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Analysis of a Fractional-Order Mathematical Model of Gonorrhea Transmission with Control Measures

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Abstract

Gonorrhea is a sexually transmitted infection caused by the bacterium *Neisseria gonorrhoeae*. It spreads primarily through sexual contact and can affect the genital tract, rectum, and throat. If left untreated, it may lead to serious health complications, including infertility and increased susceptibility to other infections. This study investigated the transmission dynamics of gonorrhea using a fractional-order mathematical model to evaluate the effects of treatment, vaccination, and contact rates on disease spread. The model established the existence and uniqueness of solutions within the fractional-order framework, confirming that it is well-posed. Stability analysis is conducted to better understand disease behavior, including the computation of the basic reproduction number. The results revealed that increasing treatment rates among infected individuals plays a crucial role in reducing the reproduction number below one, which is necessary for disease control. In contrast, higher contact rates contribute to increased transmission and help sustain the presence of the disease within the population. Simulation results further show that transmission-related parameters promote disease spread, while treatment-related parameters reduce infection levels, thereby lowering the overall disease burden. The dynamics of different population compartments under varying treatment and contact rates are examined using the fractional Adams–Bashforth–Moulton numerical scheme. The findings emphasized that effective treatment of infected individuals is essential for reducing the burden of gonorrhea. The study concludes that combining expanded treatment strategies with reduced transmission pathways is vital for controlling and potentially eradicating the disease in the population.

Keywords:

Gonorrhea, fractional-order model, Basic reproduction number, Stability analysis, Numerical simulation.

Introduction

Gonorrhea is a prevalent sexually transmitted infection (STI) caused by the Gram-negative bacterium *Neisseria gonorrhoeae*, which primarily infects mucosal surfaces such as the

urogenital tract, rectum, pharynx, and conjunctiva. It remains a major global public health concern due to its high transmissibility and potential to cause serious complications if untreated. Clinically, gonorrhea may present with symptoms such as urethral discharge and dysuria, although a large proportion of infections especially in women are asymptomatic, contributing to its continued spread [1][2]. The absence of a licensed vaccine further complicates prevention efforts, making behavioral interventions and early diagnosis essential [3]. Globally, gonorrhea continues to impose a substantial disease burden, with the World Health Organization estimating tens of millions of new infections annually. The infection disproportionately affects sexually active individuals aged 15–49 years, particularly those in high-risk populations such as sex workers and men who have sex with men [4][5]. Recent epidemiological studies indicate a rising incidence in many regions, driven by factors such as inconsistent condom use, urbanization, and limited access to sexual health services [6]. These trends highlight the need for strengthened public health interventions and surveillance systems.

Transmission of *Neisseria gonorrhoeae* occurs through unprotected vaginal, anal, or oral sexual contact with an infected individual. The bacterium adheres to and invades epithelial cells, leading to localized inflammation and tissue damage [2][7]. If left untreated, gonorrhea can ascend the reproductive tract, causing complications such as pelvic inflammatory disease, infertility, and ectopic pregnancy in women. In men, it may result in epididymitis and potential infertility. In rare cases, the infection may disseminate, leading to systemic conditions such as septic arthritis or dermatitis [7][8]. A significant challenge in the control of gonorrhea is the rapid emergence of antimicrobial resistance (AMR). Over time, *Neisseria gonorrhoeae* has developed resistance to multiple antibiotic classes, including penicillins, tetracyclines, and fluoroquinolones. More recently, decreased susceptibility to extended-spectrum cephalosporins and azithromycin—the current first-line treatments has been reported globally [3][9]. This growing resistance threatens the effectiveness of existing therapies and underscores the urgent need for new antibiotics, combination therapies, and vaccine development. Gonorrhea remains a persistent and evolving global health issue characterized by high prevalence, asymptomatic transmission, and increasing antimicrobial resistance. Addressing this challenge requires a comprehensive approach that includes improved diagnostic methods, enhanced surveillance, public health education, and ongoing research into novel treatment and prevention strategies. Strengthening healthcare systems and promoting safe sexual practices are critical steps toward reducing the burden of this infection [5][10].

Adedayo et al [11] formulated a deterministic model that incorporated treatment and behavioral control measures in the transmission dynamics of gonorrhea. Using numerical simulations, the study showed that increasing treatment rates and reducing risky sexual behavior significantly lowered infection levels. The authors concluded that combining multiple intervention strategies was more effective than relying on a single control measure. Al Basir and Abraha [12] developed a mathematical model that integrated awareness-based interventions into malaria transmission dynamics. The model examined how public health education influenced disease spread alongside treatment and prevention methods. The findings revealed that increased public awareness significantly reduced infection rates, particularly when combined with other control strategies. Ayoub et al [13] studied a population-based mathematical model focusing on gonorrhea transmission among female sex workers and their clients. The model accounted for heterogeneity in sexual contact patterns and network structure. The results indicated that targeted interventions among high-risk groups played a critical role in reducing overall

prevalence, highlighting the importance of incorporating population structure into disease models.

Oke et al [14] developed a nonlinear compartmental model to study malaria transmission, incorporating interactions between human hosts and mosquito vectors. The study applied optimal control theory to evaluate intervention strategies such as insecticide-treated nets, treatment, and vector control. The results indicated that a combination of interventions was more effective in reducing malaria prevalence than individual strategies applied in isolation. Reichert et al [15] formulated a mathematical model to evaluate strategies for deploying antibiotics in the treatment of gonorrhea. The study compared different approaches, including combination therapy and antibiotic cycling, to assess their effectiveness in delaying antimicrobial resistance. The findings demonstrated that carefully designed treatment strategies could prolong the effectiveness of antibiotics and reduce the emergence of resistant strains. Ullah et al [16] developed a compartmental model to describe the transmission dynamics of gonorrhea by incorporating self-protection, treatment, and natural immunity. The study analyzed both the local and global stability of the model and conducted sensitivity analysis to determine the most influential parameters. The findings showed that increased treatment rates and effective self-protection measures significantly reduced disease transmission, while variations in key parameters strongly affected the spread of infection.

The main objective of this research work is to model the transmission dynamics of gonorrhea infection and to examine the effect of treatment using a deterministic mathematical framework. The specific objectives are to: (i) formulate a fractional-order mathematical model to describe the transmission dynamics of gonorrhea, with emphasis on determining the basic reproduction number of the disease and its epidemiological significance;

(ii) analyze the stability of the equilibrium points of the model, including both the disease-free and endemic states, in order to understand the long-term behavior of the system; (iii) carry out numerical simulations to investigate and interpret the effects of treatment and the level of compliance with safe sexual practices on the spread and control of the disease; (iv) apply the fractional-order Adams–Bashforth–Moulton numerical method to solve the model and obtain accurate approximations of the system dynamics over time; and (v) conduct sensitivity analysis on key model parameters to identify the most influential factors affecting disease transmission and to provide insights for effective control strategies.

The novelty of this study lies in its integration of fractional-order calculus with the modeling of gonorrhea transmission dynamics, providing a more realistic representation of disease progression compared to classical integer-order models. Unlike many existing studies, this work incorporates memory effects inherent in fractional derivatives, allowing the model to better capture the history-dependent nature of infection and recovery processes. The study uniquely combines treatment, vaccination, and contact rate dynamics within a unified fractional-order framework, offering a more comprehensive understanding of the factors influencing disease spread. Another significant contribution is the establishment of the existence and uniqueness of solutions in the fractional-order sense, which ensures the mathematical well-posedness and reliability of the model. In addition, the application of a generalized Adams–Bashforth–Moulton numerical scheme for solving the model enhances computational accuracy and stability, distinguishing this approach from conventional numerical methods. The study also provides detailed stability analysis and evaluates the basic reproduction number, highlighting the critical role of treatment in reducing disease prevalence.

Preliminaries

Definition 1: [23,24] Let $f \in \Lambda^{\infty}(R)$, The left and right Caputo fractional derivatives of the function f are then defined as

$$ {}^{c}D_{t}^{\varphi} f(t) = \left(t^{0}D_{t}^{-(-n\varphi)} \left(\frac{d}{dt} \right)^{n} f(t) \right) $$

$$ {}^{c}D_{t}^{\varphi} f(t) = \frac{1}{\Gamma(n-\varphi)} \int_{0}^{t} \left((t-\lambda)^{n-\varphi-1} f^{n}(\lambda) \right) d\lambda $$

Similarly

$$ {}_{T}^{c}D_{t}^{\varphi} f(t) = \left({}_{t}D_{T}^{-(n-\varphi)} \left(\frac{-d}{dt} \right)^{n} f(t) \right) $$

$$ {}_{T}^{c}D_{T}^{\varphi} f(t) = \frac{(-1)^{n}}{\Gamma(n-\varphi)} \int_{t}^{T} \left((\lambda-t)^{n-\varphi-1} f^{n}(\lambda) \right) d\lambda $$

Definition 2:[23] The generalized Mittag-Leffler function $E_{\varphi,\beta}(x)$ for $x \in R$ is given by

$$ E_{\varphi,\beta}(x) = \sum_{n=0}^{\infty} \frac{x^{n}}{\Gamma(\varphi n + \beta)}, \varphi, \beta > 0 $$

which can be denoted as;

$$ E_{\varphi,\beta}(x) = xE_{\varphi,\varphi\frac{x-\mu}{\varphi}+\beta(x)} + \frac{1}{\Gamma(\beta)} $$

$$ E_{\varphi,\beta}(x) = L\left[t^{\beta-1} E_{\varphi,\beta}\left(\pm\omega t^{\varphi} \right) \right] = \frac{S^{\varphi-\beta}}{S^{\varphi} \pm \omega} $$

Proposition 1.1.

Let $f \in \Lambda^{\infty}(R) \cap \mathrm{C}(R)$ and $\varphi \in R,\ n-1 < \varphi < n,$

therefore, the conditions given below is satisfied [21,23,24]:

1. ${}_{t_0}^{c}D_{t}^{\varphi} I^{\varphi} f(t) = f(t),$

2. $I_{t_0}^{\varphi} D_{t}^{\varphi} f(t) = f(t) - \sum_{k=0}^{n-k} \frac{t^{k}}{k!} f^{k}(t_o).$

Model Formulation

The population is divided into seven separate groupings according to the integer-order model; susceptible individuals S_G, Individuals who that are free from disease infection make up this group; exposed individuals E_G ;People who have experienced infection exposure without developing contagious status; Asymptomatic infected individuals I_A, symptomatic infected population I_S: are the population of human infected with clinical symptoms, individuals on Gonorrhea treatment T_G, Recovered human population R_G and Bacteria population B. Susceptible human population are recruited at the rate of Λ_h, Susceptible Bacteria population are recruited at the rate of Λ_b, Asymptomatic infected humans progresses to symptomatic infected humans at the of α, Asymptomatic infected humans and symptomatic infected humans receive treatment at the rate of σ and ϕ,Gonorrhea Asymptomatic infected human population, symptomatic infected human population and humans on Gonorrhea treatment die due to the disease at the rate of δ_1, δ_2 *and* δ_3 respectively. Human population recovered due to treatment at the rate of ω and the recovered humans become susceptible again at the rate of ψ. The model is developed under several simplifying assumptions to ensure tractability and analytical clarity. It is assumed that natural death occurs uniformly across all compartments of the population, thereby contributing to a constant reduction in population size independent of disease status. The model further assumes that individuals do not acquire permanent immunity after recovery from gonorrhea, implying that recovered individuals can become susceptible again and re-enter the transmission cycle. In addition, disease-induced death is assumed to occur among infected individuals, reflecting the potential severity of untreated or complicated infections. Finally, it is assumed that the population is homogeneously mixed, meaning that every individual has an equal probability of coming into contact with any other individual, which simplifies the transmission dynamics of the disease.

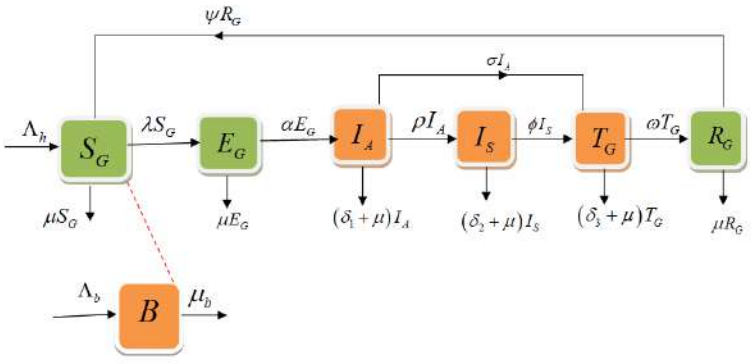


Fig.1: Model flow Diagram of Gonorrhea

Model Equations

Based on the schematic diagram and the model descriptions above we have the following differential equations

$$\frac{dS_G}{dt} = \Lambda_h + \psi R_G - \lambda S_G - \mu, \mathrm{S}_G,$$

$$\frac{dE_G}{dt} = \lambda S_G - (\alpha + \mu), \mathrm{E}_G,$$

$$\frac{dI_A}{dt} = \alpha E_G - (\rho + \sigma + \delta_1 + \mu), I_A,$$

$$\frac{dI_S}{dt} = \rho I_A - (\phi + \delta_2 + \mu) I_S,$$

$$\frac{dT_G}{dt} = \phi I_S + \sigma I_A - (\omega + \delta_3 + \mu) T_G, \tag{1}$$

$$\frac{dR_G}{dt} = \omega T_G - (\psi + \mu) \mathrm{R}_G,$$

$$\frac{dB}{dt} = \Lambda_b - \mu_b.$$

Where $\lambda = \dfrac{(\phi_1 I_A + \phi_2 I_S + \phi_3 T_G + \phi_4 B)}{N_h}$.

Table 1. Model Variables and parameter Description

Model Variables	**Descriptions**
S_G	Susceptible Humans to Gonorrhea
E_G	Exposed humans to Gonorrhea
I_A	Asymptomatic infected humans with Gonorrhea
I_S	Symptomatic infected humans with Gonorrhea
T_G	Humans on Gonorrhea treatment
R_G	Recovered human population from Gonorrhea
B	Bacteria population
Model parameters	**Descriptions**
Λ_h	Recruitment rate of human population.
Λ_b	Recruitment rate of Bacteria population.
μ	Natural death rate of human population.
μ_b	Natural death rate of Bacteria population.
α	Progression rate from exposed human population to Gonorrhea asymptomatic infected human population.

δ_1	Disease induced death rate of Asymptomatic infected human population with Gonorrhea.
δ_2	Disease induced death rate of symptomatic infected human population with Gonorrhea.
δ_3	Disease induced death rate of human population on Gonorrhea treatment.
ϕ	Treatment rate of Gonorrhea symptomatic infected human population.
σ	Treatment rate of Gonorrhea asymptomatic infected human population.
ϕ_1	Contact rate between the susceptible and Gonorrhea asymptomatic infected human population.
ϕ_2	Contact rate between the susceptible and Gonorrhea symptomatic infected human population.
ϕ_3	Contact rate between the susceptible and human population Gonorrhea treatment.
ϕ_4	Contact rate between the susceptible and Bacteria population
ω	Recovery due to treatment rate
ψ	Re-infection rate

Model Analysis

Disease Free Equilibrium Point of Gonorrhea

Disease free equilibrium point is a point where there is no disease in the population

At DFE $S_G \neq 0, E_G = 0, \mathrm{I}_A = 0, \mathrm{I}_S = 0, T_G = 0, \mathrm{R}_G = 0, B \neq 0.$

$$\left(S_G^0, E_G^0, \mathrm{I}_A^0, \mathrm{I}_S^0, \mathrm{T}_G^0, \mathrm{R}_G^0, B^0\right) = \left(\frac{\Lambda_h}{\mu}, 0, 0, 0, 0, 0, \frac{\Lambda_b}{\mu_b}\right).$$

4.2 Basic Reproduction Number of Gonorrhea

Basic Reproduction number R_0^G is the number of secondary cases caused by individual infected people [16]. Next generation method $R_0^G = \rho FV^{-1}$ is used to compute R_0^G using F, a non-negative matrix (other transition terms V) and finding the dominant Eigen value ρ.

$$F=\begin{pmatrix}0&\phi_1&\phi_2&\phi_3&\phi_4\\0&0&0&0&0\\0&0&0&0&0\\0&0&0&0&0\\0&0&0&0&0\end{pmatrix}\text{ and }V=\begin{pmatrix}Q_2&0&0&0&0\\-\alpha&Q_3&0&0&0\\0&-\rho&Q_4&0&0\\0&-\sigma&-\phi&Q_5&0\\0&0&0&0&Q_7\end{pmatrix}$$

$$V^{-1}=\begin{pmatrix}\frac{1}{Q_2}&0&0&0&0\\\frac{\alpha}{Q_3Q_2}&\frac{1}{Q_3}&0&0&0\\\frac{\rho\alpha}{Q_4Q_3Q_2}&\frac{\rho}{Q_4Q_3}&\frac{1}{Q_4}&0&0\\\frac{\alpha(\phi\rho+\sigma Q_4)}{Q_4Q_3Q_2Q_5}&\frac{\phi\rho+\sigma Q_4}{Q_4Q_3Q_5}&\frac{\phi}{Q_4Q_5}&\frac{1}{Q_5}&0\\0&0&0&0&\frac{1}{Q_7}\end{pmatrix},$$

$$FV^{-1}=\begin{pmatrix}\frac{\phi_1\alpha}{Q_3Q_2}+\frac{\phi_2\rho\alpha}{Q_4Q_3Q_2}+\frac{\phi_3\alpha(\phi\rho+\sigma Q_4)}{Q_4Q_3Q_2Q_5}&\frac{\phi_1}{Q_3}+\frac{\phi_2\rho}{Q_4Q_3}+\frac{\phi_3(\phi\rho+\sigma Q_4)}{Q_4Q_3Q_5}&\frac{\phi_2}{Q_4}+\frac{\phi_3\phi}{Q_4Q_5}&\frac{\phi_3}{Q_5}&\frac{\phi_4}{Q_7}\\0&0&0&0&0\\0&0&0&0&0\\0&0&0&0&0\\0&0&0&0&0\end{pmatrix},$$

$$Eigen\,value=\begin{pmatrix}0\\0\\0\\0\\\frac{\alpha(\phi\rho\phi_3+\phi_2\rho Q_5+\sigma Q_4\phi_3+\phi_1Q_4Q_5)}{Q_4Q_3Q_2Q_5}\end{pmatrix},$$

The basic reproduction number is:

$$R_0^G=\frac{\alpha(\phi\rho\phi_3+\phi_2\rho Q_5+\sigma Q_4\phi_3+\phi_1Q_4Q_5)}{Q_4Q_3Q_2Q_5}. \tag{2}$$

Where $Q_1 = \mu,\ Q_2 = (\alpha + \mu),\ Q_3 = (\rho + \sigma + \delta_1 + \mu),\ Q_4 = (\phi + \delta_2 + \mu),\ Q_5 = (\omega + \delta_3 + \mu)$, $Q_6 = (\psi + \mu), Q_7 = \mu_b.$

Endemic Equilibrium of Gonorrhea

Endemic Equilibrium signifies the permanent existence of Gonorrhea among human population.

At endemic equilibrium point $(S_G \neq 0, \mathrm{E}_G \neq 0, I_A \neq 0, I_S \neq 0, \mathrm{T}_G \neq 0, \mathrm{R}_G \neq 0, B \neq 0).$

We obtain the following endemic equilibrium points:

$$S_G^{**} = -\frac{\Lambda_b Q_2 Q_3 Q_4 Q_5 Q_6}{\left(-Q_3 Q_5 Q_6 (\lambda + Q_1) Q_2 + \alpha\lambda\omega\phi\psi\right) Q_4 + \alpha\lambda\omega\psi\rho\sigma},$$

$$E_G^{**} = -\frac{\Lambda_b Q_2 Q_3 Q_4 Q_5 Q_6}{\left(-Q_3 Q_5 Q_6 (\lambda + Q_1) Q_2 + \alpha\lambda\omega\phi\psi\right) Q_4 + \alpha\lambda\omega\psi\rho\sigma},$$

$$I_A^{**} = -\frac{\alpha\lambda\Lambda_b Q_4 Q_5 Q_6}{\left((\alpha\omega\phi\psi - Q_3 Q_5 Q_6 Q_2)\lambda - Q_3 Q_5 Q_6 Q_2 Q_1\right) Q_4 + \alpha\lambda\omega\psi\rho\sigma},$$

$$I_A^{**} = -\frac{\alpha\lambda\Lambda_b Q_4 Q_5 Q_6}{\left((\alpha\omega\phi\psi - Q_3 Q_5 Q_6 Q_2)\lambda - Q_3 Q_5 Q_6 Q_2 Q_1\right) Q_4 + \alpha\lambda\omega\psi\rho\sigma},$$

$$I_S^{**} = -\frac{\alpha\lambda\Lambda_b Q_5 Q_6 \rho}{\left((\alpha\omega\phi\psi - Q_3 Q_5 Q_6 Q_2) Q_4 + \psi\rho\alpha\omega\sigma\right)\lambda - Q_1 Q_2 Q_3 Q_4 Q_5 Q_6},$$

$$T_G^{**} = -\frac{\alpha\lambda\Lambda_b Q_6 (\phi Q_4 + \rho\sigma)}{\left((\alpha\omega\phi\psi - Q_3 Q_5 Q_6 Q_2)\lambda - Q_3 Q_5 Q_6 Q_2 Q_1\right) Q_4 + \alpha\lambda\omega\psi\rho\sigma},$$

$$R_G^{**} = -\frac{\omega\alpha\lambda\Lambda_b (\phi Q_4 + \rho\sigma)}{\alpha\lambda\omega\phi\psi Q_4 + \alpha\lambda\omega\psi\rho\sigma - \lambda Q_2 Q_3 Q_4 Q_5 Q_6 - Q_1 Q_2 Q_3 Q_4 Q_5 Q_6},$$

$$B^{**} = \frac{\Lambda_b}{\mu_b}.$$

Substituting into the force of infection $\lambda = \frac{(\phi_1 I_A + \phi_2 I_S + \phi_3 T + \phi_4 B)}{N_h}.$

We have;

$$Q_1\lambda + Q_2 = 0$$

$$P_1 = \begin{pmatrix} \alpha\omega\phi Q_4 Q_7 + \alpha\omega\rho\sigma Q_7 + \alpha\phi Q_4 Q_6 Q_7 + \alpha\rho\sigma Q_6 Q_7 \\ +\alpha\rho Q_5 Q_6 Q_7 + \alpha Q_4 Q_5 Q_6 Q_7 + Q_3 Q_4 Q_5 Q_6 Q_7 \end{pmatrix},$$

$$P_2 = \begin{pmatrix} \alpha\omega\phi\psi Q_4\phi_4 + \alpha\omega\psi\rho\sigma\phi_4 + Q_2 Q_3 Q_4 Q_5 Q_6 Q_7 \\ -Q_2 Q_3 Q_4 Q_5 Q_6\phi_4 - \alpha\phi Q_4 Q_6 Q_7\phi_3 - \alpha\rho\sigma Q_6 Q_7\phi_3 \\ -\alpha\rho Q_5 Q_6 Q_7\phi_2 - \alpha Q_4 Q_5 Q_6 Q_7\phi_1 \end{pmatrix},$$

$$P_2 = \left(\alpha\omega\phi\psi Q_4\phi_4 + \alpha\omega\psi\rho\sigma\phi_4 \left(1 - \begin{pmatrix} \alpha\omega\phi\psi Q_4\phi_4 + \alpha\omega\psi\rho\sigma\phi_4 + Q_2 Q_3 Q_4 Q_5 Q_6 Q_7 \\ -Q_2 Q_3 Q_4 Q_5 Q_6\phi_4 - \alpha\phi Q_4 Q_6 Q_7\phi_3 - \alpha\rho\sigma Q_6 Q_7\phi_3 \\ -\alpha\rho Q_5 Q_6 Q_7\phi_2 - \alpha Q_4 Q_5 Q_6 Q_7\phi_1 \end{pmatrix} \right) \right),$$

$$P_2 = \left(\alpha\omega\phi\psi Q_4\phi_4 + \alpha\omega\psi\rho\sigma\phi_4 \left(1 - R_0^G \right) \right).$$

This shows that the endemic equilibrium point of the model is stable and has a unique positive solution if $R_0^G > 1$.

Fractional order mathematical model

$$\begin{aligned}
{}^C D_t^\varphi S_G &= \Lambda_h + \psi R_G - \frac{\left(\phi_1 I_A + \phi_2 I_S + \phi_3 T_G + \phi_4 B\right) S_G}{N_h} - \mu \mathrm{S}_G, \\
{}^C D_t^\varphi E_G &= \frac{\left(\phi_1 I_A + \phi_2 I_S + \phi_3 T_G + \phi_4 B\right) S_G}{N_h} - \left(\alpha + \mu\right) \mathrm{E}_G, \\
{}^C D_t^\varphi I_A &= \alpha E_G - \left(\rho + \sigma + \delta_1 + \mu\right) I_A, \\
{}^C D_t^\varphi I_S &= \rho I_A - \left(\phi + \delta_2 + \mu\right) I_S, \\
{}^C D_t^\varphi T_G &= \phi I_S + \sigma I_A - \left(\omega + \delta_3 + \mu\right) T_G, \\
{}^C D_t^\varphi R_G &= \omega T_G - \left(\psi + \mu\right) \mathrm{R}_G, \\
{}^C D_t^\varphi B &= \Lambda_b - \mu_b.
\end{aligned} \tag{3}$$

Where $\lambda = \dfrac{\left(\phi_1 I_A + \phi_2 I_S + \phi_3 T_G + \phi_4 B\right)}{N_h}$.

Subject to the initial conditions

$$S_G\left(0\right) = S_{G0},\ \mathrm{E}_G\left(0\right) = E_{G0},\ \mathrm{I}_A\left(0\right) = I_{A0},\ \mathrm{I}_S\left(0\right) = I_{S0}, T_G\left(0\right) = T_{G0}, \mathrm{R}_G\left(0\right) = R_{G0}, B\left(0\right) = B_0.$$

Positivity of model solution

Positivity of a model solution means the solution stays nonnegative for all time if it starts nonnegative.
It is important because many real quantities (like population or concentration) cannot be negative [22].
A model preserves positivity if its equations do not force the solution below zero. This is often shown by checking the behavior at the boundary (e.g., when the variable is zero).

$$N(t) \le \frac{\Lambda_h}{\mu} \quad \text{as} \quad t \to \infty$$

Secondly, $\text{if } \limsup N_0(t) \le \frac{\Lambda_h}{\mu}$, then our model feasible domain is given by:

$$\Omega = \left\{ \left(S_G, E_G, I_A, I_S, \mathrm{T}_G, \mathrm{R}_G, \mathrm{B} \right) \subset R_+^7 : S_G + E_G + I_A + I_S + \mathrm{T}_G + R_G + \le \frac{\Lambda_h}{\mu} \right\},$$

so that,

$$\Omega = \Omega_h \subset R_+^7,$$

Therefore, Ω is positively invariant.

If $\left(S_{G0}, E_{G0}, I_{A0}, I_{S0}, \mathrm{T}_{G0}, \mathrm{R}_{G0}, B_0 \right)$ are non-negative, then the solution of model (1) will be non-negative for $t > 0$. From Eq. (3), selecting the first equation, we obtained:

$${}^{C}D_t^{\varphi} S_G = \Lambda_h + \psi R_G - \frac{\left(\phi_1 I_A + \phi_2 I_S + \phi_3 T_G + \phi_4 B \right) S_G}{N_h} - \mu \mathrm{S}_G,$$

$${}^{C}D_t^{\varphi} S_G + \frac{\left(\phi_1 I_A + \phi_2 I_S + \phi_3 T_G + \phi_4 B + \mu \right) S_G}{N_h} = \Lambda_h + \psi R_G, \qquad (4)$$

$${}^{C}D_t^{\varphi} S_G + \frac{\left(\phi_1 I_A + \phi_2 I_S + \phi_3 T_G + \phi_4 B + \mu \right) S_G}{N_h} = \Lambda_h + \psi R_G,$$

But $\Lambda_h + \psi R_G \ge 0$, then,

$${}^{C}D_t^{\varphi} S_G + \frac{\left(\phi_1 I_A + \phi_2 I_S + \phi_3 T_G + \phi_4 B + \mu \right) S_G}{N_h} \ge 0.$$

Applying the Laplace transform we obtained:

$$L\left[{}^{C}D_t^{\varphi} S_G \right] + L\left[\frac{\left(\phi_1 I_A + \phi_2 I_S + \phi_3 T_G + \phi_4 B + \mu \right) S_G}{N_h} \right] \ge 0.$$

$$S_G^{\varphi} S_G(s) - S_G^{\varphi-1} S_G(0) + \frac{(\phi_1 I_A + \phi_2 I_S + \phi_3 T_G + \phi_4 B + \mu) S_G}{N_h}(s) \geq 0,$$

$$S_G(s) \geq \frac{S_G^{\varphi-1}}{S_H^{\varphi} + \frac{(\phi_1 I_A + \phi_2 I_S + \phi_3 T_G + \phi_4 B + \mu)}{N_h}} S_G(0).$$

By taking the inverse Laplace transform, we obtained:

$$S_G(t) \geq E_{t\varphi,1}\left(-\left(\frac{(\phi_1 I_A + \phi_2 I_S + \phi_3 T_G + \phi_4 B + \mu)}{N_h} \right) t^{\varphi} \right) \mathrm{S}_{G0}. \quad (5)$$

Now, since the term on the right-hand side of Eq. (5) is positive, we conclude that $S_G \geq 0$ for $t \geq 0$. In the same way, we also have that $E_G \geq 0, I_A \geq 0, I_S \geq 0, T_G \geq 0, \mathrm{R}_G \geq 0, B \geq 0.$ that are positives. Therefore, the solution will remain in R_+^7 for all $t \geq 0$ with positive initial conditions.

Boundedness of fractional model solution

The total human population from our model is given by;

$$N_h(t) = S_G(t) + E_G(t) + I_A(t) + I_S(t) + T_G(t) + R_G(t) + B(t).$$

So, from our fractional model, we now obtain:

$${}^{c}D_t^{\varphi} N_h(t) = {}^{c}D_t^{\varphi} S_G(t) + {}^{c}D_t^{\varphi} E_G(t) + {}^{c}D_t^{\varphi} I_A(t) + {}^{c}D_t^{\varphi} I_S(t) + {}^{c}D_t^{\varphi} T_G(t) + {}^{c}D_t^{\varphi} R_G(t) + {}^{c}D_t^{\varphi} B(t)$$

$${}^{c}D_t^{\varphi} N_h(t) = \Lambda_h - \mu N_h(t), \quad (6)$$

Taking the Laplace transformation, we obtained:

$$L\left[{}^{c}D_t^{\varphi} N_h(t) \right] = L\left[\Lambda_h - \mu N_h(t) \right],$$

$$S_G^{\varphi} N_h(\mathrm{s}) - S_G^{\varphi-1} N_h(0) + \mu N_h(s) \leq \frac{\Lambda_h}{\mu},$$

$$N_h(\mathrm{s}) \leq \frac{S_G^{\varphi-1}}{\left(S_G^{\varphi} + \mu\right)} N_h(0) + \frac{\Lambda_h}{S_G\left(S_G^{\varphi} + \mu\right)}, \quad (7)$$

By taking the inverse Laplace transform of Eq. (7), we obtain:

$$N_h(\mathrm{t}) \leq E_{t\varphi,1}\left(-\mu t^{\varphi}\right) N_h(0) + \Lambda_h E_{t\varphi,\varphi+1}\left(-\mu t^{\varphi}\right) \quad (8)$$

At $t \to \infty$, the limit of Eq. (8) becomes:

$$\lim_{t\to\infty} SupN(t) = \frac{\Lambda_h}{\mu}.$$

This means that, if $N_{h0} \le \frac{\Lambda_h}{\mu}$.

Then, $N_h(t) \le \frac{\Lambda_h}{\mu}$ which implies that, $N_h(t)$ is bounded.

We now conclude that, this region $\Omega = \Omega_h$, is well posed and equally feasible epidemiologically.

Existence and uniqueness of our model solution

Let the real non-negative be V, we consider $U = [0, \mathrm{V}]$.

The set of all continuous function that is defined on P is represented by $N_e^0(V)$ with norm as:

$$\|K\| = Sup\{|K(t)|, \mathrm{t} \in V\}.$$

Considering model (3) along with the initial conditions specified, this can be represented as an initial value problem (IVP).

$${}^{c}D_t^{\varphi} K(t) = Z(t, \mathrm{K}(t)), 0 < t < V < \infty,$$

$$K(0) = K_0.$$

Where $K(t) = (S_G(t), E_G(t), I_A(t), I_S(t), T_G(t), \mathrm{R}_G(t), B(t))$. represent the classes and Z be a continuous function defined as follows:

$$Z\left(t,\mathrm{K}\left(t\right)\right)=\begin{pmatrix} Z_1\left(t,S_G\left(t\right)\right)\\ Z_2\left(t,\mathrm{E}_G\left(t\right)\right)\\ Z_3\left(t,\mathrm{I}_A\left(t\right)\right)\\ Z_4\left(t,\mathrm{I}_S\left(t\right)\right)\\ Z_5\left(t,\mathrm{T}_G\left(t\right)\right)\\ Z_6\left(t,\mathrm{R}_G\left(t\right)\right)\\ Z_7\left(t,B\left(t\right)\right) \end{pmatrix}=\begin{pmatrix} \Lambda_h+\psi R_G-\dfrac{\left(\phi_1 I_A+\phi_2 I_S+\phi_3 T_G+\phi_4 B\right)S_G}{N_h}-\mu\mathrm{S}_G\\ \dfrac{\left(\phi_1 I_A+\phi_2 I_S+\phi_3 T_G+\phi_4 B\right)S_G}{N_h}-\left(\alpha+\mu\right)\mathrm{E}_G\\ \alpha E_G-\left(\rho+\sigma+\delta_1+\mu\right)I_A\\ \rho I_A-\left(\phi+\delta_2+\mu\right)I_S\\ \phi I_S+\sigma I_A-\left(\omega+\delta_3+\mu\right)T_G\\ \omega T_G-\left(\psi+\mu\right)\mathrm{R}_G\\ \Lambda_b-\mu_b B \end{pmatrix}, \tag{9}$$

Using proposition (1.1), we have that,

$$\begin{aligned} S_G\left(t\right)&=S_{G0}+I_t^{\varphi}\left[\Lambda_h+\psi R_G-\frac{\left(\phi_1 I_A+\phi_2 I_S+\phi_3 T_G+\phi_4 B\right)S_G}{N_h}-\mu\mathrm{S}_G\right],\\ E_G\left(t\right)&=E_{G0}+I_t^{\varphi}\left[\frac{\left(\phi_1 I_A+\phi_2 I_S+\phi_3 T_G+\phi_4 B\right)S_G}{N_h}-\left(\alpha+\mu\right)\mathrm{E}_G\right],\\ I_A\left(t\right)&=I_{A0}+I_t^{\varphi}\left[\alpha E_G-\left(\rho+\sigma+\delta_1+\mu\right)I_A\right],\\ I_S\left(t\right)&=I_{S0}+I_t^{\varphi}\left[\rho I_A-\left(\phi+\delta_2+\mu\right)I_S\right],\\ T_G\left(t\right)&=T_{G0}+I_t^{\varphi}\left[\phi I_S+\sigma I_A-\left(\omega+\delta_3+\mu\right)T_G\right],\\ R_{G0}\left(t\right)&=R_{G0}+I_t^{\varphi}\left[\omega T_G-\left(\psi+\mu\right)\mathrm{R}_G\right],\\ B_0\left(t\right)&=B_0+I_t^{\varphi}\left[\Lambda_b-\mu_b B\right]. \end{aligned} \tag{10}$$

We now obtain the following:

$$\begin{aligned} S_{Gn}\left(t\right)&=S_{G0}+\frac{1}{\Gamma\left(\varphi\right)}\int_0^t\left(t-\lambda\right)^{\varphi-1}Z_1\left(\lambda,S_{G(n-1)}\left(\lambda\right)\right)\mathrm{d}\lambda,\\ E_{Gn}\left(t\right)&=E_{G0}+\frac{1}{\Gamma\left(\varphi\right)}\int_0^t\left(t-\lambda\right)^{\varphi-1}Z_2\left(\lambda,\mathrm{E}_{G(n-1)}\left(\lambda\right)\right)\mathrm{d}\lambda,\\ I_{An}\left(t\right)&=I_{A0}+\frac{1}{\Gamma\left(\varphi\right)}\int_0^t\left(t-\lambda\right)^{\varphi-1}Z_3\left(\lambda,\mathrm{I}_{A(n-1)}\left(\lambda\right)\right)\mathrm{d}\lambda, \end{aligned}$$

$$I_{Sn}(t)=I_{S0}+\frac{1}{\Gamma(\varphi)}\int_0^t(t-\lambda)^{\varphi-1}Z_4\left(\lambda,\mathrm{I}_{S(n-1)}(\lambda)\right)\mathrm{d}\lambda,$$

$$T_{Hn}(t)=E_{H0}+\frac{1}{\Gamma(\varphi)}\int_0^t(t-\lambda)^{\varphi-1}Z_5\left(\lambda,\mathrm{T}_{H(n-1)}(\lambda)\right)\mathrm{d}\lambda, \qquad (11)$$

$$R_{Gn}(t)=R_{G0}+\frac{1}{\Gamma(\varphi)}\int_0^t(t-\lambda)^{\varphi-1}Z_6\left(\lambda,\mathrm{R}_{G(n-1)}(\lambda)\right)\mathrm{d}\lambda,$$

$$B_n(t)=B_0+\frac{1}{\Gamma(\varphi)}\int_0^t(t-\lambda)^{\varphi-1}Z_7\left(\lambda,\mathrm{B}_{(n-1)}(\lambda)\right)\mathrm{d}\lambda.$$

Transforming the above equation yields

$$X(t)=X(0)+\frac{1}{\Gamma(\varphi)}\int_0^t(t-\lambda)^{\varphi-1}Z\left(\lambda,X(\lambda)\right)\mathrm{d}\lambda. \qquad (12)$$

Lemma 1, The Lipchitz condition described from Eq. (11) is satisfied by vector $Z\left(\mathrm{t},\mathrm{K}(t)\right)$ on a set $[0,\mathrm{V}]\times R_+^6$ with the Lipchitz constant given as:

$$\beta=\max\left(\left(\beta_1^*+\beta_2^*+\beta_3^*+\beta_4^*+\mu\right),(\alpha+\mu),(\rho+\sigma+\delta_1+\mu),(\phi+\delta_2+\mu),(\omega+\delta_3+\mu),(\psi+\mu),(\mu_b)\right).$$

Proof.

$$\left\|Z_1(t,S_h)-Z_1(t,S_{h1})\right\|$$

$$=\left\|\left(\Lambda_h+\psi R_G-\frac{\left(\phi_1 I_A+\phi_2 I_S+\phi_3 T_G+\phi_4 B+\mu\right)S_G}{N_h}\right)-\Lambda_h+\psi R_G-\frac{\left(\phi_1 I_A+\phi_2 I_S+\phi_3 T_G+\phi_4 B+\mu\right)}{N_h}S_{G1}\right\|$$

,

$$=\left\|\Lambda_h+\psi R_G-\frac{\left(\phi_1 I_A+\phi_2 I_S+\phi_3 T_G+\phi_4 B+\mu\right)}{N_h}\left(S_G-S_{G1}\right)+\mu\left(S_G-S_{G1}\right)\right\|$$

$$\le\left(\beta_1^*+\beta_2^*+\beta_3^*+\beta_4^*\right)\left\|S_G-S_{G1}\right\|+\mu\left\|S_G-S_{G1}\right\|,$$

$$\therefore\left\|Z_1(t,S_G)-Z_1(t,S_{G1})\right\|\le\left(\beta_1^*+\beta_2^*+\beta_3^*+\beta_4^*+\mu\right)\left\|S_G-S_{G1}\right\|, \quad (13)$$

Similarly, the following are obtained:

$$\left\|Z_2(t,\mathrm{E}_G)-Z_2(t,\mathrm{E}_{G1})\right\|\le(\alpha+\mu)\left\|\mathrm{E}_G-\mathrm{E}_G\right\|,$$

$$\left\|Z_3(t,\mathrm{I}_A)-Z_3(t,\mathrm{I}_{A1})\right\|\le(\rho+\sigma+\delta_1+\mu)\left\|\mathrm{I}_A-\mathrm{I}_{A1}\right\|, \qquad (14)$$

$$\left\|Z_4(t,\mathrm{I}_S)-Z_4(t,\mathrm{I}_{S1})\right\|\le(\phi+\delta_2+\mu)\left\|I_S-I_{S1}\right\|,$$

$$\left\|Z_5\left(t,\mathrm{T}_G\right)-Z_5\left(t,\mathrm{T}_{G1}\right)\right\|\le\left(\omega+\delta_3+\mu\right)\left\|\mathrm{T}_G-\mathrm{T}_{G1}\right\|,$$

$$\left\|Z_6\left(t,\mathrm{R}_G\right)-Z_6\left(t,\mathrm{R}_{G1}\right)\right\|\le\left(\psi+\mu\right)\left\|R_G-R_{G1}\right\|,$$

$$\left\|Z_7\left(t,B\right)-Z_7\left(t,B_1\right)\right\|\le\left(\mu_b\right)\left\|B-B_1\right\|.$$

From (14) we have

$$\left\|Z\left(t,\mathrm{K}_1\left(t\right)\right)-Z\left(t,\mathrm{K}_2\left(t\right)\right)\right\|\le\varphi\left\|K_1-K_2\right\|,$$

$$\beta=\max\left(\left(\beta_1^*+\beta_2^*+\beta_3^*+\beta_4^*+\mu\right),\left(\alpha+\mu\right),\left(\rho+\sigma+\delta_1+\mu\right),\left(\phi+\delta_2+\mu\right),\left(\omega+\delta_3+\mu\right),\left(\psi+\mu\right),\left(\mu_b\right)\right). \quad (15)$$

Lemma 2. The initial value problem (5), (6) exist and have a unique solution

$$K\left(t\right)\in D_c^0\left(E\right).$$

Using Picard Lindelöfand fixed-point theory, we consider the solution of

$$K\left(t\right)=S_G\left(K\left(t\right)\right),$$

where S is defined as the Picard operator expressed as:

$$S_G:\mathrm{D}_c^0\left(\mathrm{E},R_+^7\right)\to D_c^0\left(\mathrm{E},R_+^7\right).$$

Therefore,

$$S_G\left(K\left(t\right)\right)=K\left(0\right)+\frac{1}{\Gamma\left(\varphi\right)}\int_0^t\left(t-\lambda\right)^{\varphi-1}Z\left(\lambda,\mathrm{K}\left(\lambda\right)\right)\mathrm{d}\,\lambda.$$

which becomes:

$$\left\|S_G\left(K_1\left(t\right)\right)-S_G\left(K_2\left(t\right)\right)\right\|,$$

$$=\left\|\frac{1}{\Gamma\left(\varphi\right)}\left[\int_0^t\left(t-\lambda\right)^{\varphi-1}Z\left(\lambda,\mathrm{K}_1\left(\lambda\right)\right)-Z\left(\lambda,\mathrm{K}_2\left(\lambda\right)\right)\mathrm{d}\,\lambda\right]\right\|,$$

$$\le\frac{1}{\Gamma\left(\varphi\right)}\int_0^t\left(t-\lambda\right)^{\varphi-1}\left\|Z\left(\lambda,\mathrm{K}_1\left(\lambda\right)\right)-Z\left(\lambda,\mathrm{K}_2\left(\lambda\right)\right)\mathrm{d}\,\lambda\right\|.$$

$$\le\frac{\psi}{\Gamma\left(\varphi\right)}\int_0^t\left(t-\lambda\right)^{\varphi-1}\left\|K_1-K_2\right\|d\lambda.$$

$$\left\| S_G\left(K_1(t)\right) - S_G\left(K_2(t)\right) \right\| \le \frac{\beta}{\Gamma(\varphi+1) S_G}. \qquad (16)$$

When $\frac{\beta}{\Gamma(\varphi+1)} S_G \le 1$,

Then, the Picard operator gives a contradiction, so Eq.(3) solution is unique

Implementation of fractional Adams–Bashforth–Moulton method

The approach developed by [21, 22] and Freed is adopted in this study. An approximate solution to the fractional Gonorrhea model in equation (3) is obtained using the fractional Adams–Bashforth–Moulton scheme. Consequently, the fractional model in (3) can be rewritten as follows:

$$^{c}D_t^{\varphi} P(t) = Q\left(t, \mathrm{q}(t)\right),\ 0 < t < \beta, \qquad (17)$$

$$P^{(n)}(0) = P_0^{(n)},\ n = 1, 0, \ldots, \mathrm{q}, \mathrm{q} = [\varphi].$$

Where $P = \left(S_G^*, E_G^*, I_A^*, I_S^*, \mathrm{T}_G^*, \mathrm{R}_G^*, \mathrm{B}^*\right) \in R_+^7$ and $V\left(t, \mathrm{q}(t)\right)$ is a real valued function that is continuous.

Eq. (17) can therefore be represented using the concept of fractional integral as follows:

$$P(t) = \sum_{n=0}^{m-1} P_0^{(n)} \frac{t^n}{n!} + \frac{1}{\Gamma(\varphi)} \int_0^t (t-y)^{\varphi-1} R\left(\mathrm{k}, m(k)\right) dk$$

(18)

Let the step size $g = \frac{\beta}{N}$, $N \in \mathrm{N}$ with a grid that is uniform on $[0, \beta]$. Where $t_c = cr,\ c = 0,1,1,\ldots N.$ Therefore, the fractional order model of Gonorrhea model presented in (6) can be approximated as:

$$S_{Gk+1}(t) = S_{G0} + \frac{g^{\varphi}}{\Gamma(\varphi+2)} \left\{ \Lambda_h + \psi R_G^n - \frac{\left(\phi_1 I_A + \phi_2 I_S + \phi_3 T_G + \phi_4 B\right) S_G^n}{N_h} - \mu \mathrm{S}_G^n \right\} +$$

$$\frac{g^{\varphi}}{\Gamma(\varphi+2)} \sum_{y=0}^{k} dy, k+1 \left\{ \Lambda_h + \psi R_{Gy} - \frac{\left(\phi_1 I_{Ay} + \phi_2 I_{Sy} + \phi_3 T_{Gy} + \phi_4 B_y\right) S_{Gy}}{N_h} - \mu \mathrm{S}_{Gy} \right\},$$

$$E_{Gk+1}(t)=E_{G0}+\frac{g^{\varphi}}{\Gamma(\varphi+2)}\left\{\frac{\left(\phi_1 I_A^n+\phi_2 I_S^n+\phi_3 T_G^n+\phi_4 B^n\right)S_G^n}{N_h}-(\alpha+\mu)E_G^n\right\}+$$

$$\frac{g^{\varphi}}{\Gamma(\varphi+2)}\sum_{y=0}^{k} dy,k+1\left\{\frac{\left(\phi_1 I_{Ay}+\phi_2 I_{Sy}+\phi_3 T_{Gy}+\phi_4 B_y\right)S_{Gy}}{N_{hy}}-(\alpha+\mu)\mathrm{E}_{Gy}\right\},$$

(19)

$$I_{A(k+1)}(t)=I_{A0}+\frac{g^{\varphi}}{\Gamma(\varphi+2)}\left\{\alpha E_G^n-(\rho+\sigma+\delta_1+\mu)I_A^n\right\}+$$

$$\frac{g^{\varphi}}{\Gamma(\varphi+2)}\sum_{y=0}^{k} dy,k+1\left\{\alpha E_{Gy}-(\rho+\sigma+\delta_1+\mu)I_{Ay}\right\},$$

$$I_{S(k+1)}(t)=I_{S0}+\frac{g^{\varphi}}{\Gamma(\varphi+2)}\left\{\rho I_A^n-(\phi+\delta_2+\mu)I_S^n\right\}+$$

$$\frac{g^{\varphi}}{\Gamma(\varphi+2)}\sum_{y=0}^{k} dy,k+1\left\{\rho I_{Ay}-(\phi+\delta_2+\mu)I_{Sy}\right\},$$

$$T_{G(k+1)}(t)=T_{G0}+\frac{g^{\varphi}}{\Gamma(\varphi+2)}\left\{\phi I_S^n+\sigma I_A^n-(\omega+\delta_3+\mu)T_G^n\right\}+$$

$$\frac{g^{\varphi}}{\Gamma(\varphi+2)}\sum_{y=0}^{k} dy,k+1\left\{\phi I_{Sy}+\sigma I_{Ay}-(\omega+\delta_3+\mu)T_{Gy}\right\},$$

$$R_{G(k+1)}(t)=R_{G0}+\frac{g^{\varphi}}{\Gamma(\varphi+2)}\left\{\omega T_G^n-(\psi+\mu)R_G^n\right\}+$$

$$\frac{g^{\varphi}}{\Gamma(\varphi+2)}\sum_{y=0}^{k} dy,k+1\left\{\omega T_{Gy}-(\psi+\mu)\mathrm{R}_{Gy}\right\},$$

$$B_{(k+1)}(t)=B_0+\frac{g^{\varphi}}{\Gamma(\varphi+2)}\left\{\Lambda_b-\mu_b B^n\right\}+$$

$$\frac{g^{\varphi}}{\Gamma(\varphi+2)}\sum_{y=0}^{k} dy,k+1\left\{\Lambda_b-\mu_b B_y\right\}.$$

Where

$$S_{G(k+1)}^n(t)=S_{G0}+\frac{1}{\Gamma(\varphi)}\sum_{y=0}^{k} f_{y,k+1}\left\{\Lambda_h+\psi R_{Gy}-\frac{\left(\phi_1 I_{Ay}+\phi_2 I_{Sy}+\phi_3 T_{Gy}+\phi_4 B_y\right)S_{Gy}}{N_h}-\mu\mathrm{S}_{Gy}\right\},$$

$$E^{n}_{G(k+1)}(t) = E_{G0} + \frac{1}{\Gamma(\varphi)}\sum_{y=0}^{k} f_{y,k+1}\left\{\frac{\left(\phi_1 I_{Ay} + \phi_2 I_{Sy} + \phi_3 T_{Gy} + \phi_4 B_y\right)S_{Gy}}{N_{hy}} - (\alpha+\mu)\mathrm{E}_{Gy}\right\},$$

$$I^{n}_{A(k+1)}(t) = I_{A0} + \frac{1}{\Gamma(\varphi)}\sum_{y=0}^{k} f_{y,k+1}\left\{\alpha E_{Gy} - (\rho+\sigma+\delta_1+\mu)I_{Ay}\right\},$$

(20)

$$I^{n}_{S(k+1)}(t) = I_{S0} + \frac{1}{\Gamma(\varphi)}\sum_{y=0}^{k} f_{y,k+1}\left\{\rho I_{Ay} - (\phi+\delta_2+\mu)I_{Sy}\right\},$$

$$T^{n}_{G(k+1)}(t) = T_{G0} + \frac{1}{\Gamma(\varphi)}\sum_{y=0}^{k} f_{y,k+1}\left\{\phi I_{Sy} + \sigma I_{Ay} - (\omega+\delta_3+\mu)T_{Gy}\right\},$$

$$R^{n}_{G(k+1)}(t) = R_{G0} + \frac{1}{\Gamma(\varphi)}\sum_{y=0}^{k} f_{y,k+1}\left\{\omega T_{Gy} - (\psi+\mu)\mathrm{R}_{Gy}\right\},$$

$$B^{n}_{(k+1)}(t) = B_0 + \frac{1}{\Gamma(\varphi)}\sum_{y=0}^{k} f_{y,k+1}\left\{\Lambda_b - \mu_b B_y\right\}.$$

From (19) and (20) we have:

$$dy,_{K+1} = K^{\varphi+1} - (k-\varphi)(k+\varphi)^{\varphi},\ y = 0,$$

$$(k-y+2)^{\varphi+1} + (k-\varphi)^{\varphi+1} - 2(k-y+1)^{\varphi+1},\ 1 \le y \le k,$$

and $f_{y,k+1} = \frac{g^{\varphi}}{\varphi}\left[(k-y+1)^{\varphi}(k-y)^{\varphi}\right],\ 0 \le y \le k.$

Fractional Order Gonorrhea Model Simulation

This section presents the numerical simulation of the Gonorrhea model using the parameter values presented in table 2 below

Table 2. Parameter Values used for simulations

Parameter	Value	Source
Λ_h	0.8	[17]
μ	0.02	[17]
α	0.60	[17]
ρ	0.50	[18]
ϕ	0.45	[18]
σ	0.10	[19]
ω	0.50	[17]
ψ	0.15	[17]

δ_1	0.004	Assumed
δ_2	0.006	[15]
δ_3	0.002	Assumed
Λ_b	50	Assumed
μ_b	0.25	[20]
ϕ_1	0.30	[20]
ϕ_2	0.45	[20]
ϕ_3	0.08	[17]
ϕ_4	0.20	Assumed

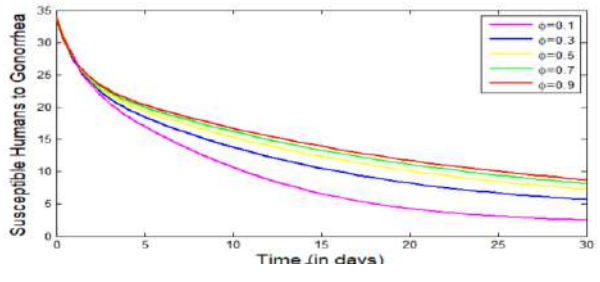


Figure 2a: Simulation of the effect of ϕ on susceptible humans to Gonorrhea

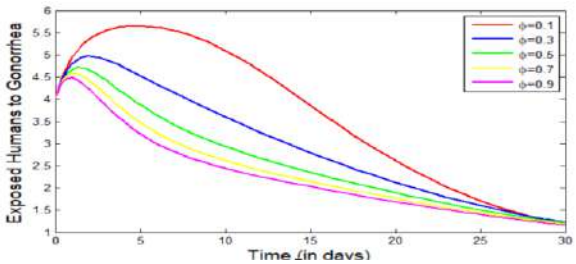


Figure 2b: Simulation of the effect of ϕ on exposed humans to Gonorrhea

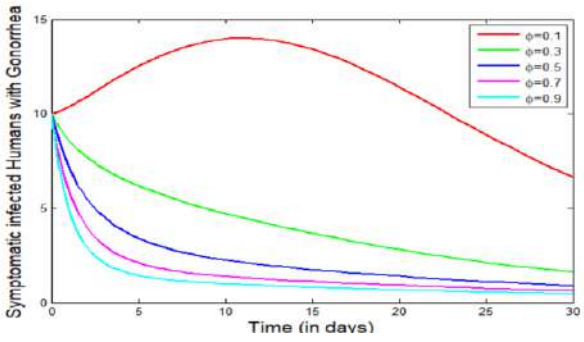


Figure 3a: Simulation of the effect of ϕ on infected humans with Gonorrhea

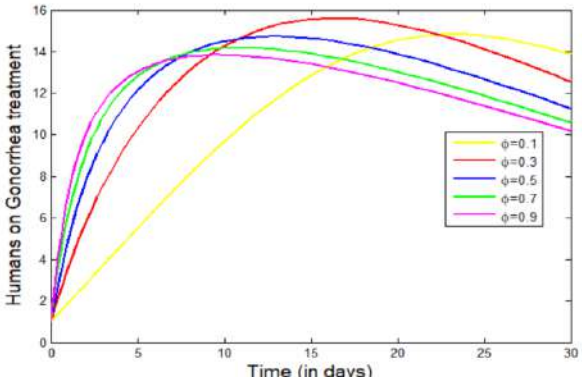
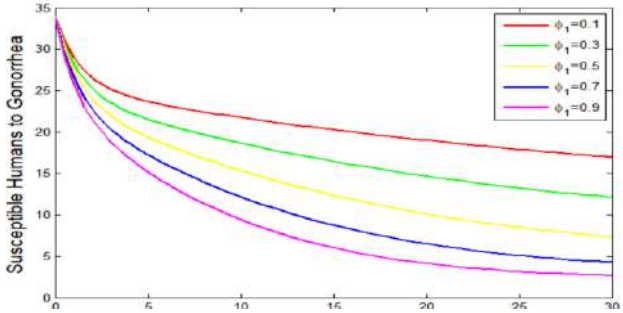


Figure 3b: Simulation of the effect of ϕ on Gonorrhea treatment



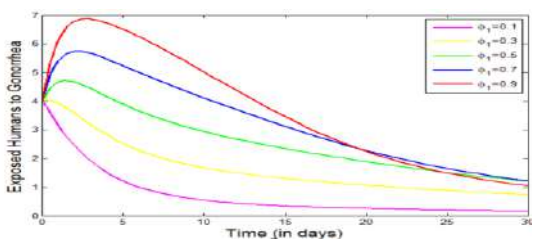
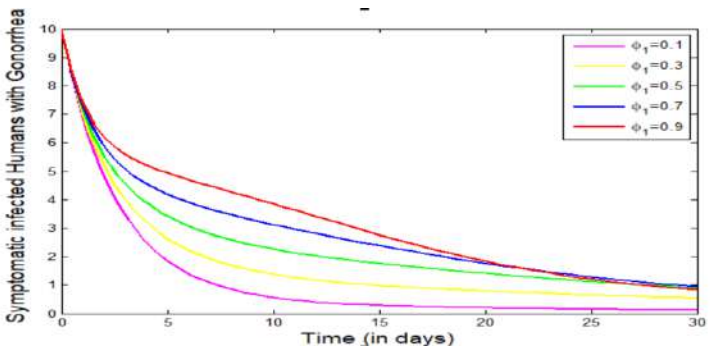


Figure 4a: Simulation of the effect of ϕ_1 on susceptible humans to Gonorrhea

Figure 4b: Simulation of the effect of ϕ_1 on exposed humans to Gonorrhea



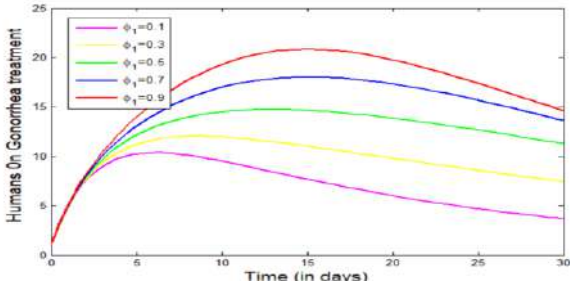


Figure 5a: Simulation of the effect of ϕ_1 on susceptible humans to Gonorrhea

Figure 5b: Simulation of the effect of ϕ_1 on symptomatic infected humans with Gonorrhea

Figure 2a shows the simulation of the effect of treatment rate ϕ on human population susceptible to Gonorrhea. It is observed that as the treatment rate ϕ increases, human population susceptible to Gonorrhea increases over time. Figure 2b depicts the simulation of the effect of treatment rate ϕ on human population exposed to Gonorrhea. It is observed that as the treatment rate ϕ increases, human population exposed to Gonorrhea decreases over time. Figure 3a depicts the simulation of the effect of treatment rate ϕ on human population symptomatic infected with Gonorrhea. It is observed that as the treatment rate ϕ increases, human population infected with Gonorrhea decreases over time. Figure 3b displays the simulation of the effect of treatment rate ϕ on human population on Gonorrhea treatment. It is observed that as the treatment rate ϕ increases, human population on Gonorrhea treatment increases over time. Figure 4a represents the simulation of the effect of contact rate ϕ_1 on human population susceptible to Gonorrhea. It is observed that as the contact rate ϕ_1 increases, human population susceptible to Gonorrhea increases over time. Figure 4b displays the simulation of the effect of contact rate ϕ_1 on human population exposed to Gonorrhea. It is observed that as the contact rate ϕ_1 increases, human population exposed to Gonorrhea increases over time. Figure 5a depicts the simulation of the effect of contact rate ϕ_1 on symptomatic infected human population with Gonorrhea. It is observed that as the contact rate ϕ_1 increases, symptomatic infected human population with Gonorrhea increases over time. Figure 5b shows the simulation of the effect of contact rate ϕ_1 on human population on Gonorrhea treatment. It is observed that as the contact rate ϕ_1 increases, human population on Gonorrhea treatment increases over time.

Conclusion

In this study, a fractional-order mathematical model for the transmission dynamics of Gonorrhea was successfully developed and analyzed to examine the effects of treatment and contact rates on disease progression. The analytical results established the existence and uniqueness of solutions, confirming the well-posedness and reliability of the model. Furthermore, the stability analysis demonstrated that the disease-free equilibrium is attainable when the basic reproduction number is less than unity, emphasizing its critical role as a threshold parameter for disease control. The findings revealed that effective and sustained treatment strategies, particularly for exposed and infected individuals, significantly reduce disease transmission and overall burden. Conversely, increased contact rates were shown to accelerate the spread of infection, leading to higher exposure and infection levels within the population. The numerical simulations further highlighted the importance of incorporating memory effects through fractional-order modeling, which provides a more realistic representation of disease dynamics over time. The study revealed the necessity of a comprehensive control strategy that combines high treatment coverage, possible vaccination efforts, and reduction in risky contact behaviors. Failure to maintain adequate treatment levels or continued high-risk interactions may allow the disease to persist within the population. Therefore, an integrated approach involving improved access to treatment, public health awareness, and behavioral interventions is essential for effectively controlling and potentially eradicating Gonorrhea.

Based on the findings of this study, the following recommendations are proposed:

i. Public health education programs should be intensified to reduce high-risk behaviors that contribute to increased contact rates and the transmission of Gonorrhea. Such programs should emphasize awareness, prevention strategies, and responsible sexual practices.
ii. Early detection and prompt treatment of infected individuals should be strongly encouraged in order to minimize disease spread and prevent further complications.
iii. Healthcare systems should be strengthened and adequately equipped to ensure the provision of effective, affordable, and accessible treatment services for all individuals.
iv. Routine screening programs should be implemented, particularly targeting high-risk populations, to facilitate early diagnosis and timely intervention.
v. Policies and interventions aimed at improving personal hygiene practices and reducing transmission pathways should be developed and strictly enforced.

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