\|C_1 - C_2\| \\
&\mathscr{M}_F\|C_1 - C_2\|,
\end{aligned} \tag{48}
$$

where $\mathscr{M}_F = (\eta_C + \sigma + \gamma) > 0$.

We have

$$
\begin{aligned}
\|H(I_1) - H(I_2)\| &= \left\|(1-q)\varphi E + \sigma C - (\eta_I + \delta + \gamma)I_1 - \left((1-q)\varphi E + \sigma C - (\eta_I + \delta + \gamma)I_2\right)\right\| \\
&= \|-\eta_I(I_1 - I_2) - \delta(I_1 - I_2) - \gamma(I_1 - I_2)\| \\
&\leq \eta_I\|I_1 - I_2\| + \delta\|I_1 - I_2\| + \gamma\|I_1 - I_2\| \\
&\leq \mathscr{M}_H\|I_1 - I_2\|,
\end{aligned} \tag{49}
$$

where $\mathscr{M}_H = (\eta_I + \delta + \gamma) > 0$.

We have

$$
\begin{aligned}
\|P(T_1) - P(T_2)\| &= \left\|\eta_C C + \eta_I I - (\phi + \varepsilon\delta + \gamma)T_1 - \left(\eta_C C + \eta_I I - (\phi + \varepsilon\delta + \gamma)T_2\right)\right\| \\
&= \|-\phi(T_1 - T_2) - \varepsilon\delta(T_1 - T_2) - \gamma(T_1 - T_2)\| \\
&\leq \phi\|T_1 - T_2\| + \varepsilon\delta\|T_1 - T_2\| + \gamma\|T_1 - T_2\| \\
&\leq \mathscr{M}_P\|T_1 - T_2\|,
\end{aligned} \tag{50}
$$

where $\mathscr{M}_P = (\phi + \varepsilon\delta + \gamma) > 0$.

We have

$$
\begin{aligned}
\|B(R_1) - B(R_2)\| &= \left\|\phi T - (\mu + \gamma)R_1 - \left(\phi T - (\mu + \gamma)R_2\right)\right\| \\
&= \|-\mu(R_1 - R_2) - \gamma(R_1 - R_2)\| \\
&\leq \mu\|R_1 - R_2\| + \gamma\|R_1 - R_2\| \\
&\leq \mathscr{M}_B\|R_1 - R_2\|,
\end{aligned} \tag{51}
$$

where $\mathscr{M}_B = (\mu + \gamma) > 0$.

Finally, we have

$$
\begin{aligned}
\|Z(Q_1) - Z(Q_2)\| &= \left\|\tau_1 C + \tau_2 I - \xi Q_1 - \left(\tau_1 C + \tau_2 I - \xi Q_2\right)\right\| \\
&= \|-\xi(Q_1 - Q_2)\| \\
&\leq \xi\|Q_1 - Q_2\| \\
&\leq \mathscr{M}_Z\|Q_1 - Q_2\|,
\end{aligned} \tag{52}
$$

where $\mathscr{M}_Z = \xi > 0$.

Theorem 2. *If* $(A, W, U, F, H, P, B, Z)\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^{\beta}}{\beta\pi} < 1$, *then the meningitis fractional-order model (9–11) has a unique solution on* $[0, \mathcal{T}]$, *assuming that* $(A, W, U, F, H, P, B, Z) : [0, \mathcal{T}] \times \mathcal{X} \to \mathcal{X}$ *are continuous and satisfies the Lipschitz criteria*.

Proof. Let consider the mapping $\varrho : \mathbb{H}^1([0, \mathcal{T}], \mathcal{X}) \to \mathbb{H}^1([0, \mathcal{T}], \mathcal{X})$ and given that ϱ is defined in

$(A, W, U, F, H, P, B, Z) : [0, \mathcal{T}] \times \mathcal{X} \to \mathcal{X}$. Utilizing (45–52) and $\forall\, ((S_1, S_2), (V_1, V_2), (E_1, E_2), (C_1, C_2), (I_1, I_2), (T_1, T_2), (R_1, R_2), (Q_1, Q_2)) \in \mathbb{H}^1([0, \mathcal{T}], \mathcal{X})$ and $0 \le t \le \mathcal{T}$, we obtained

$$\begin{aligned}
\left\|\varrho(S_1(t)) - \varrho(S_2(t))\right\| &= \left\| S(0) + \frac{1}{\Gamma(\beta)} \int_0^t (t-s)^{\beta-1} A\big(s, S_1(s)\big) ds \right. \\
&\quad \left. - \left(S(0) + \frac{1}{\Gamma(\beta)} \int_0^t (t-s)^{\beta-1} A\big(s, S_2(s)\big) ds \right) \right\| \\
&\le \frac{1}{\Gamma(\beta)} \int_0^t (t-s)^{\beta-1} \left\| A\big(s, S_1(s)\big) - A\big(s, S_2(s)\big) \right\| ds \\
&\le \frac{\mathcal{M}_A}{\Gamma(\beta)} \int_0^t (t-s)^{\beta-1} \left\| S_1(s) - S_2(s) \right\| ds \\
&\le \mathcal{M}_A \left(\frac{\mathcal{T}^\beta}{\Gamma(\beta+1)} \right) \left\| S_1 - S_2 \right\|_{\mathbb{H}^1} \\
&\le \mathcal{M}_A \left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi} \right) \left\| S_1 - S_2 \right\|_{\mathbb{H}^1}. \qquad (53)
\end{aligned}$$

In Similar reasoning, we have

$$\begin{aligned}
\left\|\varrho(V_1(t)) - \varrho(V_2(t))\right\| &\le \mathcal{M}_W \left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi} \right) \left\| V_1 - V_2 \right\|_{\mathbb{H}^1}, \\
\left\|\varrho(E_1(t)) - \varrho(E_2(t))\right\| &\le \mathcal{M}_U \left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi} \right) \left\| E_1 - E_2 \right\|_{\mathbb{H}^1}, \\
\left\|\varrho(C_1(t)) - \varrho(C_2(t))\right\| &\le \mathcal{M}_F \left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi} \right) \left\| C_1 - C_2 \right\|_{\mathbb{H}^1}, \\
\left\|\varrho(I_1(t)) - \varrho(I_2(t))\right\| &\le \mathcal{M}_H \left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi} \right) \left\| I_1 - I_2 \right\|_{\mathbb{H}^1}, \\
\left\|\varrho(T_1(t)) - \varrho(T_2(t))\right\| &\le \mathcal{M}_P \left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi} \right) \left\| T_1 - T_2 \right\|_{\mathbb{H}^1}, \\
\left\|\varrho(R_1(t)) - \varrho(R_2(t))\right\| &\le \mathcal{M}_B \left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi} \right) \left\| R_1 - R_2 \right\|_{\mathbb{H}^1}, \\
\left\|\varrho(Q_1(t)) - \varrho(Q_2(t))\right\| &\le \mathcal{M}_Z \left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi} \right) \left\| Q_1 - Q_2 \right\|_{\mathbb{H}^1}. \qquad (54)
\end{aligned}$$

This indicates from the condition that $(A, W, U, F, H, P, B, Z) \frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi} < 1$. The application of the Banach contraction mapping principle indicates that the parameter ϱ has a unique fixed point in $0 \le t \le \mathcal{T}$, since it is a contraction mapping. □

Next, we shall examine the existence of solutions of the meningitis fractional-order model (9–11) using the Schaefar's fixed-point theorem.

Theorem 3. *Given that* $(A, W, U, F, H, P, B, Z) : [0, \mathcal{T}] \times \mathcal{X} \to \mathcal{X}$ *are continuous and that there exist constants* $(\mathcal{M}_{A1}, \mathcal{M}_{W1}, \mathcal{M}_{U1}, \mathcal{M}_{F1}, \mathcal{M}_{H1}, \mathcal{M}_{P1}, \mathcal{M}_{B1}, \mathcal{M}_{Z1}) > 0$ *such that*

$$\begin{aligned}
&\|A(t,S)\| \le \mathcal{M}_{A1}(d + \|S\|), \quad \|W(t,V)\| \le \mathcal{M}_{W1}(d + \|V\|), \quad \|U(t,E)\| \le \mathcal{M}_{U1}(d + \|E\|), \\
&\|F(t,C)\| \le \mathcal{M}_{F1}(d + \|C\|), \quad \|H(t,I)\| \le \mathcal{M}_{H1}(d + \|I\|), \quad \|P(t,T)\| \le \mathcal{M}_{P1}(d + \|T\|), \\
&\|B(t,R)\| \le \mathcal{M}_{B1}(d + \|R\|), \quad \|Z(t,Q)\| \le \mathcal{M}_{Z1}(d + \|Q\|),
\end{aligned}$$

where $0 < d \le 1$ *is an arbitrary number, then* (9–11) *has atleast one solution.*

Proof. The operate ϱ is continuous from (53, 54). Let $\{S^{n+1}\}_\infty$, $\{V^{n+1}\}_\infty$, $\{E^{n+1}\}_\infty$, $\{C^{n+1}\}_\infty$, $\{I^{n+1}\}_\infty$, $\{T^{n+1}\}_\infty$, $\{R^{n+1}\}_\infty$, and $\{Q^{n+1}\}_\infty$ be sequences such that $S^{n+1} \to S^n$, $V^{n+1} \to V^n$, $E^{n+1} \to E^n$, $C^{n+1} \to C^n$, $I^{n+1} \to I^n$, $T^{n+1} \to T^n$,

$R^{n+1} \to R^n$, and $Q^{n+1} \to Q^n$ in $\mathbb{H}^1([0,\mathcal{T}],\mathcal{X})$, then for $0 \le t \le \mathcal{T}$ we have

$$\begin{aligned}
\left\|\varrho S^{n+1}(t) - \varrho S^n(t)\right\| &= \frac{1}{\Gamma(\beta)}\left\|\int_0^t (t-s)^{\beta-1} A\left(s, S^{n+1}(s)\right) ds - \int_0^t (t-s)^{\beta-1} A(s, S^n(s)) ds\right\| \\
&\le \frac{1}{\Gamma(\beta)}\int_0^t (t-s)^{\beta-1}\left\|A\left(s, S^{n+1}(s)\right) - A(s, S^n(s))\right\| ds \\
&\le \mathscr{M}_{A1}\left(\frac{\mathcal{T}^\beta}{\Gamma(\beta+1)}\right)\left\|S^{n+1} - S^n\right\|_{\mathbb{H}^1} \\
&\le \mathscr{M}_{A1}\left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi}\right)\left\|S^{n+1} - S^n\right\|_{\mathbb{H}^1},
\end{aligned} \tag{55}$$

where $\left\|S^{n+1} - S^n\right\|_{\mathbb{H}^1} \to 0$ as $n \to \infty$.

Utilizing the same process, we obtained the following results

$$\begin{aligned}
\left\|\varrho V^{n+1}(t) - \varrho V^n(t)\right\| &\le \mathscr{M}_{W1}\left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi}\right)\left\|V^{n+1} - V^n\right\|_{\mathbb{H}^1}, \\
\left\|\varrho E^{n+1}(t) - \varrho E^n(t)\right\| &\le \mathscr{M}_{U1}\left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi}\right)\left\|E^{n+1} - E^n\right\|_{\mathbb{H}^1}, \\
\left\|\varrho C^{n+1}(t) - \varrho C^n(t)\right\| &\le \mathscr{M}_{F1}\left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi}\right)\left\|C^{n+1} - C^n\right\|_{\mathbb{H}^1}, \\
\left\|\varrho I^{n+1}(t) - \varrho I^n(t)\right\| &\le \mathscr{M}_{H1}\left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi}\right)\left\|I^{n+1} - I^n\right\|_{\mathbb{H}^1}, \\
\left\|\varrho T^{n+1}(t) - \varrho T^n(t)\right\| &\le \mathscr{M}_{P1}\left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi}\right)\left\|T^{n+1} - T^n\right\|_{\mathbb{H}^1}, \\
\left\|\varrho R^{n+1}(t) - \varrho R^n(t)\right\| &\le \mathscr{M}_{B1}\left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi}\right)\left\|R^{n+1} - R^n\right\|_{\mathbb{H}^1}, \\
\left\|\varrho Q^{n+1}(t) - \varrho Q^n(t)\right\| &\le \mathscr{M}_{Z1}\left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi}\right)\left\|Q^{n+1} - Q^n\right\|_{\mathbb{H}^1},
\end{aligned} \tag{56}$$

where $\left\|V^{n+1} - V^n\right\|_{\mathbb{H}^1} \to 0$, $\left\|E^{n+1} - E^n\right\|_{\mathbb{H}^1} \to 0$, $\left\|C^{n+1} - C^n\right\|_{\mathbb{H}^1} \to 0$, $\left\|I^{n+1} - I^n\right\|_{\mathbb{H}^1} \to 0$,

$\left\|T^{n+1} - T^n\right\|_{\mathbb{H}^1} \to 0$, $\left\|R^{n+1} - R^n\right\|_{\mathbb{H}^1} \to 0$, and $\left\|Q^{n+1} - Q^n\right\|_{\mathbb{H}^1} \to 0$ as $n \to \infty$. Therefore, ϱ is continuous. □

Furthermore, we have to demonstrate that the operator ϱ is a one-to-one bounded function on the set $\mathbb{H}^1([0,\mathcal{T}],\mathcal{X})$. For each $S \in \Upsilon_S$, $V \in \Upsilon_V$, $E \in \Upsilon_E$, $C \in \Upsilon_C$, $I \in \Upsilon_I$, $T \in \Upsilon_T$, $R \in \Upsilon_R$, $Q \in \Upsilon_Q$, and for $x > 0$, there exist a corresponding value $y > 0$ such that $\|\varrho S\| \le y$, $\|\varrho V\| \le y$, $\|\varrho E\| \le y$, $\|\varrho C\| \le y$, $\|\varrho I\| \le y$, $\|\varrho T\| \le y$, $\|\varrho R\| \le y$, $\|\varrho Q\| \le y$, and the subset of Banach space of all continuous functions on the interval $0 \le t \le \mathcal{T}$ are defined by

$$\begin{aligned}
\Upsilon_S &= \left\{S \in \mathbb{H}^1([0,\mathcal{T}],\mathcal{X}) : \|S\| \le x\right\}, \\
\Upsilon_V &= \left\{V \in \mathbb{H}^1([0,\mathcal{T}],\mathcal{X}) : \|V\| \le x\right\}, \\
\Upsilon_E &= \left\{E \in \mathbb{H}^1([0,\mathcal{T}],\mathcal{X}) : \|E\| \le x\right\}, \\
\Upsilon_C &= \left\{C \in \mathbb{H}^1([0,\mathcal{T}],\mathcal{X}) : \|C\| \le x\right\}, \\
\Upsilon_I &= \left\{I \in \mathbb{H}^1([0,\mathcal{T}],\mathcal{X}) : \|I\| \le x\right\}, \\
\Upsilon_T &= \left\{T \in \mathbb{H}^1([0,\mathcal{T}],\mathcal{X}) : \|T\| \le x\right\}, \\
\Upsilon_R &= \left\{R \in \mathbb{H}^1([0,\mathcal{T}],\mathcal{X}) : \|R\| \le x\right\}, \\
\Upsilon_Q &= \left\{Q \in \mathbb{H}^1([0,\mathcal{T}],\mathcal{X}) : \|Q\| \le x\right\}.
\end{aligned} \tag{57}$$

Therefore, for any $0 \le t \le \mathcal{T}$,

$$
\begin{aligned}
\|\varrho S\| &\leq \|S(0)\| + \frac{1}{\Gamma(\beta)}\int_0^t (t-s)^{\beta-1}\|A(s,S(s))\|ds \\
&\leq \|S(0)\| + \frac{\|A(s,S(s))\|}{\Gamma(\beta)}\int_0^t (t-s)^{\beta-1}ds \\
&\leq \|S(0)\| + \mathscr{M}_{A1}(d+\|S\|)\left(\frac{\mathcal{T}^\beta}{\Gamma(\beta+1)}\right) \\
&\leq \|S(0)\| + \mathscr{M}_{A1}(d+x)\left(\frac{\mathcal{T}^\beta}{\Gamma(\beta+1)}\right) \\
&\leq \|S(0)\| + \mathscr{M}_{A1}(d+x)\left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi}\right). \qquad (58)
\end{aligned}
$$

Using a similar method, we obtained the following results

$$
\begin{aligned}
\|\varrho V\| &\leq \|V(0)\| + \mathscr{M}_{W1}(d+x)\left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi}\right), \\
\|\varrho E\| &\leq \|E(0)\| + \mathscr{M}_{U1}(d+x)\left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi}\right), \\
\|\varrho C\| &\leq \|C(0)\| + \mathscr{M}_{F1}(d+x)\left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi}\right), \\
\|\varrho I\| &\leq \|I(0)\| + \mathscr{M}_{H1}(d+x)\left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi}\right), \\
\|\varrho T\| &\leq \|T(0)\| + \mathscr{M}_{P1}(d+x)\left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi}\right), \\
\|\varrho R\| &\leq \|R(0)\| + \mathscr{M}_{B1}(d+x)\left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi}\right), \\
\|\varrho Q\| &\leq \|Q(0)\| + \mathscr{M}_{Z1}(d+x)\left(\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^\beta}{\beta\pi}\right), \qquad (59)
\end{aligned}
$$

Conversely, let $\hbar$ maps bounded set together with equal continuous sets in $\mathbb{H}^1([0,\mathcal{T}],\mathscr{X})$. if $0 \leq t_1 < t_2 \leq \mathcal{T}$, $S \in \Upsilon_S$, $V \in \Upsilon_V$, $E \in \Upsilon_E$, $C \in \Upsilon_C$, $I \in \Upsilon_I$, $T \in \Upsilon_T$, $R \in \Upsilon_R$, and $Q \in \Upsilon_Q$ where $t_1, t_2 \in [0,\mathcal{T}]$, then

$$
\begin{aligned}
\left\|\varrho S(t_1) - \varrho S(t_2)\right\| &= \frac{1}{\Gamma(\beta)}\left\|\int_0^{t_1}(t_1-s)^{\beta-1}A(s,S(s)) + \int_0^{t_2}(t_2-s)^{\beta-1}A(s,S(s))\right\|ds \\
&\leq \frac{1}{\Gamma(\beta)}\left\|\int_0^{t_1}\left((t_1-s)^{\beta-1} - (t_2-s)^{\beta-1}\right)A(s,S(s))ds\right\| \\
&\quad + \frac{1}{\Gamma(\beta)}\left\|\int_{t_1}^{t_2}(t_2-s)^{\beta-1}A(s,S(s))ds\right\| \\
&\leq \frac{\mathscr{M}_{A1}(d+x)}{\Gamma(\beta)}\left\|\int_0^{t_1}\left((t_1-s)^{\beta-1} - (t_2-s)^{\beta-1}\right)ds + \int_{t_1}^{t_2}(t_2-s)^{\beta-1}ds\right\| \\
&\leq \mathscr{M}_{A1}(d+x)\left(\frac{\mathcal{T}^\beta}{\Gamma(\beta+1)}\right)\left(t_1^\beta - t_2^\beta + 2\left(t_2-t_1\right)^\beta\right) \\
&\leq \left(\frac{\mathscr{M}_{A1}(d+x)\Gamma(1-\beta)\sin(\pi\beta)}{\beta\pi}\right)\left(t_1^\beta - t_2^\beta + 2\left(t_2-t_1\right)^\beta\right). \qquad (60)
\end{aligned}
$$

Using a similar process yields the following results

$$\begin{aligned}
\left\|\varrho V(t_1)-\varrho V(t_2)\right\| &\le \left(\frac{\mathscr{M}_{W1}(d+x)\Gamma(1-\beta)\sin(\pi\beta)}{\beta\pi}\right)\left(t_1^{\beta}-t_2^{\beta}+2\left(t_2-t_1\right)^{\beta}\right),\\
\left\|\varrho E(t_1)-\varrho E(t_2)\right\| &\le \left(\frac{\mathscr{M}_{U1}(d+x)\Gamma(1-\beta)\sin(\pi\beta)}{\beta\pi}\right)\left(t_1^{\beta}-t_2^{\beta}+2\left(t_2-t_1\right)^{\beta}\right),\\
\left\|\varrho C(t_1)-\varrho C(t_2)\right\| &\le \left(\frac{\mathscr{M}_{F1}(d+x)\Gamma(1-\beta)\sin(\pi\beta)}{\beta\pi}\right)\left(t_1^{\beta}-t_2^{\beta}+2\left(t_2-t_1\right)^{\beta}\right),\\
\left\|\varrho I(t_1)-\varrho I(t_2)\right\| &\le \left(\frac{\mathscr{M}_{H1}(d+x)\Gamma(1-\beta)\sin(\pi\beta)}{\beta\pi}\right)\left(t_1^{\beta}-t_2^{\beta}+2\left(t_2-t_1\right)^{\beta}\right),\\
\left\|\varrho T(t_1)-\varrho T(t_2)\right\| &\le \left(\frac{\mathscr{M}_{P1}(d+x)\Gamma(1-\beta)\sin(\pi\beta)}{\beta\pi}\right)\left(t_1^{\beta}-t_2^{\beta}+2\left(t_2-t_1\right)^{\beta}\right),\\
\left\|\varrho R(t_1)-\varrho R(t_2)\right\| &\le \left(\frac{\mathscr{M}_{B1}(d+x)\Gamma(1-\beta)\sin(\pi\beta)}{\beta\pi}\right)\left(t_1^{\beta}-t_2^{\beta}+2\left(t_2-t_1\right)^{\beta}\right),\\
\left\|\varrho Q(t_1)-\varrho Q(t_2)\right\| &\le \left(\frac{\mathscr{M}_{Z1}(d+x)\Gamma(1-\beta)\sin(\pi\beta)}{\beta\pi}\right)\left(t_1^{\beta}-t_2^{\beta}+2\left(t_2-t_1\right)^{\beta}\right).
\end{aligned} \tag{61}$$

The expression on the right-hand sides of the inequalities tends to zero as $t_1 \to t_2$. ϱ is a continuous function by Arzela–Ascoli theorem. Lastly, we demonstrate that

$$\mathfrak{R}(\varrho)=\left\{(S,V,E,C,I,T,R,Q)\in\mathbb{H}^1([0,\mathcal{T}],\mathscr{X}):(S,V,E,C,I,T,R,Q)=\Psi(S,V,E,C,I,T,R,Q)\right\}$$

is bounded for some $\Psi\in(0,1)$ using Lemma (1). Given that $(S,V,E,C,I,T,R,Q)\in\mathfrak{R}(\varrho)$, such that $(S,V,E,C,I,T,R,Q)=\Psi\varrho(S,V,E,C,I,T,R,Q)$ for each $t\in[0,\mathcal{T}]$ yields

$$\begin{aligned}
\|S(t)\| &\le S(0)+\frac{1}{\Gamma(\beta)}\int_0^t(t-s)^{\beta-1}\|A(s,S(s))\|ds\\
&\le S(0)+\frac{\mathscr{M}_{A1}}{\Gamma(\beta)}\int_0^t(t-s)^{\beta-1}(d+\|S(s)\|)ds\\
&\le S(0)+\frac{d\mathscr{M}_{A1}}{\Gamma(\beta)}\int_0^t(t-s)^{\beta-1}ds+\frac{\mathscr{M}_{A1}}{\Gamma(\beta)}\int_0^t(t-s)^{\beta-1}\|S(s)\|ds\\
&\le S(0)+\left(\mathscr{M}_{A1}\frac{\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^{\beta}}{\beta\pi}\right)+\left(\frac{\mathscr{M}_{A1}\Gamma(1-\beta)\sin(\pi\beta)}{\pi}\right)\int_0^t(t-s)^{\beta-1}\|S(s)\|ds\\
&\le \left(S(0)+\frac{\mathscr{M}_{A1}\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^{\beta}}{\beta\pi}\mathbb{E}_{\beta}\left(\mathscr{M}_{A1}\mathcal{T}^{\beta}\right)\right)<\infty.
\end{aligned} \tag{62}$$

Using a similar procedure, we obtained the following results

$$\begin{aligned}
\|V(t)\| &\le \left(V(0)+\frac{\mathscr{M}_{W1}\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^{\beta}}{\beta\pi}\mathbb{E}_{\beta}\left(\mathscr{M}_{W1}\mathcal{T}^{\beta}\right)\right)<\infty,\\
\|E(t)\| &\le \left(E(0)+\frac{\mathscr{M}_{U1}\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^{\beta}}{\beta\pi}\mathbb{E}_{\beta}\left(\mathscr{M}_{U1}\mathcal{T}^{\beta}\right)\right)<\infty,\\
\|C(t)\| &\le \left(C(0)+\frac{\mathscr{M}_{F1}\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^{\beta}}{\beta\pi}\mathbb{E}_{\beta}\left(\mathscr{M}_{F1}\mathcal{T}^{\beta}\right)\right)<\infty,\\
\|I(t)\| &\le \left(I(0)+\frac{\mathscr{M}_{H1}\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^{\beta}}{\beta\pi}\mathbb{E}_{\beta}\left(\mathscr{M}_{H1}\mathcal{T}^{\beta}\right)\right)<\infty,\\
\|T(t)\| &\le \left(T(0)+\frac{\mathscr{M}_{P1}\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^{\beta}}{\beta\pi}\mathbb{E}_{\beta}\left(\mathscr{M}_{P1}\mathcal{T}^{\beta}\right)\right)<\infty,\\
\|R(t)\| &\le \left(R(0)+\frac{\mathscr{M}_{B1}\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^{\beta}}{\beta\pi}\mathbb{E}_{\beta}\left(\mathscr{M}_{B1}\mathcal{T}^{\beta}\right)\right)<\infty,\\
\|Q(t)\| &\le \left(Q(0)+\frac{\mathscr{M}_{Z1}\Gamma(1-\beta)\sin(\pi\beta)\mathcal{T}^{\beta}}{\beta\pi}\mathbb{E}_{\beta}\left(\mathscr{M}_{Z1}\mathcal{T}^{\beta}\right)\right)<\infty.
\end{aligned} \tag{63}$$

$\mathfrak{R}(\varrho)$ is bounded as proven, and using Schaefer's fixed-point theorem, ϱ has a fixed point and hence the solution of the meningitis fractional-order model.

2.6 | Basic Reproduction Number

The basic reproduction number, denoted by $\mathscr{R}_0$, is a crucial threshold parameter that determines the potential for disease spread within a population. It is defined as the expected number of secondary cases of infection that will arise when a single infected individual is introduced into a completely susceptible population. The basic reproduction number for the meningitis fractional-order model (9–11) can be determined using the next-generation operator method, which is described in [52].

To calculate the basic reproduction number for the meningitis fractional-order model, it is essential to first determine the disease-free equilibrium of the model. The MM disease-free equilibrium (MMDFE) represents a steady state in which there is no infection of MM in the population. This is accomplished by setting the infected variables of the meningitis fractional-order model (9) (that is $E = C = I = T = Q = 0$) to zero and equating the right-hand side of the model to zero, then solving for the remaining variables. Consequently, the MMDFE is obtained as follows:

$$\mathcal{E}_0 = (S^*, V^*, E^*, C^*, I^*, T^*, R^*, Q^*) = \left(\frac{\Lambda(\psi+\gamma)}{\gamma(\theta+\psi+\mu)}, \frac{\Lambda\theta}{\gamma(\theta+\psi+\mu)}, 0, 0, 0, 0, 0, 0\right) \tag{64}$$

Subsequently, employing the next-generation operator method in [52], the nonnegative matrix $\mathcal{F}$, which represents the new infection, and the nonsingular matrix $\mathcal{V}$, which represents the remaining transition terms at the MMDFE, are given by

$$\mathcal{F} = \begin{bmatrix} (1-\varpi)\left(\frac{\alpha_h(C+I)}{N} + \frac{\alpha_e Q}{K+Q}\right)S \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \quad \textit{and} \quad \mathcal{V} = \begin{bmatrix} (\varphi+\gamma)E \\ -q\varphi E + (\eta_C+\sigma+\gamma)C \\ -(1-q)\varphi E - \sigma C + (\eta_I+\delta+\gamma)I \\ -\eta_C C - \eta_I I + (\phi+\varepsilon\delta+\gamma)T \\ -\tau_1 C - \tau_2 I + \xi Q \end{bmatrix}$$

$$\mathcal{F} = \begin{bmatrix} 0 & \frac{(1-\varpi)\alpha_h S^*}{S^*+V^*} & \frac{(1-\varpi)\alpha_h S^*}{S^*+V^*} & 0 & \frac{(1-\varpi)\alpha_e S^*}{K} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix},$$

and

$$\mathcal{V} = \begin{bmatrix} (\varphi+\gamma) & 0 & 0 & 0 & 0 \\ -q\varphi & (\eta_C+\sigma+\gamma) & 0 & 0 & 0 \\ -(1-q)\varphi & -\sigma & (\eta_I+\delta+\gamma) & 0 & 0 \\ 0 & -\eta_C & -\eta_I & (\phi+\varepsilon\delta+\gamma) & 0 \\ 0 & -\tau_1 & -\tau_2 & 0 & \xi \end{bmatrix}$$

Note that at disease-free equilibrium $N = S^* + V^*$. Thus, it follows from [52], that $\mathscr{R}_0 = \rho(FV^{-}1)$, where ρ is the largest eigenvalue or spectral radius of $(\mathcal{F}\mathcal{V})$. Hence, the MM basic reproduction number is given as

$$\begin{aligned} \mathscr{R}_0 = {} & \frac{(1-\varpi)\alpha_h(\psi+\gamma)\left(q\varphi\left(\sigma+\eta_I+\delta+\gamma\right)+(1-q)\varphi\left(\eta_C+\sigma+\gamma\right)\right)}{(\theta+\psi+\mu)(\varphi+\gamma)\left(\eta_C+\sigma+\gamma\right)\left(\eta_I+\delta+\gamma\right)} \\ & + \frac{(1-\varpi)\alpha_e\Lambda(\psi+\gamma)\left(\tau_1 q\varphi\left(\eta_I+\delta+\gamma\right)+\tau_2\left(q\varphi\sigma+(1-q)\varphi\left(\eta_C+\sigma+\gamma\right)\right)\right)}{K\gamma\xi(\theta+\psi+\mu)(\varphi+\gamma)\left(\eta_C+\sigma+\gamma\right)\left(\eta_I+\delta+\gamma\right)} \end{aligned} \tag{65}$$

2.7 | Local Stability of the Disease-Free Equilibrium

Theorem 4. *The MMDFE* $(\mathcal{E}_0)$ *is locally asymptotically stable if* $\mathcal{R}_0 < 1$, *and unstable whenever* $\mathcal{R}_0 > 1$.

Proof. We investigate the local asymptotic stability of the meningitis fractional-order model (9–11) by evaluating the Jacobian matrix of the meningitis fractional-order model system (9–11) at MMDFE, given by

$$\mathcal{J}(\mathcal{E}_0) = \begin{bmatrix} -X_1 & \psi & 0 & -\frac{(1-\varpi)\alpha_h S^*}{S^*+V^*} & -\frac{(1-\varpi)\alpha_h S^*}{S^*+V^*} & 0 & \mu & -\frac{(1-\varpi)\alpha_e S^*}{K} \\ \theta & -X_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -X_3 & \frac{(1-\varpi)\alpha_h S^*}{S^*+V^*} & \frac{(1-\varpi)\alpha_h S^*}{S^*+V^*} & 0 & 0 & \frac{(1-\varpi)\alpha_e S^*}{K} \\ 0 & 0 & q\varphi & -X_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & X_5 & \sigma & -X_6 & 0 & 0 & 0 \\ 0 & 0 & 0 & \eta_C & \eta_I & -X_7 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \phi & -X_8 & 0 \\ 0 & 0 & 0 & \tau_1 & \tau_2 & 0 & 0 & -\xi \end{bmatrix}$$

where $X_1 = \theta + \gamma$, $X_2 = \psi + \gamma$, $X_3 = \varphi + \gamma$, $X_4 = \eta_C + \sigma + \gamma$, $X_5 = (1-q)\varphi$, $X_6 = \eta_I + \delta + \gamma$, $X_7 = \phi + \varepsilon\delta + \gamma$, $X_8 = \mu + \gamma$.

The characteristic polynomial of the Jacobian matrix $\mathcal{J}(\mathcal{E}_0)$ is given by

$$\mathcal{G}(\lambda) = \lambda^8 + \mathcal{P}_1\lambda^7 + \mathcal{P}_2\lambda^6 + \mathcal{P}_3\lambda^5 + \mathcal{P}_4\lambda^4 + \mathcal{P}_5\lambda^3 + \mathcal{P}_6\lambda^2 + \mathcal{P}_7\lambda + \mathcal{P}_8 \tag{66}$$

where the expression for $\mathcal{P}_i$ (with $i = 1, 2, 3, \ldots, 8$) are in the Appendix section.

Utilizing the Routh-Hurwitz criterion [53, 54], which states that all the roots of the polynomial $\mathcal{G}(\lambda)$ in (66) are negative or have negative real part if $\mathcal{P}_i > 0$ (with $i = 1, 2, 3, \ldots, 8$). For the Routh-Hurwitz criterion to be satisfied, $\mathcal{R}_0$ must be less than one for $\mathcal{P}_8 > 0$. Hence, the disease-free equilibrium point $(\mathcal{E}_0)$ for the meningitis fractional-order model (9) is locally asymptotically stable if $\mathcal{R}_0 < 1$. □

2.8 | Generalized Ulam-Hyers-Russias Stability

In this section, we shall examine the stability of the meningitis fractional-order model (9–11) using the Ulam-Hyers-Rassias stability method as outlined in [55].

Definition 5. The meningitis fractional-order model (9–11) is generalized Ulam-Hyers-Rassias (UHR) stable with respect to $\lambda(t) \in \mathbb{H}^1([0,\mathcal{T}],\mathcal{X})$ if there exists a real value $\kappa_\epsilon > 0$ with $\epsilon > 0$ and for all solutions

$(S, V, E, C, I, T, R, Q) \in \mathbb{H}^1([0,\mathcal{T}],\mathcal{X})$ of the following inequalities

$$\begin{aligned} &|{}^c\mathcal{D}_t^\beta S(t) - A(t, S(t))| \le \lambda(t), \quad |{}^c\mathcal{D}_t^\beta V(t) - W(t, V(t))| \le \lambda(t), \\ &|{}^c\mathcal{D}_t^\beta E(t) - U(t, E(t))| \le \lambda(t), \quad |{}^c\mathcal{D}_t^\beta C(t) - F(t, C(t))| \le \lambda(t), \\ &|{}^c\mathcal{D}_t^\beta I(t) - H(t, I(t))| \le \lambda(t), \quad |{}^c\mathcal{D}_t^\beta T(t) - P(t, T(t))| \le \lambda(t), \\ &|{}^c\mathcal{D}_t^\beta R(t) - B(t, R(t))| \le \lambda(t), \quad |{}^c\mathcal{D}_t^\beta Q(t) - Z(t, Q(t))| \le \lambda(t), \end{aligned}$$

there exist a solution $(S, V, E, C, I, T, R, Q) \in \mathbb{H}^1([0,\mathcal{T}],\mathcal{X})$ of the model (9–11) with

$$\begin{aligned} &|S(t) - \overline{S}(t)| \le \kappa_\epsilon\lambda(t), \quad |V(t) - \overline{V}(t)| \le \kappa_\epsilon\lambda(t), \quad |E(t) - \bar{E}(t)| \le \kappa_\epsilon\lambda(t), \\ &|C(t) - \overline{C}(t)| \le \kappa_\epsilon\lambda(t), \quad |I(t) - \bar{I}(t)| \le \kappa_\epsilon\lambda(t), \quad |T(t) - \overline{T}(t)| \le \kappa_\epsilon\lambda(t), \\ &|R(t) - \overline{R}(t)| \le \kappa_\epsilon\lambda(t), \quad |Q(t) - \overline{Q}(t)| \le \kappa_\epsilon\lambda(t). \end{aligned}$$

Theorem 5. *The meningitis fractional-order model (9–11) is generalized Ulam-Hyers-Rassias stable with regards to* $\lambda \in \mathbb{H}^1([0,\mathcal{T}],\mathcal{X})$ *if* $\left(\mathcal{M}_A,\mathcal{M}_W,\mathcal{M}_U,\mathcal{M}_F,\mathcal{M}_H,\mathcal{M}_P,\mathcal{M}_B,\mathcal{M}_Z\right)\mathcal{T}^\beta < 1$.

Proof. From Definition (5), let λ denote a nondecreasing function of t, then there exist $\epsilon > 0$ such that

$$\int_0^t (t-s)^{\beta-1}\lambda(s)ds \le \epsilon\lambda(t), \tag{67}$$

for all $t \in [0,\mathcal{T}]$. It has already been proven that the functions A, W, U, F, H, P, B, Z are continuous and $\left(\mathcal{M}_A,\mathcal{M}_W,\mathcal{M}_U,\mathcal{M}_F,\mathcal{M}_H,\mathcal{M}_P,\mathcal{M}_B,\mathcal{M}_Z\right) > 0$ satisfies the Lipschitz condition as demonstrated earlier from theorem (2), the meningitis fractional-order model (9–11) has a unique solution

$$\overline{S}(t) = S(0) + \frac{1}{\Gamma(\beta)}\int_0^t (t-s)^{\beta-1}A\left(s,\overline{S}(s)\right)ds, \tag{68}$$

integrating the inequalities in Definition (5), we obtained

$$\begin{aligned}\left|S(t) - S(0) - \frac{1}{\Gamma(\beta)}\int_0^t (t-s)^{\beta-1}A(s,S(s))ds\right| &\le \frac{1}{\Gamma(\beta)}\int_0^t (t-s)^{\beta-1}\lambda(s)ds \\ &\le \frac{\epsilon\lambda(t)\mathcal{T}^\beta}{\Gamma(\beta+1)}.\end{aligned} \tag{69}$$

Utilizing Lemma (1) and Equation (69), we have

$$\begin{aligned}|S(t)-\overline{S}(t)| &\le \left|S(t) - \left(S(0) + \frac{1}{\Gamma(\beta)}\int_0^t (t-s)^{\beta-1}A\left(s,\overline{S}(s)\right)ds\right)\right| \\ &\le \left|S(t) - S(0) - \left(\frac{1}{\Gamma(\beta)}\int_0^t (t-s)^{\beta-1}A\left(s,\overline{S}(s)\right)ds\right.\right. \\ &\quad \left.\left. + \frac{1}{\Gamma(\beta)}\int_0^t (t-s)^{\beta-1}A(s,S(s))ds - \frac{1}{\Gamma(\beta)}\int_0^t (t-s)^{\beta-1}A(s,S(s))ds\right)\right| \\ &\le \left|S(t) - S(0) - \frac{1}{\Gamma(\beta)}\int_0^t (t-s)^{\beta-1}A(s,S(s))ds\right| \\ &\quad + \frac{1}{\Gamma(\beta)}\int_0^t (t-s)^{\beta-1}\left|A(s,S(s)) - A\left(s,\overline{S}(s)\right)\right|ds \\ &\le \frac{\epsilon\lambda(t)\mathcal{T}^\beta}{\Gamma(\beta+1)} + \frac{\mathcal{M}_A\mathcal{T}^\beta}{\Gamma(\beta+1)}\int_0^t (t-s)^{\beta-1}\left|S(t)-\overline{S}(t)\right|ds \\ &\le \frac{\epsilon\lambda(t)\mathcal{T}^\beta}{\Gamma(\beta+1)}\mathbb{E}_\beta\left(\mathcal{M}_A\mathcal{T}^\beta\right).\end{aligned} \tag{70}$$

By setting $\kappa_\beta = \frac{\epsilon\mathcal{T}^\beta}{\Gamma(\beta+1)}\mathbb{E}_\beta\left(\mathcal{M}_A\mathcal{T}^\beta\right)$, we get

$$\left|S(t)-\overline{S}(t)\right| \le \kappa_\beta\lambda(t), \quad t \in [0,\mathcal{T}]. \tag{71}$$

Employing a similar process, we obtained the following results

$$\begin{aligned}&\left|V(t)-\overline{V}(t)\right| \le \kappa_\beta\lambda(t), \quad \left|E(t)-\bar{E}(t)\right| \le \kappa_\beta\lambda(t), \quad \left|C(t)-\overline{C}(t)\right| \le \kappa_\beta\lambda(t), \\ &\left|I(t)-\bar{I}(t)\right| \le \kappa_\beta\lambda(t), \quad \left|T(t)-\overline{T}(t)\right| \le \kappa_\beta\lambda(t), \quad \left|R(t)-\overline{R}(t)\right| \le \kappa_\beta\lambda(t), \\ &\left|Q(t)-\overline{Q}(t)\right| \le \kappa_\beta\lambda(t), \quad t \in [0,\mathcal{T}].\end{aligned} \tag{72}$$

Therefore, this indicates that the meningitis fractional-order model (9–11) is Ulam-Hyers-Rassias stable with regards to $\lambda(t)$. □

3 | Result

3.1 | Numerical Scheme

Let $t_k = kh$, $k = 0, 1, 2, \ldots, m$ be the uniform grid points with some integer m, and $h = \frac{T}{m}$ which represent the grid step size. Consequently, Equation (9) is reduced to the fractional variant of the one-step Adam–Moulten method (corrector formula) which is achieved by utilizing the piece-wise interpolation with nodes and knots at t_j, $j = 0, 1, 2, \ldots, k+1$,

$$
\begin{aligned}
S(t_{k+1}) - S(0) &= \frac{h^\beta}{\Gamma(\beta+2)}\left(\sum_{j=0}^{k} u_{j,k+1} A(t_j, S(t_j)) + A(t_{k+1}, S^p(t_{k+1}))\right),\\
V(t_{k+1}) - V(0) &= \frac{h^\beta}{\Gamma(\beta+2)}\left(\sum_{j=0}^{k} u_{j,k+1} W(t_j, V(t_j)) + W(t_{k+1}, V^p(t_{k+1}))\right),\\
E(t_{k+1}) - E(0) &= \frac{h^\beta}{\Gamma(\beta+2)}\left(\sum_{j=0}^{k} u_{j,k+1} U(t_j, E(t_j)) + U(t_{k+1}, E^p(t_{k+1}))\right),\\
C(t_{k+1}) - C(0) &= \frac{h^\beta}{\Gamma(\beta+2)}\left(\sum_{j=0}^{k} u_{j,k+1} F(t_j, C(t_j)) + F(t_{k+1}, C^p(t_{k+1}))\right),\\
I(t_{k+1}) - I(0) &= \frac{h^\beta}{\Gamma(\beta+2)}\left(\sum_{j=0}^{k} u_{j,k+1} H(t_j, I(t_j)) + H(t_{k+1}, I^p(t_{k+1}))\right),\\
T(t_{k+1}) - T(0) &= \frac{h^\beta}{\Gamma(\beta+2)}\left(\sum_{j=0}^{k} u_{j,k+1} P(t_j, T(t_j)) + P(t_{k+1}, T^p(t_{k+1}))\right),\\
R(t_{k+1}) - R(0) &= \frac{h^\beta}{\Gamma(\beta+2)}\left(\sum_{j=0}^{k} u_{j,k+1} B(t_j, R(t_j)) + B(t_{k+1}, R^p(t_{k+1}))\right),\\
Q(t_{k+1}) - Q(0) &= \frac{h^\beta}{\Gamma(\beta+2)}\left(\sum_{j=0}^{k} u_{j,k+1} Z(t_j, Q(t_j)) + Z(t_{k+1}, Q^p(t_{k+1}))\right).
\end{aligned} \tag{73}
$$

From the one-step Adams–Bashforth method, the predictor formula is given as follows

$$
\begin{aligned}
S^p(t_{k+1}) - S(0) &= \frac{1}{\Gamma(\beta)}\sum_{j=0}^{k} v_{j,k+1} A(t_j, S(t_j)),\\
V^p(t_{k+1}) - V(0) &= \frac{1}{\Gamma(\beta)}\sum_{j=0}^{k} v_{j,k+1} W(t_j, V(t_j)),\\
E^p(t_{k+1}) - E(0) &= \frac{1}{\Gamma(\beta)}\sum_{j=0}^{k} v_{j,k+1} U(t_j, E(t_j)),\\
C^p(t_{k+1}) - C(0) &= \frac{1}{\Gamma(\beta)}\sum_{j=0}^{k} v_{j,k+1} F(t_j, C(t_j)),\\
I^p(t_{k+1}) - I(0) &= \frac{1}{\Gamma(\beta)}\sum_{j=0}^{k} v_{j,k+1} H(t_j, I(t_j)),\\
T^p(t_{k+1}) - T(0) &= \frac{1}{\Gamma(\beta)}\sum_{j=0}^{k} v_{j,k+1} P(t_j, T(t_j)),\\
R^p(t_{k+1}) - R(0) &= \frac{1}{\Gamma(\beta)}\sum_{j=0}^{k} v_{j,k+1} B(t_j, R(t_j)),\\
Q^p(t_{k+1}) - Q(0) &= \frac{1}{\Gamma(\beta)}\sum_{j=0}^{k} v_{j,k+1} Z(t_j, Q(t_j)),
\end{aligned} \tag{74}
$$

with the weight

$$\upsilon_{j,k+1} = \beta^{-1} h^{\beta} \left((k-j+1)^{\beta} - (k-j)^{\beta} \right) \tag{75}$$

3.2 | Data-Fitting and Parameter Estimation

In this section, we estimate the parameters of the fractional-order meningitis model by fitting it to the cumulative confirmed cases of cerebrospinal meningitis reported in Nigeria from epidemiological week 5 to week 19 of 2023. The dataset was obtained from the Nigeria Centre for Disease Control (NCDC) [56]. The model fitting procedure aligns the cumulative number of confirmed cases with the corresponding model variable representing the total number of treated symptomatic infected individuals. Parameter estimation was performed using the fmincon function from the MATLAB Optimization Toolbox [57, 58], which implements a constrained optimization algorithm. This approach employs the least squares method, a well-established and reliable technique for model fitting [58]. The goal is to minimize the discrepancy between the observed data points, Y_i, and the model-predicted values, X_i, by reducing the sum of squared errors (SSE). The SSE is mathematically expressed as

$$SSE = \sum_{i=1}^{n} \left(Y_1 - X_i \right)^2. \tag{76}$$

The initial conditions of the state variables used for the fitting are $S(0) = 200,000,000$, $V(0) = 945043$, $E(0) = 150,000$, $C(0) = 1200$, $I(0) = 300$, $T(0) = 50$, $R(0) = 0$, $Q(0) = 5000$, and the initial value of the total number of the treated symptomatic infected individuals is 21 to align with the observed data of the confirmed cerebrospinal cases of meningitis in Nigeria. The data for the cerebrospinal cases of meningitis in Nigeria are presented in Table 2, and the outcome of the fitting is depicted in Figure 2.

3.3 | Numerical Simulation

In this section, we present the numerical simulation of the fractional-order meningitis model using the fractional predictor-corrector approach. Specifically, the Adams–Bashforth linear multistep method in the Caputo sense was employed to derive the numerical scheme, ensuring convergence of the method. The initial conditions for the state variables used in the simulations are as follows: $S(0) = 200{,}000{,}000$, $V(0) = 945{,}043$, $E(0) = 150{,}000$, $C(0) = 1200$, $I(0) = 300$, $T(0) = 50$, $R(0) = 0$, $Q(0) = 5000$. The corresponding parameter values utilized in the model are summarized in Table 3.

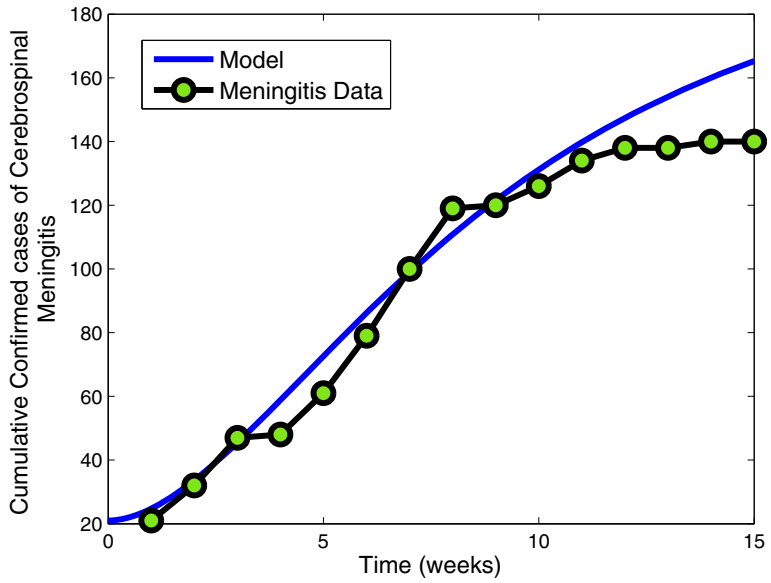

FIGURE 2 | Data-fitting of the Meningitis model to the cumulative confirmed cases of cerebrospinal meningitis.

TABLE 2 | Data on Cerebrospinal Meningitis cases in Nigeria, from week 5 to week 19, 2023.

Week	Suspected cases	Sample collected	Confirmed cases
5	—	68	21
6	—	38	11
7	—	35	15
8	—	30	1
9	—	29	13
10	—	31	18
11	—	52	21
12	—	43	19
13	212	37	1
14	213	14	6
15	219	30	8
16	210	18	4
17	158	27	0
18	113	5	2
19	83	1	0

TABLE 3 | Description of the model variables and parameters.

Parameter	Value	Source
Λ	0.03295	[59]
γ	0.01174	[60]
α_h	0.4310	Fitted
α_e	0.0741	Fitted
ϖ	0.849352	Fitted
φ	0.165843	Fitted
q	0.412506	Fitted
μ	0.005	Fitted
θ	0.85	Estimated from [61]
ψ	0.0188	Fitted
σ	0.878065	Fitted
δ	0.975056	Fitted
ϕ	0.005	Fitted
ε	0.97	Fitted
η_C	0.499756	Fitted
η_I	0.000529	Fitted
τ_1	0.452542	Fitted
τ_2	0.441587	Fitted
ξ	99148.798482	Fitted

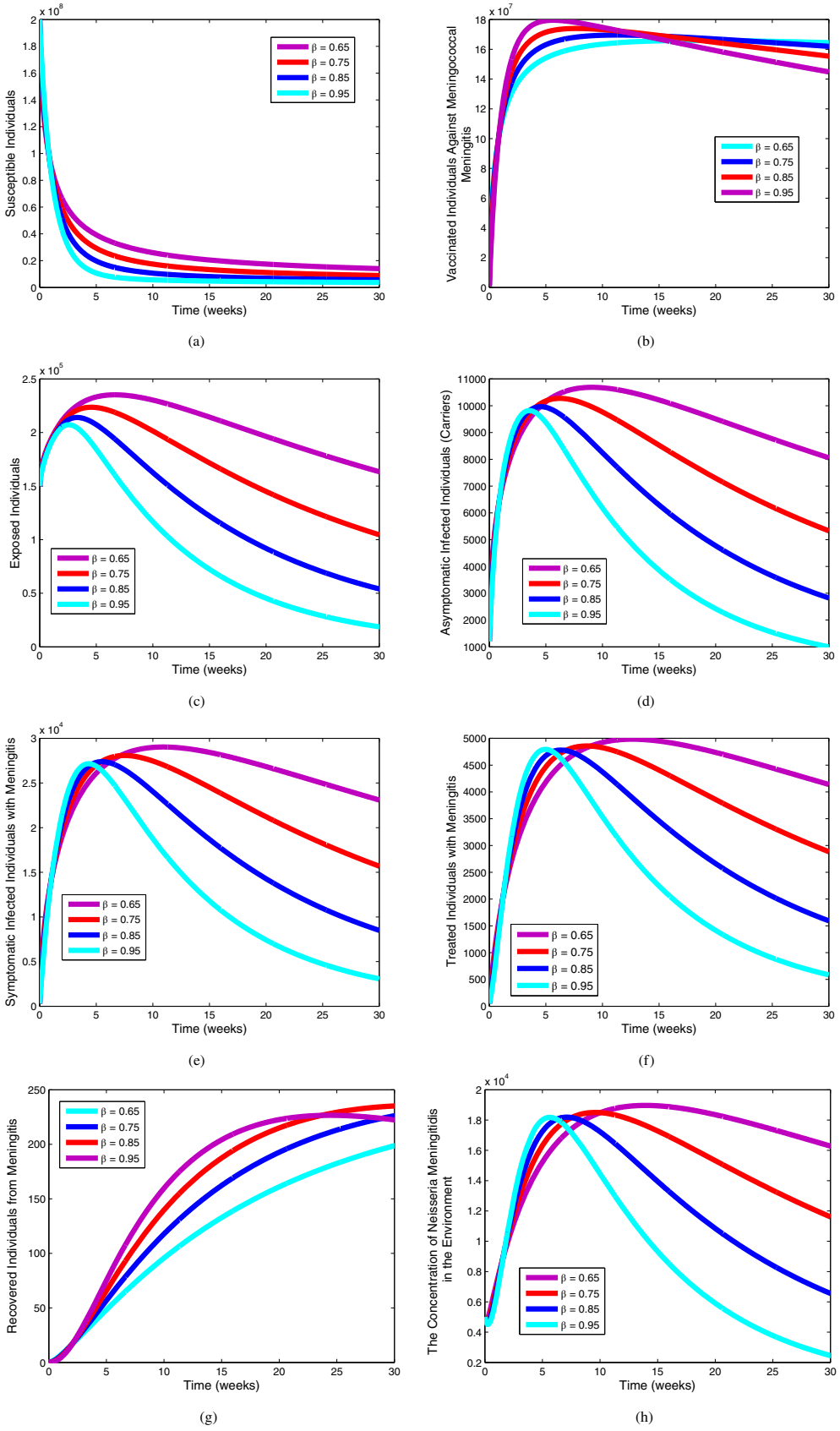

FIGURE 3 | Simulation of the human compartments and the compartment for the concentration of *Neisseria Meningitidis* in the environment for different values of the fractional order, $\beta = 0.65, 0.75, 0.85$, and 0.95. (a) susceptible individuals, (b) vaccinated individuals, (c) exposed individuals, (d) asymptomatic infected individuals (carriers), (e) symptomatic infected individuals, (f) treated individuals, (g) recovered individuals, (h) the concentration of *Neisseria Meningitidis* in the environment.

3.4 | Discussion of Results

Figure 3 represents the simulation of the human compartments and the compartment for the concentration of *Neisseria Meningitidis* in the environment for different values of the fractional order, $\beta = 0.65, 0.75, 0.85$, and 0.95. Figure 3a is the simulation of the total number of susceptible individuals against time. It is observed that the susceptible individuals decrease as time increases; furthermore, as the fractional order (β) increases, the number of susceptible individuals decreases. Figure 3b depicts the simulation of the population of vaccinated individuals against time. It is observed that from within week 1 to week 12, the number of the vaccination population increases as the fractional order increases. The reverse was the case from week 13 to week 30; it was observed that the number of the vaccinated population decreases as the fractional order increases. In Figure 3c–f, and h, it is observed that the fractional order rates is inversely proportional to the number of exposed individuals, asymptomatic infected individuals (Carriers), symptomatic infected individuals with MM, treated individuals, and the concentration of *Neisseria Meningitidis* in the environment. It is observed that an increase in the value of the fractional order results in a decrease in the total number of exposed individuals, asymptomatic infected individuals (Carriers), symptomatic infected individuals with MM, treated individuals, and the concentration of *Neisseria Meningitidis* in the environment. In Figure 3g, it is observed that as the fractional order increases, the number of recovered individuals increases. Particularly, in week 24, the number of recovered individuals for the highest fractional

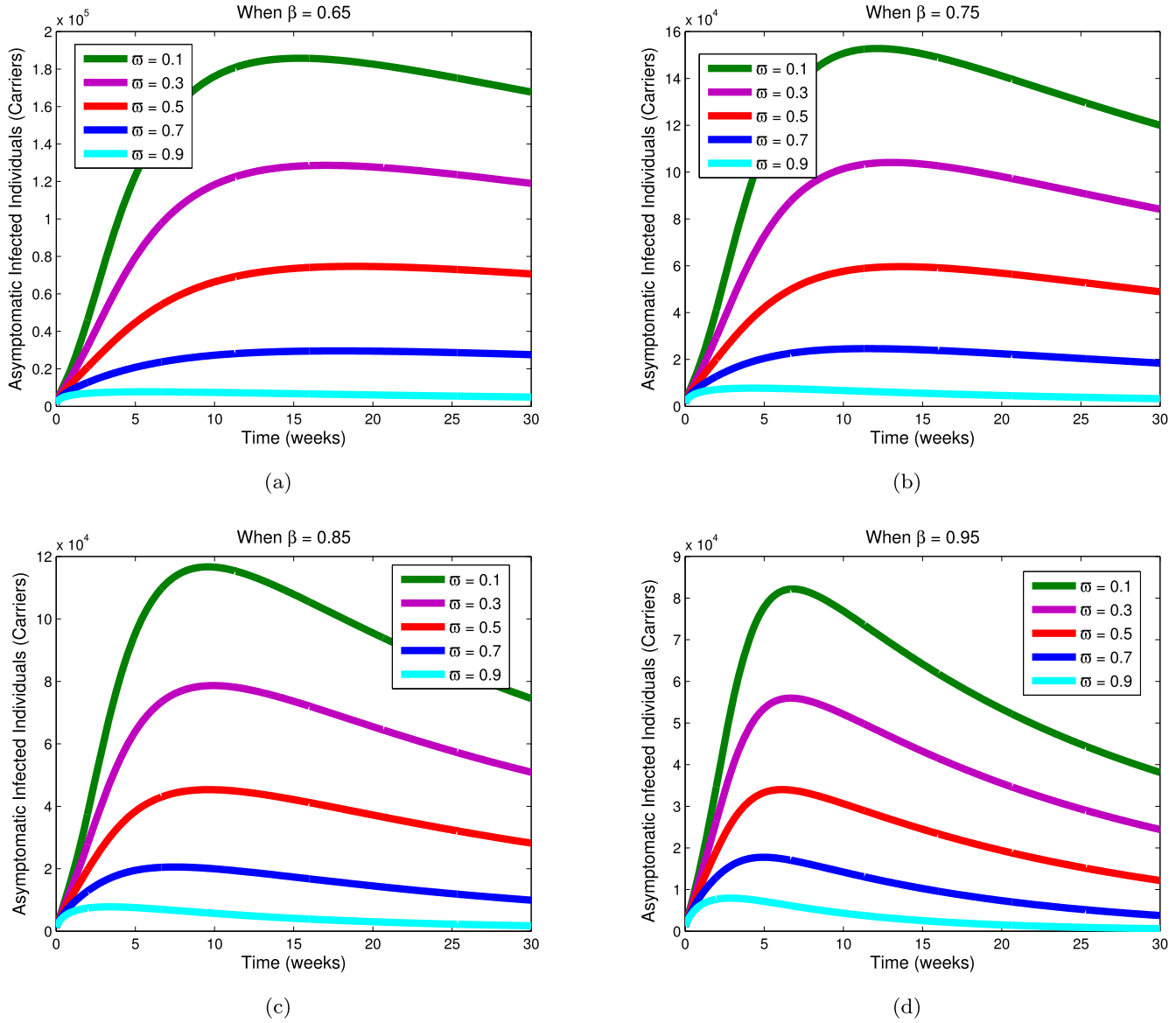

FIGURE 4 | Simulation of the effect of the hygiene consciousness rate (ϖ) on the asymptomatic infected population (Carriers) (a) for $\beta = 0.65$ (b) for $\beta = 0.75$ (c) for $\beta = 0.85$ (d) for $\beta = 0.95$.

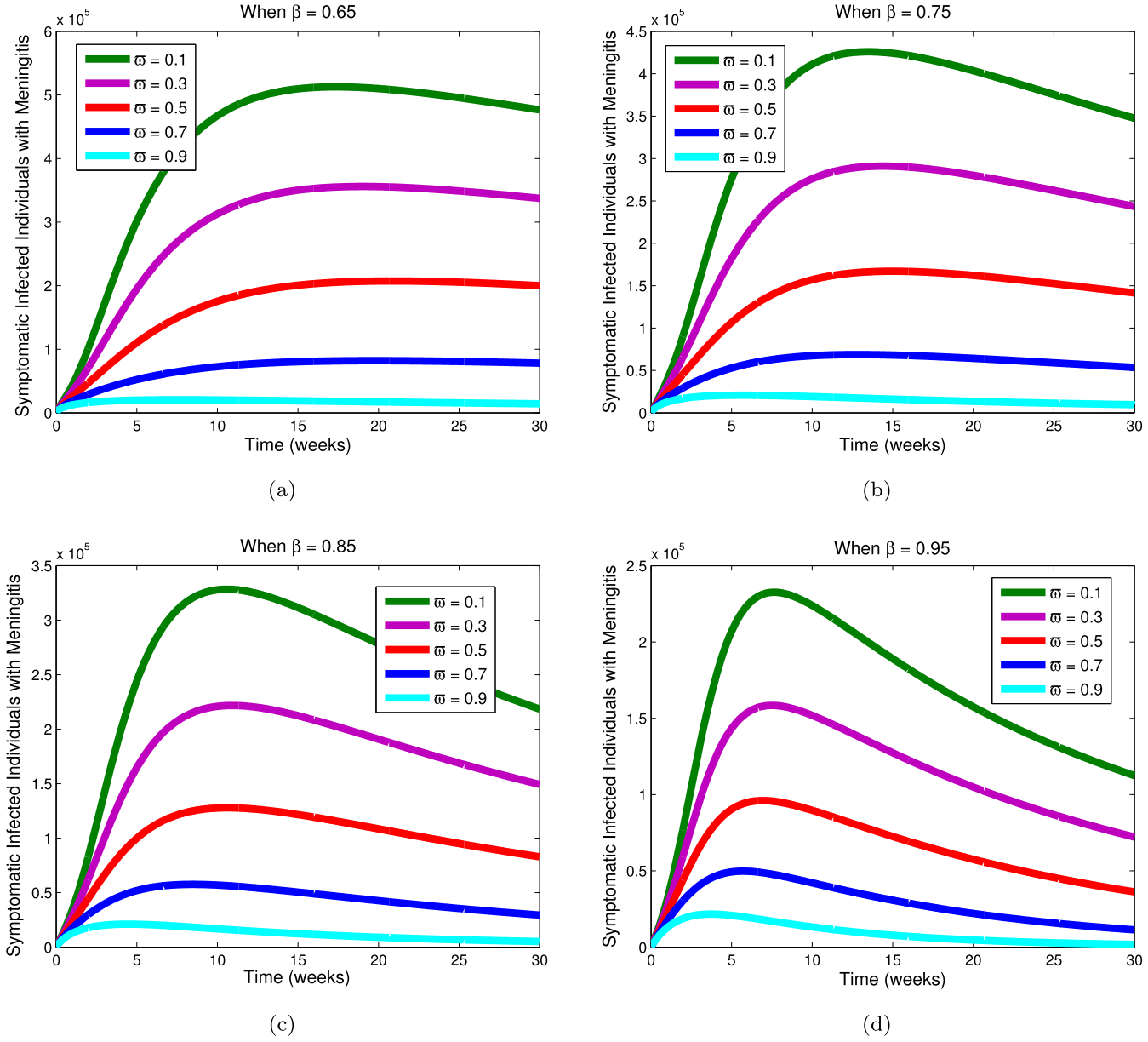

FIGURE 5 | Simulation of the effect of the hygiene consciousness rate (ϖ) on the symptomatic infected population (a) for $\beta = 0.65$ (b) for $\beta = 0.75$ (c) for $\beta = 0.85$ (d) for $\beta = 0.95$.

order (which is 0.95) declines while the number of recovered individuals increases for the other fractional orders (0.65, 0.75, 0.85).

Figure 4 is the effect of the hygiene consciousness rate (ϖ) on the asymptomatic infected population (Carriers) for different value of the fractional order, $\beta = 0.65, 0.75, 0.85$, and 0.95. In Figure 4a, it is observed that when $\beta = 0.65$ and the hygiene consciousness rate is stepped up to around 70%, the maximum number of asymptomatic infected individuals is around 28,000 within 30 weeks. It is observed in Figure 4b that when $\beta = 0.75$ and the hygiene consciousness rate is stepped up to around 70%, the number of asymptomatic infected individuals peaked at 22,000 within 10 weeks, and decreases to 20,000 within 30 weeks. In Figure 4c, when $\beta = 0.85$ and the hygiene consciousness rate is stepped up to around 70%, the number of asymptomatic infected individuals peaked at 20,000 in week 5–6, and decreases to around 10,000 within 30 weeks. In Figure 4d, when $\beta = 0.95$ and the hygiene consciousness rate is stepped up to around 70%, the maximum number of asymptomatic infected individuals is below 20,000 within 5 weeks, and decreases to around 4000 within 30 weeks. This result indicates that at an increased hygiene consciousness rate, the higher the fractional order rate, the lower the number of asymptomatic infected individuals.

Figure 5 represents the effect of the hygiene consciousness rate (ϖ) on the symptomatic infected population for different values of the fractional order, $\beta = 0.65, 0.75, 0.85$, and 0.95. It is observed in Figure 5a that when $\beta = 0.65$ and the hygiene consciousness rate is improved to 70%, the maximum number of the symptomatic infected individuals with meningitis is around 80,000 within 30 weeks. In Figure 5b, when $\beta = 0.75$ and the hygiene consciousness rate is increased to 70%, it is observed that the maximum number of symptomatic infected individuals is around 60,000 in 10 weeks, and decreases to 50,000 within 30 weeks. In Figure 5c, when $\beta = 0.85$ and the hygiene consciousness rate is increased to 70%, it is observed that the maximum number of symptomatic infected individuals is around 60,000 within 6 weeks, and decreases to 35,000 within 30 weeks. Figure 5d, when $\beta = 0.95$ and the hygiene consciousness rate is increased to 70%, it is observed that the maximum number of symptomatic infected individuals is around 50,000 in 5 weeks, and decreases to 20,000 within 30 weeks.

Figure 6 is the effect of the hygiene consciousness rate (ϖ) on the concentration of *Neisseria Meningitidis* in the environment for different values of the fractional order, $\beta = 0.65, 0.75, 0.85$, and 0.95. In Figure 6a, it is observed that when the hygiene consciousness rate is stepped up to 70% and $\beta = 0.65$, the maximum number of the concentration of *Neisseria*

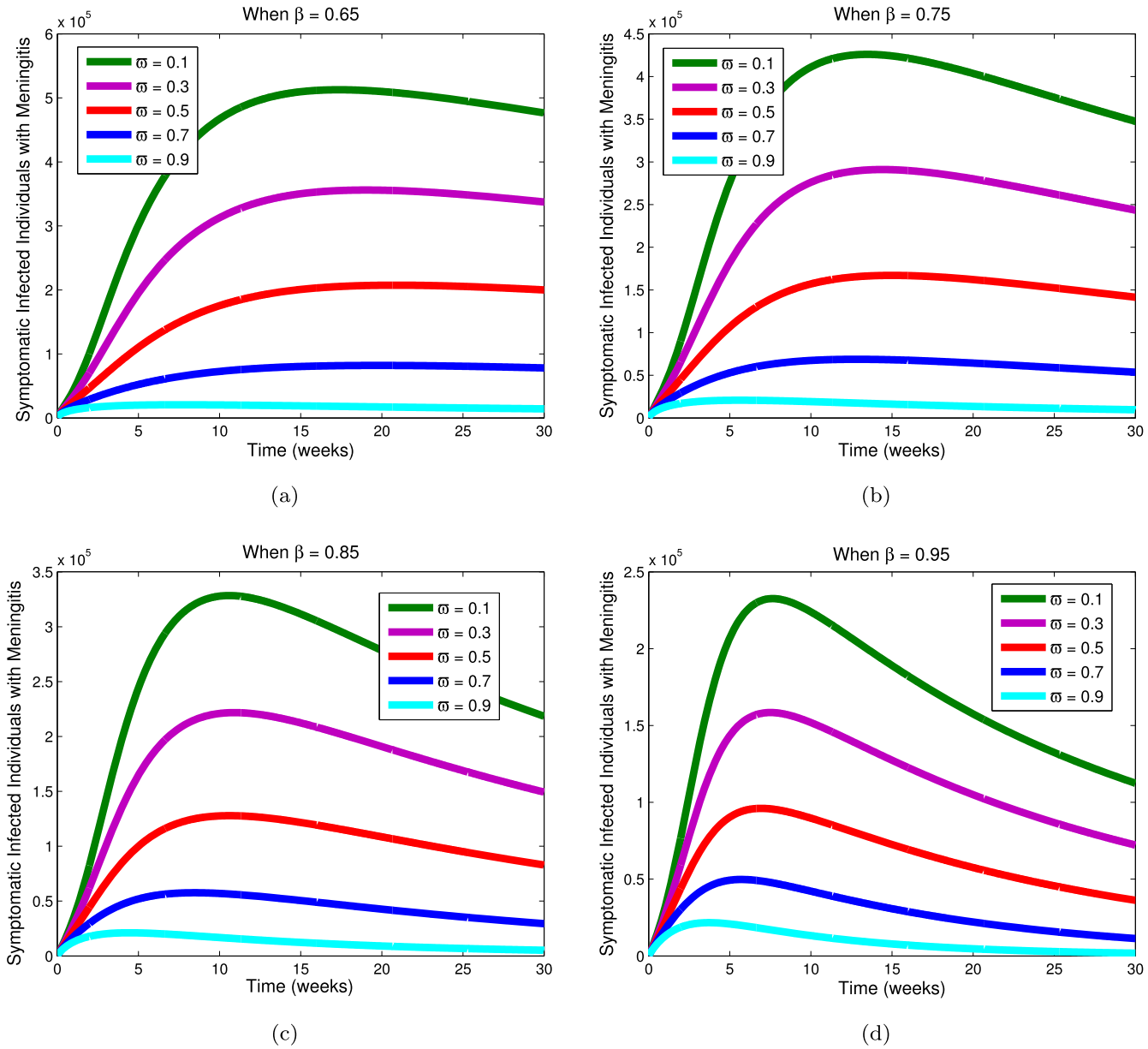

FIGURE 6 | Simulation of the effect of the hygiene consciousness rate (ϖ) on the concentration of *Neisseria Meningitidis* in the environment (a) for $\beta = 0.65$ (b) for $\beta = 0.75$ (c) for $\beta = 0.85$ (d) for $\beta = 0.95$.

Meningitidis in the environment is 50,000 within 30 weeks. In Figure 6b, it is observed that when the hygiene consciousness rate is stepped up to 70% and $\beta = 0.75$, the maximum number of the concentration of *Neisseria Meningitidis* in the environment is below 45,000 within 30 weeks. In Figure 6c, it is observed that when the hygiene consciousness rate is stepped up to 70% and $\beta = 0.85$, the highest number of the concentration of *Neisseria Meningitidis* in the environment is less than 40,000 in 30 weeks. In Figure 6d, when the hygiene consciousness rate is stepped up to 70% and $\beta = 0.95$, the highest number of the concentration of *Neisseria Meningitidis* in the environment is below 35,000 within 30 weeks. This result indicates that the fractional order is inversely proportional to the concentration of *Neisseria Meningitidis* in the environment as the hygiene consciousness rate increases.

Figure 7 represents the effect of the Vaccination rate (θ) on the symptomatic infected population for different values of the fractional order, $\beta = 0.65$, 0.75, 0.85, and 0.95. In Figure 7a, when the vaccination rate is increased to at least 80% and $\beta = 0.65$, it is observed that the number of symptomatic infected individuals peaked at around 30,000 in 10 weeks, and decreases to around 25,000 within 30 weeks. In Figure 7b, when the vaccination rate is increased to at least 80% and $\beta = 0.75$, it is observed that the maximum number of symptomatic infected individuals is around 30,000 within 8 weeks,

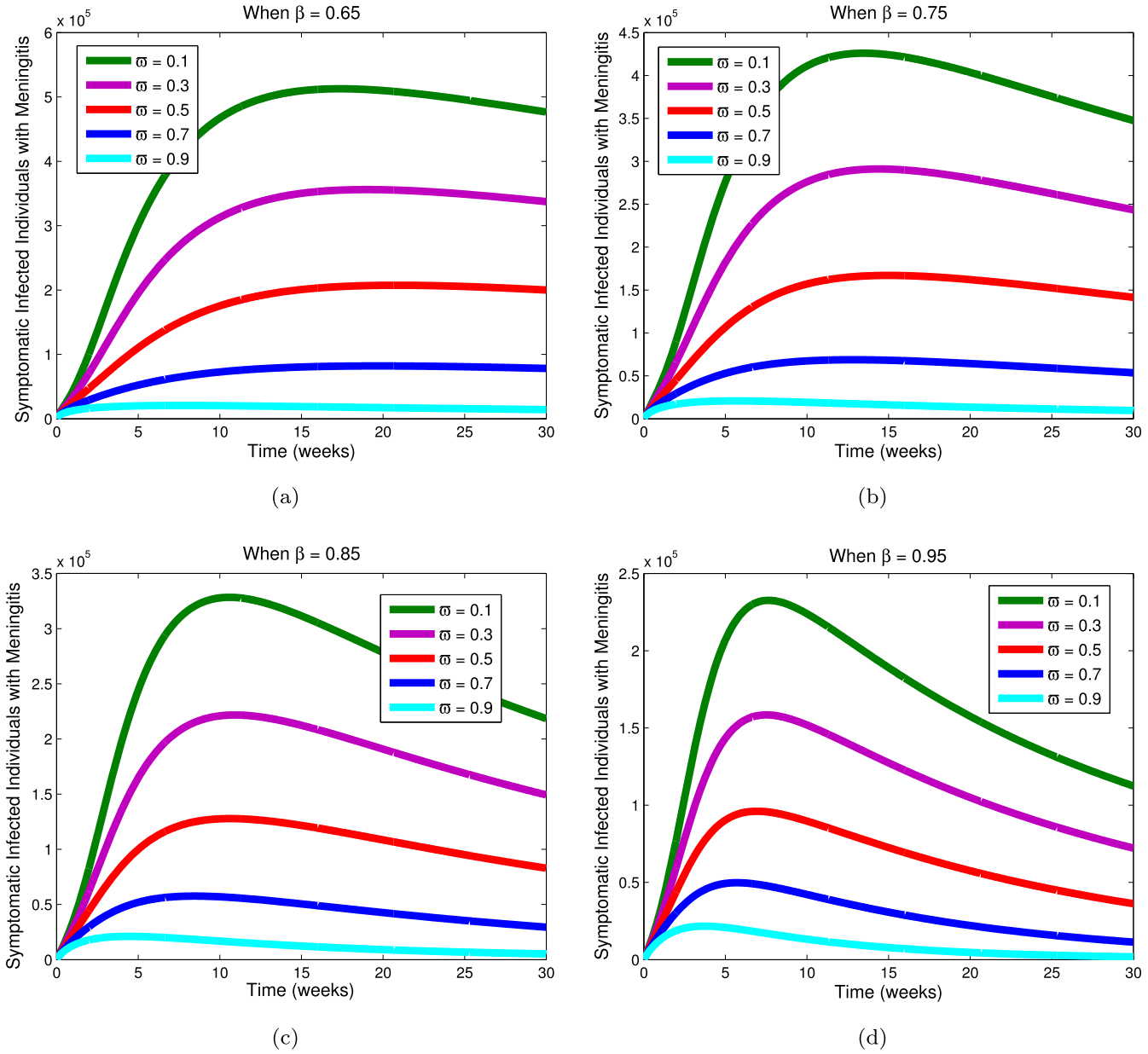

FIGURE 7 | Simulation of the effect of the Vaccination rate (θ) on the symptomatic infected population (a) for $\beta = 0.65$ (b) for $\beta = 0.75$ (c) for $\beta = 0.85$ (d) for $\beta = 0.95$.

and decreases to around 17,500 within 30 weeks. In Figure 7c, when the vaccination rate is increased to at least 80% and $\beta = 0.85$, it is observed that the maximum number of symptomatic infected individuals is around 29,000 within 6 weeks, and declines to around 9900 within 30 weeks. In Figure 7d, when the vaccination rate is increased to at least 80% and $\beta = 0.95$, it is observed that the number of symptomatic infected individuals peaked at around 28,000 within 5 weeks, and decreases to around 4000 within 30 weeks.

4 | Conclusion

In this study, we developed and thoroughly analyzed a fractional-order model to describe the transmission dynamics of MM, explicitly incorporating the environmental concentration of Neisseria meningitidis. The existence and uniqueness of solutions to the model were established using Banach's and Schauder's fixed-point theorems. The basic reproduction number, $\mathscr{R}_0$, was derived, and application of the Routh-Hurwitz criterion demonstrated that the disease-free equilibrium is locally asymptotically stable whenever $\mathscr{R}_0 < 1$. The stability of the model was further examined through the Ulam-Hyers-Rassias stability framework. To validate the model, we fitted it to cumulative confirmed cases of cerebrospinal meningitis in Nigeria, spanning from epidemiological week 5 to week 19 of 2023, using data obtained from the NCDC. The model showed a good fit to the observed data. Numerical simulations revealed an inverse relationship between the fractional derivative order and the number of asymptomatic carriers, symptomatic infected individuals, and the environmental concentration of Neisseria meningitidis. Specifically, increasing the order of the fractional derivative resulted in a decrease in both infection levels and bacterial concentration in the environment. Moreover, the results underscore the effectiveness of increasing vaccine uptake and promoting hygiene awareness among the susceptible population. These interventions significantly reduce the prevalence of MM and the associated environmental contamination, highlighting their importance in public health strategies aimed at controlling the disease.

Author Contributions

Festus Abiodun Oguntolu: writing – original draft, visualization, validation, supervision, software, resources, project administration, methodology, investigation, funding acquisition, formal analysis, data curation, conceptualization. **Olumuyiwa James Peter:** writing – original draft, visualization, validation, resources, project administration, investigation, funding acquisition, formal analysis, data curation. **Benjamin Idoko Omede:** visualization, validation, supervision, software, resources, project administration. **Ghaniyyat Bolanle Balogun:** writing – original draft, visualization, validation. **Zainab Olabisi Dere:** writing – original draft, visualization, validation. **Sania Qureshi:** writing – original draft, visualization, validation.

Funding

The authors received no specific funding for this work.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

In this paper, we propose a Caputo-based fractional-order derivative model for the transmission dynamics of meningococcal meningitis, incorporating the environmental concentration of Neisseria meningitidis as well as factors such as vaccination and the hygiene consciousness of susceptible individuals. The existence and uniqueness of solutions to the model are established using Banach's and Schauder's fixed-point theorems. Additionally, we compute the basic reproduction number and examine the local asymptotic stability of the disease-free equilibrium using the Routh-Hurwitz criterion. We analyze the stability of the fractional-order meningitis model using the Ulam-Hyers-Rassias stability method. Furthermore, we fit the model to the cumulative confirmed cases of cerebrospinal meningitis in Nigeria, using data obtained from the Nigeria Centre for Disease Control (NCDC), to validate the model. The model demonstrates a good fit with the reported cumulative cases.

References

1. M. A. Afolabi, K. S. Adewoye, A. I. Folorunso, and M. A. Omoloye, "A Mathematical Model on Transmission Dynamics of Meningococcal Meningitis," *Iconic Research and Engineering Journal* 4, no. 10 (2021): 59–66.

2. D. Van de Beek, C. Cabellos, O. Dzupova, et al., "ESCMID Guideline: Diagnosis and Treatment of Acute Bacterial Meningitis," *Clinical Microbiology and Infection* 22 (2016): S37–S62.

3. A. Purmohamad, E. Abasi, T. Azimi, et al., "Global Estimate of *Neisseria Meningitidis* Serogroups Proportion in Invasive Meningococcal Disease: A Systematic Review and Meta-Analysis," *Microbial Pathogenesis* 134 (2019): 103571.

4. A. Pormohammad, S. Lashkarbolouki, T. Azimi, et al., "Clinical Characteristics and Molecular Epidemiology of Children With Meningitis in Tehran, Iran: A Prospective Study," *New Microbes New Infections* 32 (2019): 100594.

5. V. L. Feigin, E. Nichols, T. Alam, et al., "Global, Regional, and National Burden of Neurological Disorders, 1990–2016: A Systematic Analysis for the Global Burden of Disease Study 2016," *Lancet Neurology* 18, no. 5 (2019): 459–480.

6. E. W. Weyori, B. B. Abubakari, B. Nkrumah, et al., "Predictive Signs and Symptoms of Bacterial Meningitis Isolates in Northern Ghana," *Scientific Reports* 13, no. 1 (2023): 13400.

7. K. A. Linder and P. N. Malani, "Meningococcal Meningitis," *JAMA* 321, no. 10 (2019): 1014.

8. K. J. Olbrich, D. Müller, S. Schumacher, E. Beck, K. Meszaros, and F. Koerber, "Systematic Review of Invasive Meningococcal Disease: Sequelae and Quality of Life Impact on Patients and Their Caregivers," *Infectious Diseases and Therapy* 7 (2018): 421–438.

9. D. S. Stephens, B. Greenwood, and P. Brandtzaeg, "Epidemic Meningitis, Meningococcaemia, and *Neisseria Meningitidis*," *Lancet* 369, no. 9580 (2007): 2196–2210.

10. R. Joshi and S. D. Saroj, "Survival and Evasion of *Neisseria Meningitidis* From Macrophages," *Medicine in Microecology* 17 (2023): 100087.

11. V. I. Caracoti, C. Ş. Caracoti, D. L. Ancuţa, et al., "Developing a Novel Murine Meningococcal Meningitis Model Using a Capsule-Null Bacterial Strain," *Diagnostics* 14, no. 11 (2024): 1116.

12. S. Yadav and G. Rammohan, "Meningococcal Meningitis," in *StatPearls [Internet]* (StatPearls Publishing, 2022).

13. J. M. Collard, B. Issaka, M. Zaneidou, et al., "Epidemiological Changes in Meningococcal Meningitis in Niger From 2008 to 2011 and the Impact of Vaccination," *BMC Infectious Diseases* 13 (2013): 1–10.

14. E. H. Ahmed-Abakur, "Meningococcal Meningitis: Etiology, Diagnosis, Epidemiology and Treatment," *American Journal of the Medical Sciences* 4, no. 6 (2014): 266–271.

15. A. Cohn and J. MacNeil, "The Changing Epidemiology of Meningococcal Disease," *Infectious Disease Clinics of North America* 29, no. 4 (2015): 667–677.

16. S. R. Parikh, H. Campbell, J. A. Bettinger, et al., "The Everchanging Epidemiology of Meningococcal Disease Worldwide and the Potential for Prevention Through Vaccination," *Journal of Infection* 81, no. 4 (2020): 483–498.

17. T. Al-Sanouri, S. Mahdi, I. A. Khader, et al., "The Epidemiology of Meningococcal Meningitis: Multicenter, Hospital-Based Surveillance of Meningococcal Meningitis in Iraq," *IJID Regions* 1 (2021): 100–106.

18. W. Szymański, K. Simon, and M. Rorat, "Differences in the Courses of Meningococcal and Pneumococcal Cerebrospinal Meningitis," *Neurologia i Neurochirurgia Polska* 54, no. 1 (2020): 39–46.

19. N. Shaheen, A. Mohamed, Y. Soliman, et al., "Up-To-Date Review of Meningococcal Meningitis: Global Challenges and Recommendations," *Dubai Medical Journal* 6, no. 1 (2023): 1–13.

20. M. Pizza and R. Rappuoli, "*Neisseria meningitidis*: Pathogenesis and Immunity," *Current Opinion in Microbiology* 23 (2015): 68–72.

21. S. I. Pelton, "The Global Evolution of Meningococcal Epidemiology Following the Introduction of Meningococcal Vaccines," *Journal of Adolescent Health* 59, no. 2 (2016): S3-S11.

22. C. W. Chukwu, J. Mushanyu, and M. L. Juga, "A Mathematical Model for Co-Dynamics of Listeriosis and Bacterial Meningitis Diseases," *Communications in Mathematical Biology and Neuroscience* 2020 (2020): 1–20.

23. M. F. Martínez, E. G. Merino, E. G. Sánchez, J. G. Sánchez, A. M. del Rey, and G. R. Sánchez, "A Mathematical Model to Study the Meningococcal Meningitis," *Procedia Computer Science* 18 (2013): 2492–2495.

24. T. Aminu and O. Bamigbola, "A New Meningitis Mathematical Model and Its Dynamics," *International Journal of Science Research and Technology* 7, no 9 (2025): https://doi.org/10.70382/tijsrat.v07i9.021.

25. K. Maseno, "Mathematical Model for Malaria and Meningitis Co-Infection Among Children," *Applied Mathematical Sciences* 5, no. 47 (2011): 2337–2359.

26. M. A. Belay, O. J. Abonyo, and D. M. Theuri, "Mathematical Model Analysis for the Transmission Dynamics of Bacterial Meningitis Disease Incorporating Drug-Resistance Class," *Communications in Mathematical Biology and Neuroscience* 2022 (2022): 1–30.

27. K. G. Varshney and Y. K. Dwivedi, "Mathematical Modelling of Influenza-Meningitis Under the Quarantine Effect of Influenza," *Turkish Journal of Computer and Mathematics Education* 12, no. 11 (2021): 7214–7225.

28. C. Türkün, M. Gölgeli, and F. M. Atay, "A Mathematical Interpretation for Outbreaks of Bacterial Meningitis Under the Effect of Time-Dependent Transmission Parameters," *Nonlinear Dynamics* 111, no. 15 (2023): 14467–14484.

29. S. S. Musa, S. Zhao, N. Hussaini, A. G. Habib, and D. He, "Mathematical Modeling and Analysis of Meningococcal Meningitis Transmission Dynamics," *International Journal of Biomathematics* 13, no. 1 (2020): 2050006.

30. K. B. Blyuss, "Mathematical Modelling of the Dynamics of Meningococcal Meningitis in Africa," *UK Success Stories in Industrial Mathematics* (2016): 221–226, https://doi.org/10.1007/978-3-319-25454-8_28.

31. M. V. Crankson, O. Olotu, A. S. Afolabi, and A. Abidemi, "Modeling the Vaccination Control of Bacterial Meningitis Transmission Dynamics: A Case Study," *Mathematical Modelling and Control* 3, no. 4 (2023): 416–434.

32. J. K. K. Asamoah, F. Nyabadza, B. Seidu, M. Chand, and H. Dutta, "Mathematical Modelling of Bacterial Meningitis Transmission Dynamics With Control Measures," *Computational and Mathematical Methods in Medicine* 2018, no. 1 (2018): 2657461.

33. I. M. ELmojtaba and S. O. Adam, "A Mathematical Model for Meningitis Disease," *Red Sea University Journal of Basic and Applied Science* 2, no. 2 (2017): 467–472.

34. M. I. Amar, M. R. Nisardi, and M. F. Nurahmad, "A Deterministic Mathematical Model of Meningitis Transmission Dynamics With Vaccination and Screening," *Journal Matematika UNAND* 14, no. 1 (2025): 14–30.

35. T. K. Yano and J. Bitok, "Computational Modelling of Two Strain Meningitis Disease Outbreak," *Applied and Computational Mathematics* 11, no. 5 (2022): 123–129.

36. B. S. Kotola and T. T. Mekonnen, "Mathematical Model Analysis and Numerical Simulation for Codynamics of Meningitis and Pneumonia Infection With Intervention," *Scientific Reports* 12, no. 1 (2022): 2639.

37. H. M. Ali, I. G. Ameen, and Y. A. Gaber, "The Effect of Curative and Preventive Optimal Control Measures on a Fractional Order Plant Disease Model," *Mathematics and Computers in Simulation* 220 (2024): 496–515, https://doi.org/10.1016/j.matcom.2024.02.009.

38. A. E. Matouk, I. G. Ameen, and Y. A. Gaber, "Analyzing the Dynamics of Fractional Spatio-Temporal SEIR Epidemic Model," *AIMS Mathematics* 9, no. 11 (2024): 30838–30863, https://doi.org/10.3934/math.20241489.

39. A. El-Sayed, Z. A. Rida, and Y. Gaber, "On the Stability Analysis and Solutions of Fractional Order Pine Wilt Disease Model," *Applied Mathematics Information Sciences* 14, no. 6 (2020): 20, https://doi.org/10.18576/amis/140620.

40. K. S. Nisar, M. Farman, M. Abdel-Aty, and J. Cao, "A Review on Epidemic Models in Sight of Fractional Calculus," *Alexandria Engineering Journal* 75 (2023): 81–113.

41. K. S. Nisar, M. Farman, M. Abdel-Aty, and C. Ravichandran, "A Review of Fractional Order Epidemic Models for Life Sciences Problems: Past, Present and Future," *Alexandria Engineering Journal* 95 (2024): 283–305.

42. D. Baleanu, M. H. Abadi, A. Jajarmi, K. Z. Vahid, and J. J. Nieto, "A New Comparative Study on the General Fractional Model of COVID-19 With Isolation and Quarantine Effects," *Alexandria Engineering Journal* 61, no. 6 (2022): 4779–4791.

43. A. Omame, U. K. Nwajeri, M. Abbas, and C. P. Onyenegecha, "A Fractional Order Control Model for Diabetes and COVID-19 Co-Dynamics With Mittag-Leffler Function," *Alexandria Engineering Journal* 61, no. 10 (2022): 7619–7635.

44. U. B. Odionyenma, N. Ikenna, and B. Bolaji, "Analysis of a Model to Control the Co-Dynamics of Chlamydia and Gonorrhea Using Caputo Fractional Derivative," *Mathematical Modelling and Numerical Simulation With Applications* 3, no. 2 (2023): 111–140.

45. M. A. Khan and H. P. Odinsyah, "Fractional Model of HIV Transmission With Awareness Effect," *Chaos, Solitons & Fractals* 138 (2020): 109967.

46. A. Elsonbaty, T. M. Al-shami, and A. El-Mesady, "Unveiling the Dynamics of Meningitis Infections: A Comprehensive Study of a Novel Fractional-Order Model With Optimal Control Strategies," *Boundary Value Problems* 1 (2025): 48.

47. O. J. Peter, A. Yusuf, M. M. Ojo, S. Kumar, N. Kumari, and F. A. Oguntolu, "A Mathematical Model Analysis of Meningitis With Treatment and Vaccination in Fractional Derivatives," *International Journal of Applied and Computational Mathematics* 8, no. 3 (2022): 117.

48. S. Rashid, B. Kanwal, A. G. Ahmad, E. Bonyah, and S. K. Elagan, "Novel Numerical Estimates of the Pneumonia and Meningitis Epidemic Model via the Nonsingular Kernel With Optimal Analysis," *Complexity* 2022, no. 1 (2022): 4717663.

49. A. Carpinteri and F. Mainardi, *Fractals and Fractional Calculus in Continuum Mechanics*, vol. 378 (Springer, 2014).

50. U. K. Nwajeri, A. Omame, and C. P. Onyenegecha, "Analysis of a Fractional Order Model for HPV and CT Co-Infection," *Results in Physics* 28 (2021): 104643.

51. N. Sene, "SIR Epidemic Model With Mittag–Leffler Fractional Derivative," *Chaos, Solitons & Fractals* 137 (2020): 109833.

52. P. Van den Driessche and J. Watmough, "Reproduction Numbers and Sub-Threshold Endemic Equilibria for Compartmental Models of Disease Transmission," *Mathematical Biosciences* 180, no. 1–2 (2002): 29–48.

53. Nigeria Centre for Disease Control (NCDC). An Update of Meningitis Outbreak in Nigeria: Situation Reports, (2024), https://ncdc.gov.ng/diseases/sitreps/.

54. B. I. Omede, S. A. Jose, J. Anuwat, and T. Park, "Mathematical Analysis on the Transmission Dynamics of Delta and Omicron Variants of COVID-19 in the United States," *Modeling Earth Systems and Environment* 10 (2024): 1–38.

55. K. Liu, M. Fečkan, and J. Wang, "Hyers–Ulam Stability and Existence of Solutions to the Generalized Liouville–Caputo Fractional Differential Equations," *Symmetry* 12, no. 6 (2020): 955.

56. Nigeria Center for Disease Control (NCDC), https://ncdc.gov.ng/diseases/sitreps/?cat=6name=AnUpdateofMeningitisOutbreakinNigeria.

57. J. McCall, "Genetic Algorithms for Modelling and Optimisation," *Journal of Computational and Applied Mathematics* 184, no. 1 (2005): 205–222.

58. F. A. Oguntolu, O. J. Peter, B. I. Omede, G. B. Balogun, and T. A. Ayoola, "Mathematical Model on the Transmission Dynamics of Leptospirosis in Human and Animal Population With Optimal Control Strategies Using Real Statistical Data," *Quality & Quantity* 59 (2024): 1–40.

59. "Nigeria - Birth Rate, Crude".

60. "Nigeria - Death Rate, Crude".

61. "How a New Vaccine Stamped Out a Meningitis Flare-up in Nigeria's Jigawa State".

Appendix A

$$
\begin{aligned}
\mathcal{P}_1 &= X_1 + X_2 + X_3 + X_4 + X_6 + X_7 + X_8 + \xi,\\
\mathcal{P}_2 &= X_1\left(X_2 + X_3 + X_4 + X_6 + X_7 + X_8\right) + X_2\left(X_3 + X_4 + X_6 + X_7 + X_8\right) + X_3\left(X_4 + X_6 + X_7 + X_8\right)\\
&\quad + X_4\left(X_6 + X_7 + X_8\right) + X_6\left(X_7 + X_8\right) + \xi\left(X_1 + X_2 + X_3 + X_4 + X_6 + X_7 + X_8\right) - \psi\theta\\
&\quad - \frac{(1-\varpi)\alpha_h X_2^2\left(q\varphi + X_5\right)}{(\theta+\psi+\mu)},\\
\mathcal{P}_3 &= \xi X_1\left(X_2 + X_3 + X_4 + X_6 + X_7 + X_8\right) + X_2\left(\xi + X_1\right)\left(X_3 + X_4 + X_6 + X_7 + X_8\right)\\
&\quad + X_3\left(\xi + X_1 + X_2\right)\left(X_4 + X_6 + X_7 + X_8\right) + X_4\left(\xi + X_1 + X_2 + X_3\right)\left(X_6 + X_7 + X_8\right)\\
&\quad + X_6\left(\xi + X_1 + X_2 + X_3 + X_4\right)\left(X_7 + X_8\right) + X_7X_8\left(\xi + X_1 + X_2 + X_3 + X_4 + X_6\right) - \frac{(1-\varpi)\alpha_h\xi X_2X_5}{(\theta+\psi+\mu)}\\
&\quad - \frac{(1-\varpi)\alpha_h q\varphi X_2\left(\sigma + \xi + X_1 + X_2 + X_6 + X_7 + X_8\right)}{(\theta+\psi+\mu)} - \frac{(1-\varpi)\alpha_h X_2X_5\left(X_1 + X_2 + X_4 + X_7 + X_8\right)}{(\theta+\psi+\mu)}\\
&\quad - \psi\theta\left(\xi + X_3 + X_4 + X_6 + X_7 + X_8\right) - \frac{(1-\varpi)\alpha_e\Lambda X_2\left(\tau_1 q\varphi + \tau_2 X_5\right)}{K\gamma(\theta+\psi+\mu)},\\
\mathcal{P}_4 &= \xi X_1X_2\left(X_3 + X_4 + X_6 + X_7 + X_8\right) + X_3\left(\xi\left(X_1 + X_2\right) + X_1X_2\right)\left(X_4 + X_6 + X_7 + X_8\right)\\
&\quad + X_4\left(\xi\left(X_1 + X_2 + X_3\right) + X_1\left(X_2 + X_3\right) + X_2X_3\right)\left(X_6 + X_7 + X_8\right) + \xi X_6\left(X_1 + X_2 + X_3 + X_4\right)\left(X_7 + X_8\right)\\
&\quad + X_6\left(X_1\left(X_2 + X_3 + X_4\right) + X_2\left(X_3 + X_4\right) + X_3X_4\right)\left(X_7 + X_8\right) + \xi X_7X_8\left(X_1 + X_2 + X_3 + X_4 + X_6\right)\\
&\quad + X_7X_8\left(X_1\left(X_2 + X_3 + X_4 + X_6\right) + X_2\left(X_3 + X_4 + X_6\right) + X_3\left(X_4 + X_6\right) + X_4X_6\right) + \frac{(1-\varpi)\alpha_h q\varphi\psi\theta X_2}{(\theta+\psi+\mu)}\\
&\quad - \frac{(1-\varpi)\alpha_h q\varphi\sigma X_2\left(\xi + X_1 + X_2 + X_7 + X_8\right)}{(\theta+\psi+\mu)} - \frac{(1-\varpi)\alpha_h q\varphi\xi X_2\left(X_1 + X_2 + X_6 + X_7 + X_8\right)}{(\theta+\psi+\mu)}\\
&\quad - \frac{(1-\varpi)\alpha_h q\varphi X_2\left(X_2 + X_6 + X_7 + X_8\right)}{(\theta+\psi+\mu)} - \frac{(1-\varpi)\alpha_h q\varphi\xi X_2\left(X_1 + X_2 + X_6 + X_7 + X_8\right)}{(\theta+\psi+\mu)}\\
&\quad - \frac{(1-\varpi)\alpha_h q\varphi X_2\left(X_6 + X_7 + X_8\right)}{(\theta+\psi+\mu)} - \frac{(1-\varpi)\alpha_h X_2\left(q\varphi X_6 + X_4X_5\right)\left(X_7 + X_8\right)}{(\theta+\psi+\mu)} - \frac{(1-\varpi)\alpha_e\Lambda\tau_2 q\varphi\sigma X_2}{K\gamma(\theta+\psi+\mu)}\\
&\quad - \frac{(1-\varpi)\alpha_h X_2X_7X_8\left(q\varphi + X_5\right)}{(\theta+\psi+\mu)} - \frac{(1-\varpi)\alpha_e\Lambda\tau_1 q\varphi X_2\left(X_1 + X_2 + X_6 + X_7 + X_8\right)}{K\gamma(\theta+\psi+\mu)}\\
&\quad - \xi\psi\theta\left(X_3 + X_4 + X_6 + X_7 + X_8\right) - \psi\theta X_3\left(X_4 + X_6 + X_7 + X_8\right) - \psi\theta X_4\left(X_6 + X_7 + X_8\right)\\
&\quad - \psi\theta X_6\left(X_7 + X_8\right) - \psi\theta X_7X_8 + \frac{(1-\varpi)\alpha_h\psi\theta X_2X_5}{(\theta+\psi+\mu)} - \frac{(1-\varpi)\alpha_e\Lambda\tau_2 X_2X_5\left(X_1 + X_2 + X_4 + X_7 + X_8\right)}{K\gamma(\theta+\psi+\mu)}\\
&\quad - \frac{(1-\varpi)\alpha_h\xi X_2X_5\left(X_1 + X_2 + X_4 + X_7 + X_8\right)}{(\theta+\psi+\mu)} - \frac{(1-\varpi)\alpha_h X_1X_2X_5\left(X_2 + X_4 + X_7 + X_8\right)}{(\theta+\psi+\mu)}\\
&\quad - \frac{(1-\varpi)\alpha_h X_2^2X_5\left(X_4 + X_7 + X_8\right)}{(\theta+\psi+\mu)},\\
\mathcal{P}_5 &= \xi X_1X_2X_3\left(X_4 + X_6 + X_7 + X_8\right) + X_4\left(\xi\left(X_1X_2 + X_1X_3 + X_2X_3\right) + X_1X_2X_3\right)\left(X_6 + X_7 + X_8\right)\\
&\quad + X_6\left(\xi\left(X_1\left(X_2 + X_3 + X_4\right) + X_2\left(X_3 + X_4\right) + X_3X_4\right) + X_1X_2\left(X_3 + X_3\right) + X_3X_4\left(X_1 + X_2\right)\right)\left(X_7 + X_8\right)
\end{aligned}
$$

$$
\begin{aligned}
&+\xi X_7X_8\big(X_1\big(X_2+X_3+X_4+X_6\big)+X_2\big(X_3+X_4+X_6\big)+X_3\big(X_4+X_6\big)+X_4X_6\big)\\
&+X_7X_8\big(X_1X_2\big(X_3+X_4+X_6\big)+X_1X_3\big(X_4+X_6\big)+X_2X_3\big(X_4+X_6\big)+X_4X_6\big(X_1+X_2+X_3\big)\big)\\
&+\frac{(1-\varpi)\alpha_h q\varphi\theta\psi X_2\big(\sigma+\xi+X_6+X_7+X_8\big)}{(\theta+\psi+\mu)}+\frac{(1-\varpi)\alpha_e\Lambda\tau_1 q\varphi\theta\psi X_2}{K\gamma(\theta+\psi+\mu)}\\
&-\frac{(1-\varpi)\alpha_h q\varphi\sigma\xi X_2\big(X_1+X_2+X_7+X_8\big)}{(\theta+\psi+\mu)}-\frac{(1-\varpi)\alpha_h q\varphi\sigma X_1X_2\big(X_2+X_7+X_8\big)}{(\theta+\psi+\mu)}\\
&-\frac{(1-\varpi)\alpha_h q\varphi X_2\big(\sigma X_2+\xi X_6+X_1X_6+X_2X_6\big)\big(X_7+X_8\big)}{(\theta+\psi+\mu)}-\frac{(1-\varpi)\alpha_e\Lambda\tau_2 q\varphi\sigma X_2\big(X_1+X_2+X_7+X_8\big)}{K\gamma(\theta+\psi+\mu)}\\
&-\frac{(1-\varpi)\alpha_h q\varphi X_2X_7X_8\big(\sigma+\xi+X_1+X_2+X_6\big)}{(\theta+\psi+\mu)}-\frac{(1-\varpi)\alpha_h q\varphi X_2^2\big(\xi+X_1\big)\big(X_6+X_7+X_8\big)}{(\theta+\psi+\mu)}
\end{aligned}
$$

$$
\begin{aligned}
&-\frac{(1-\varpi)\alpha_h q\varphi\xi X_1X_2\big(X_2+X_6+X_7+X_8\big)}{(\theta+\psi+\mu)}-\frac{(1-\varpi)\alpha_e\Lambda\tau_1 q\varphi X_1X_2\big(X_2+X_6+X_7+X_8\big)}{K\gamma(\theta+\psi+\mu)}\\
&-\frac{(1-\varpi)\alpha_e\Lambda\tau_1 q\varphi X_2^2\big(X_6+X_7+X_8\big)}{K\gamma(\theta+\psi+\mu)}-\frac{(1-\varpi)\alpha_e\Lambda\tau_1 q\varphi X_2X_6\big(X_7+X_8\big)}{K\gamma(\theta+\psi+\mu)}-\frac{(1-\varpi)\alpha_e\Lambda\tau_1 q\varphi X_2X_7X_8}{K\gamma(\theta+\psi+\mu)}\\
&+\frac{(1-\varpi)\alpha_h\theta\psi X_2X_5\big(\xi+X_4+X_7+X_8\big)}{(\theta+\psi+\mu)}+\frac{(1-\varpi)\alpha_e\Lambda\tau_2\theta\psi X_2X_5}{K\gamma(\theta+\psi+\mu)}-\theta\psi\xi X_3\big(X_4+X_6+X_7+X_8\big)\\
&-\theta\psi X_4\big(\xi+X_3\big)\big(X_6+X_7+X_8\big)-\theta\psi X_6\big(\xi+X_3+X_4\big)\big(X_7+X_8\big)-\theta\psi X_7X_8\big(\xi+X_3+X_4+X_6\big)\\
&-\frac{(1-\varpi)\alpha_h\xi X_1X_2X_5\big(X_2+X_4+X_7+X_8\big)}{(\theta+\psi+\mu)}-\frac{(1-\varpi)\alpha_h X_2^2X_5\big(\xi+X_1\big)\big(X_4+X_7+X_8\big)}{(\theta+\psi+\mu)}\\
&-\frac{(1-\varpi)\alpha_h X_2X_4X_5\big(\xi+X_1+X_2\big)\big(X_7+X_8\big)}{(\theta+\psi+\mu)}-\frac{(1-\varpi)\alpha_h X_2X_5X_7X_8\big(\xi+X_1+X_2+X_4\big)}{(\theta+\psi+\mu)}\\
&-\frac{(1-\varpi)\alpha_e\Lambda\tau_2 X_1X_2X_5\big(X_2+X_4+X_7+X_8\big)}{K\gamma(\theta+\psi+\mu)}-\frac{(1-\varpi)\alpha_e\Lambda\tau_2 X_2^2X_5\big(X_4+X_7+X_8\big)}{K\gamma(\theta+\psi+\mu)}\\
&-\frac{(1-\varpi)\alpha_e\Lambda\tau_2 X_2X_4X_5\big(X_7+X_8\big)}{K\gamma(\theta+\psi+\mu)}-\frac{(1-\varpi)\alpha_e\Lambda\tau_2 X_2X_5X_7X_8}{K\gamma(\theta+\psi+\mu)},
\end{aligned}
$$

$$
\begin{aligned}
\mathcal{P}_6=\;&\xi X_1X_2X_3X_4\big(X_6+X_7+X_8\big)+\xi X_1X_6\big(X_2X_3+X_2X_4+X_3X_4\big)\big(X_7+X_8\big)\\
&+X_2X_3X_4X_6\big(\xi+X_1\big)\big(X_7+X_8\big)+\xi X_1X_7X_8\big(X_2X_3+X_2X_4+X_2X_6+X_3X_4+X_3X_6+X_4X_6\big)\\
&+X_7X_8\big(\xi\big(X_2X_3\big(X_4+X_6\big)+X_4X_6\big(X_2+X_3\big)\big)+X_1\big(X_2X_3\big(X_4+X_6\big)+X_4X_6\big(X_2+X_3\big)\big)+X_2X_3X_4X_6\big)\\
&+\frac{(1-\varpi)\alpha_h q\varphi\theta\psi\sigma X_2\big(\xi+X_7+X_8\big)}{(\theta+\psi+\mu)}-\frac{(1-\varpi)\alpha_h q\varphi\xi X_2\big(X_1X_2-\theta\psi\big)\big(X_6+X_7+X_8\big)}{(\theta+\psi+\mu)}\\
&+\frac{(1-\varpi)\alpha_h q\varphi\theta\psi X_2X_6\big(X_7+X_8\big)}{(\theta+\psi+\mu)}+\frac{(1-\varpi)\alpha_h q\varphi\theta\psi X_2X_7X_8}{(\theta+\psi+\mu)}+\frac{(1-\varpi)\alpha_e\Lambda\tau_1 q\varphi\theta\psi X_2\big(X_6+X_7+X_8\big)}{K\gamma(\theta+\psi+\mu)}\\
&+\frac{(1-\varpi)\alpha_e\Lambda\tau_2 q\varphi\theta\psi\sigma X_2}{K\gamma(\theta+\psi+\mu)}-\frac{(1-\varpi)\alpha_h q\varphi\sigma\xi X_1X_2\big(X_2+X_7+X_8\big)}{(\theta+\psi+\mu)}-\frac{(1-\varpi)\alpha_h q\varphi\sigma\xi X_2X_7X_8}{(\theta+\psi+\mu)}\\
&-\frac{(1-\varpi)\alpha_h q\varphi\sigma X_2^2\big(\xi+X_1\big)\big(X_7+X_8\big)}{(\theta+\psi+\mu)}-\frac{(1-\varpi)\alpha_h q\varphi\sigma X_2X_7X_8\big(X_1+X_2\big)}{(\theta+\psi+\mu)}-\frac{(1-\varpi)\alpha_e\Lambda\tau_2 q\varphi\sigma X_2X_7X_8}{K\gamma(\theta+\psi+\mu)}\\
&-\frac{(1-\varpi)\alpha_e\Lambda\tau_2 q\varphi\sigma X_1X_2\big(X_2+X_7+X_8\big)}{K\gamma(\theta+\psi+\mu)}-\frac{(1-\varpi)\alpha_e\Lambda\tau_2 q\varphi\sigma X_2^2\big(X_7+X_8\big)}{K\gamma(\theta+\psi+\mu)}\\
&-\frac{(1-\varpi)\alpha_h q\varphi X_2X_6\big(\xi\big(X_1+X_2\big)+X_1X_2\big)\big(X_7+X_8\big)}{(\theta+\psi+\mu)}-\frac{(1-\varpi)\alpha_h q\varphi\xi X_2X_7X_8\big(X_1+X_2+X_6\big)}{(\theta+\psi+\mu)}\\
&-\frac{(1-\varpi)\alpha_h q\varphi X_2X_7X_8\big(X_1X_2+X_1X_6+X_2X_6\big)}{(\theta+\psi+\mu)}-\frac{(1-\varpi)\alpha_e\Lambda\tau_1 q\varphi X_1X_2^2\big(X_6+X_7+X_8\big)}{K\gamma(\theta+\psi+\mu)}\\
&-\frac{(1-\varpi)\alpha_e\Lambda\tau_1 q\varphi X_2X_6\big(X_1+X_2\big)\big(X_7+X_8\big)}{K\gamma(\theta+\psi+\mu)}-\frac{(1-\varpi)\alpha_e\Lambda\tau_1 q\varphi X_2X_7X_8\big(X_1+X_2+X_6\big)}{K\gamma(\theta+\psi+\mu)}\\
&-\frac{(1-\varpi)\alpha_h\xi X_2X_5\big(X_1X_2-\theta\psi\big)\big(X_4+X_7+X_8\big)}{(\theta+\psi+\mu)}-\theta\psi\xi X_3X_4\big(X_6+X_7+X_8\big)
\end{aligned}
$$

$$
\begin{aligned}
&- \theta\psi\xi X_6\big(X_3+X_4\big)\big(X_7+X_8\big) - \theta\psi\xi X_7X_8\big(X_3+X_4+X_6\big) + \frac{(1-\varpi)\alpha_h\theta\psi X_2X_4X_5\big(X_7+X_8\big)}{(\theta+\psi+\mu)}\\
&+ \frac{(1-\varpi)\alpha_h\theta\psi X_2X_5X_7X_8}{(\theta+\psi+\mu)} + \frac{(1-\varpi)\alpha_e\Lambda\tau_2\theta\psi X_2X_5\big(X_4+X_7+X_8\big)}{K\gamma(\theta+\psi+\mu)} - \theta\psi X_3X_4X_6\big(X_7+X_8\big)\\
&- \theta\psi X_7X_8\big(X_3X_4+X_3X_6+X_4X_6\big) - \frac{(1-\varpi)\alpha_h X_2X_4X_5\big(\xi\big(X_1+X_2\big)+X_1X_2\big)\big(X_7+X_8\big)}{(\theta+\psi+\mu)}\\
&- \frac{(1-\varpi)\alpha_h X_2X_5X_7X_8\big(\xi\big(X_1+X_2+X_4\big)+X_1\big(X_2+X_4\big)+X_2X_4\big)}{(\theta+\psi+\mu)}\\
&- \frac{(1-\varpi)\alpha_e\Lambda\tau_2X_1X_2^2X_5\big(X_4+X_7+X_8\big)}{K\gamma(\theta+\psi+\mu)} - \frac{(1-\varpi)\alpha_e\Lambda\tau_2X_2X_4X_5\big(X_1+X_2\big)\big(X_7+X_8\big)}{K\gamma(\theta+\psi+\mu)} -\\
&\frac{(1-\varpi)\alpha_e\Lambda\tau_2X_2X_5X_7X_8\big(X_1+X_2+X_4\big)}{K\gamma(\theta+\psi+\mu)},\\
\mathcal{P}_7 &= \xi X_4X_6X_7X_8\big(X_1X_2+X_1X_3+X_2X_3\big) + \xi X_1X_2X_3X_7X_8\big(X_4+X_6\big) + \xi X_1X_2X_3X_4X_6\big(X_7+X_8\big)\\
&+ X_1X_2X_3X_4X_6X_7X_8 + \frac{(1-\varpi)\alpha_h q\varphi\theta\psi\xi X_2\big(\sigma+X_6\big)\big(X_7+X_8\big)}{(\theta+\psi+\mu)} + \frac{(1-\varpi)\alpha_h q\varphi\theta\psi X_2X_7X_8\big(\sigma+\xi+X_6\big)}{(\theta+\psi+\mu)}\\
&+ \frac{(1-\varpi)\alpha_e\Lambda q\varphi\theta\psi X_2\big(\tau_1X_6+\tau_2\sigma\big)\big(X_7+X_8\big)}{K\gamma(\theta+\psi+\mu)} + \frac{(1-\varpi)\alpha_e\Lambda q\varphi\theta\psi\tau_1X_2X_7X_8}{K\gamma(\theta+\psi+\mu)}\\
&- \frac{(1-\varpi)\alpha_h q\varphi\xi X_1X_2^2\big(\sigma+X_6\big)\big(X_7+X_8\big)}{(\theta+\psi+\mu)} - \frac{(1-\varpi)\alpha_h q\varphi\sigma X_2X_7X_8\big(\xi\big(X_1+X_2\big)+X_1X_2\big)}{(\theta+\psi+\mu)}\\
&- \frac{(1-\varpi)\alpha_h q\varphi\xi X_1X_2X_7X_8\big(X_2+X_6\big)}{(\theta+\psi+\mu)} - \frac{(1-\varpi)\alpha_h q\varphi X_2^2X_6X_7X_8\big(\xi+X_1\big)}{(\theta+\psi+\mu)}\\
&- \frac{(1-\varpi)\alpha_e\Lambda q\varphi X_1X_2^2\big(\tau_1X_6+\tau_2\sigma\big)\big(X_7+X_8\big)}{K\gamma(\theta+\psi+\mu)} - \frac{(1-\varpi)\alpha_e\Lambda q\varphi\tau_1X_2X_7X_8\big(X_1\big(X_2+X_6\big)+X_2X_6\big)}{K\gamma(\theta+\psi+\mu)}\\
&- \frac{(1-\varpi)\alpha_h\xi X_2X_4X_5\big(X_1X_2-\theta\psi\big)\big(X_7+X_8\big)}{(\theta+\psi+\mu)} + \frac{(1-\varpi)\alpha_h\theta\psi X_2X_5X_7X_8\big(\xi+X_4\big)}{(\theta+\psi+\mu)}\\
&- \frac{(1-\varpi)\alpha_h X_2X_5X_7X_8\big(\xi\big(X_1\big(X_2+X_4\big)+X_2X_4\big)+X_1X_2X_4\big)}{(\theta+\psi+\mu)} - \theta\psi X_4X_6X_7X_8\big(\xi+X_3\big)\\
&- \theta\psi\xi X_3\big(X_4X_6\big(X_7+X_8\big)+X_7X_8\big(X_4+X_6\big)\big) - \frac{(1-\varpi)\alpha_e\Lambda\tau_2X_2X_4X_5\big(X_1X_2-\theta\psi\big)\big(X_7+X_8\big)}{K\gamma(\theta+\psi+\mu)}\\
&- \frac{(1-\varpi)\alpha_e\Lambda\tau_2X_2X_5X_7X_8\big(\big(X_1X_2+X_1X_4+X_2X_4\big)-\theta\psi\big)}{K\gamma(\theta+\psi+\mu)},\\
\mathcal{P}_8 &= \xi X_3X_4X_6X_7X_8\big(X_1X_2-\theta\psi\big)\big(1-\mathcal{R}_0\big).
\end{aligned}
\tag{A1}
$$