# Fractional –order analysis of malaria and tuberculosis co-dynamics: A Laplace Adomian decomposition approach

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Abstract: Malaria and tuberculosis (TB) remain two of the world's most persistent infectious diseases, often coexisting in regions with limited healthcare resources. Co-infection with both diseases poses significant challenges for diagnosis, treatment, and control, as the interaction between them can complicate disease progression and outcomes. In this study, we develop a fractional-order mathematical model to better understand the transmission dynamics of malaria-TB co-infection within a human population. The model captures the complex interplay between the two diseases through a detailed compartmental framework and introduces fractional calculus to more accurately reflect memory and hereditary properties in disease spread. To solve the model, we apply the Laplace-Adomian Decomposition Method, which provides approximate analytical solutions in the form of a rapidly converging series. Key epidemiological parameters are estimated by fitting the model to real-world malaria and TB data using MATLAB's fmincon optimization algorithm. The strength of this approach lies in its ability to combine mathematical rigor with real data to uncover meaningful trends and relationships. Our findings indicate that enhancing treatment coverage and effectiveness has a significant impact on reducing the burden of both infections. The study emphasizes the importance of integrated disease management strategies, particularly in regions where co-infection is prevalent. By providing a more nuanced view of malaria and TB co-infection dynamics, this work offers valuable insights for policymakers and public health practitioners aiming to design more effective intervention programs.

*Keywords:* Data fitting, Fractional-order model, Laplace-Adomian decomposition, Malaria/tuberculosis (TB) co-infection, Mathematical modelling.

### 1. Introduction

Malaria is a life-threatening disease caused by *Plasmodium parasites*, which are transmitted to humans through the bites of infected *Anopheles mosquitoes*. It remains a major global health issue, especially in sub-Saharan Africa, Asia, and parts of Latin America, where environmental conditions support the breeding of the *Anopheles vector*. The World Health Organization (WHO) estimates that in 2022, there were 247 million cases of malaria worldwide, resulting in approximately 619,000 deaths, with the majority of these occurring in children under five years of age in Africa [1, 2]. The disease manifests in symptoms such as fever, chills, headache, and in severe cases, anemia, organ failure, and death. The two most common species of *Plasmodium* responsible for malaria in humans are *Plasmodium falciparum* and *Plasmodium vivax*, with *P. falciparum* being responsible for the most severe cases. The

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primary mode of prevention and control for malaria includes the use of insecticide-treated nets (ITNs), indoor residual spraying, and chemoprevention with antimalarial drugs. However, the rise of *Plasmodium* strains resistant to antimalarial drugs, particularly *P. falciparum*, poses a significant threat to malaria control efforts. The emergence of resistance to first-line drugs such as artemisinin is a pressing concern, as it could undermine the global progress made in reducing malaria incidence and mortality [3]. Malaria prevention also heavily relies on environmental management strategies, such as eliminating mosquito breeding sites, which require coordinated public health interventions. Despite substantial progress in malaria control, including a 30% decrease in global malaria mortality since 2000, challenges such as drug resistance, inadequate health infrastructure, and climate change continue to complicate efforts toward eradication [4].

Tuberculosis (TB) is an infectious disease caused by Mycobacterium tuberculosis that primarily affects the lungs but can also spread to other parts of the body. TB remains one of the top 10 causes of death worldwide, with approximately 10 million new cases and 1.5 million deaths reported annually [6]. TB is spread through the air when an infected person coughs, sneezes, or talks, and it disproportionately affects low- and middle-income countries where overcrowded living conditions and inadequate healthcare access are common. The disease manifests with symptoms such as persistent cough, chest pain, fever, night sweats, and weight loss. While TB is curable with a six-month regimen of antibiotics, challenges such as multidrug-resistant tuberculosis (MDR-TB) and extensively drugresistant tuberculosis (XDR-TB) have made treatment more complex and prolonged  $\lceil 5 \rceil$ . The emergence of drug-resistant strains of TB, particularly in regions with high TB burden, is a major challenge to global TB control efforts. Effective TB control strategies include early diagnosis, proper antibiotic treatment, and public healt interventions, such as contact tracing and vaccination with the BCG vaccine. However, the global fight against TB is hampered by factors such as delayed diagnosis, poor adherence to treatment regimens, and socio-economic conditions that increase the risk of infection. Co-infection with HIV has also been a significant factor contributing to the high incidence of TB, as immune-compromised individuals are more susceptible to the disease. Advances in diagnostic tools, such as the GeneXpert, have improved TB detection, but ensuring access to treatment remains a significant hurdle in many parts of the world [6]. Despite these challenges, progress has been made in reducing the global burden of TB, and there are ongoing efforts to develop better treatments, diagnostic methods, and vaccines to improve TB control globally.

The co-infection of malaria and tuberculosis (TB) has become an increasingly important area of concern in regions where both diseases are endemic. Co-infection of malaria and TB presents a dual burden on public health systems, exacerbating the severity of both diseases. Co-infected individuals face an elevated risk of severe complications and death, as both diseases can impair immune function and increase vulnerability to the other. Malaria has been shown to impact the immune system, making individuals more susceptible to TB infection, while TB can also enhance malaria susceptibility by altering immune responses [7]. Co-infection complicates diagnosis and treatment, as the symptoms of the two diseases often overlap, leading to delays in diagnosis and treatment initiation. The interaction between malaria and TB is complex, and both diseases can worsen the progression of the other. For example, Plasmodium infection can suppress immune responses, which may lead to the reactivation of latent TB, while the presence of active TB can increase the risk of malaria due to immune suppression and the impact on hemoglobin levels  $\lceil 8 \rceil$ . Additionally, the presence of both diseases can affect the efficacy of treatments, as TB medications, particularly rifampicin, may interact with antimalarial drugs, leading to reduced drug effectiveness and the potential for treatment failure [1]. This drug-drug interaction necessitates careful management to ensure that patients receive effective treatment for both diseases. In regions with a high burden of both malaria and TB, integrated approaches to disease management are crucial to controlling the dual epidemic. Integrated programs aim to provide comprehensive care that addresses both diseases simultaneously, improving outcomes for co-infected individuals. For example, combining malaria vector control measures, such as insecticide-treated bed nets, with TB case finding and treatment, can reduce the incidence of both diseases [9]. Furthermore,

addressing the social determinants of health, such as poverty, malnutrition, and inadequate access to healthcare, is essential for reducing the impact of both diseases. The development of combined treatment strategies, better diagnostic tools, and vaccines for both malaria and TB could potentially lead to more efficient control of these diseases.

The Laplace-Adomian Decomposition Method (LADM) is an effective technique used to solve complex nonlinear differential equations, particularly those involving fractional-order derivatives. It combines the strengths of the Laplace transform, which simplifies the problem by converting it into an algebraic form, with the Adomian decomposition method, which decomposes the solution into a series of terms. This method is especially useful in obtaining approximate analytical solutions for problems where traditional methods may fail or be computationally expensive [10]. By applying LADM, researchers can derive solutions that converge to the exact values, providing both accuracy and efficiency in modeling complex systems like disease transmission dynamics [11]. The method is widely used in various fields, including physics, engineering, and epidemiology, due to its ability to handle both linear and nonlinear problems effectively. The aim of this study is to develop and analyze a fractionalorder compartmental model to explore the co-dynamics of malaria and tuberculosis (TB) transmission within a population, utilizing the Laplace-Adomian Decomposition Method (LADM) to obtain approximate analytical solutions. The primary objectives are to formulate a fractional-order model that captures the interactions between malaria and TB, solve the model using LADM to derive solutions that converge to the exact values, estimate key model parameters through data fitting using real-world disease data, assess the impact of various disease control strategies on transmission dynamics, and evaluate the public health implications of malaria and TB co-infection. This approach aims to provide insights into effective interventions for controlling these diseases and their co-occurrence in affected populations.

Here is a detailed discussion of four recent works on mathematical modeling of malaria and tuberculosis (TB) co-dynamics:

Saha, et al. [12] developed a deterministic compartmental model to explore the co-infection dynamics of malaria and TB. They introduced a variety of human compartments and examined both diseases' progression through the population. Their findings emphasized that combined control strategies, such as integrated treatment and vaccination programs, could significantly reduce both diseases' prevalence. The study also highlighted the importance of considering co-infection's complex dynamics when planning public health interventions. Alzahrani and Khan [13] proposed a fractionalorder model that accounts for the biological complexities in malaria and TB co-infection. Their model used fractional calculus to represent memory effects and time delays in disease dynamics, offering a more realistic depiction of infection transmission. The study concluded that the best strategies for controlling both diseases involve optimized treatment and vaccination programs, which could effectively reduce the burden of malaria and TB in the population. Manogaran, et al. [14] focused on the effect of treatment adherence and vector control measures in the co-infection dynamics of malaria and TB. Their model incorporated different human compartments and emphasized the importance of sustained treatment adherence to control the spread of both diseases. The study found that improving vector control, alongside enhanced treatment regimes, could effectively reduce the incidence of co-infection, particularly when both diseases were managed simultaneously. Siddique, et al.  $\lceil 15 \rceil$  focused on optimal control theory to analyze the co-dynamics of malaria and TB. They modeled various intervention strategies, including vaccination, vector control, and treatment. Their results showed that combined interventions were far more effective than isolated strategies. The study highlighted that the strategic allocation of resources to optimize intervention could lead to more significant reductions in co-infection rates and improve overall public health outcomes

In Jan, et al. [16] the authors developed a fractional-order mathematical model to investigate the dynamics of COVID-19, incorporating the influence of vaccination. They explored fundamental model characteristics and identified threshold values crucial for determining system stability. The research underscored the significant roles played by asymptomatic individuals, waning immunity, and

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vaccination rates in managing the pandemic, offering key insights for public health planning. In Yadav, et al. [17] a fractional-order model for diabetes mellitus was proposed using the Atangana-Baleanu-Caputo (ABC) derivative to capture the disease's memory-dependent behavior and enhance simulation accuracy. However, the model was limited by its omission of hereditary factors, and the practical benefits of the ABC derivative were not fully examined. Moreover, the suggested extension to study the coexistence of diabetes and tuberculosis lacked illustrative examples to support its applicability. Yaday, et al. [18] presented an in-depth investigation of an Ebola virus model based on the ABC fractional derivative with a Mittag-Leffler kernel. This study highlighted the strengths of non-local differential operators in reflecting the intricate dynamics of Ebola transmission. Using the Picard-Lindelöf theorem, the authors demonstrated the existence and uniqueness of solutions, and introduced a numerical approximation method that combined fractional calculus with two-step Lagrange interpolation. Their simulations revealed how varying control parameters affected the model's behavior across different fractional orders, proving the superiority of fractional approaches over traditional integer-order models in capturing disease dynamics. In Peter, et al. [19] a deterministic framework was formulated to examine the spread of the Monkeypox virus (MPXV), establishing conditions for both local and global stability in disease-free and endemic scenarios. The model also revealed the possibility of backward bifurcation, where a stable disease-free equilibrium can coexist with an endemic state. Numerical analyses validated these theoretical results and emphasized the effectiveness of isolating infected individuals in curbing transmission. Other useful studies include [20-27].

#### 2. Model Formulation

A deterministic compartmental model on the transmission dynamics of the co-infection of malaria and Tuberculosis is been proposed. We sub-divided the total human population  $N_{H}(t)$ , into thirteen (13) distinct classes of susceptible humans  $S_H$ , Exposed humans to malaria only  $E_M$ , exposed humans to TB only  $E_T$ , co-exposed humans to Malaria and TB  $E_{MT}$ , Infected humans with malaria only  $I_M$ , acutely infected humans with TB only  $A_{T}$ , chronically infected humans with TB only  $C_{T}$ , co-infected humans with acute TB and malaria  $I_{\rm AM}$ , co-infected humans with chronic TB and Malaria  $I_{\rm CM}$ , Treated humans due to malaria only  $T_M$ , Treated humans due to TB only  $T_T$ , Treated humans due to chronic TB and malaria co-infection  $T_{CM}$  and Recovered humans R from these diseases. We further sub-divided the mosquito vector population into three (3) distinct classes of susceptible vectors  $S_V$ , exposed vectors  $E_V$  and infected vectors  $I_V$ . The recruitment rate of humans into the susceptible population is at the rate of  $\Lambda_H$ , so that  $\beta_M$  is the effective contact rate with the probability of infection per contact with an infected mosquito with malaria. Similarly,  $\beta_T$  is the effective contact rate with the probability of infection per contact with infected human with TB. The rate at which an individual exposed to malaria becomes co-exposed with TB is given as  $K_M$ , the rate at which an individual exposed to TB only becomes co-exposed to malaria is given as  $K_{T}$ . The rate at which individuals exposed to malaria only, TB only and malaria - TB progresses into their respective infected classes due to the high infectiousness of these diseases is given as  $\theta_M, \theta_T, \theta_{MT}$  respectively.  $\mathcal{E}_1, \mathcal{E}_2$  are the modification parameters that accounts for reduced rate of co-infection of malaria with acute TB and acute TB with malaria respectively. au is the rate at which an acutely infected human with TB progresses to been chronically infected with TB? Also,  $ho_{\rm AM}$  is the rate at which an individual co-infected with acute TB and malaria progresses to become co-infected with chronic TB and malaria.  $\mathcal{E}_3, \mathcal{E}_4$  are the modification parameters that accounts for reduced rate of malaria co-infection of chronic TB individual and chronic TB co-infection with malaria respectively. The treatment rate of humans infected with malaria only, acute Tb only, chronic TB only, co-infected malaria – TB only is given as  $\gamma_M, \gamma_T, \gamma_C, \gamma_{CM}$ receptively. The rate of recovery of humans from the malaria only, TB only and co-infected malaria – TB classes is given at the rate of  $\alpha_M, \alpha_T, \alpha_{CM}$ . The rate at which a recovered individual from malaria becomes susceptible to it again is given as  $\omega_M$ . The natural death rate of humans is given as  $\mu_H$ . The disease induced death rates of humans due to malaria only, TB only, acute TB only, chronic TB only, coinfected humans with acute TB and malaria, co-infected humans with chronic TB and malaria is given as  $\delta_M, \delta_T, \delta_{CM}, \delta_{AM}$  respectively. The rate of compliance to the usage of treated bed nets so as to reduce

the burden of malaria is given as  $\phi$ . The biting rate of the malaria causing mosquitoes per time is denoted by m.

Also, the recruitment rate of the malaria causing anopheles mosquitoes is given as  $\Lambda_V$ , so that  $\beta_V$  is the effective contact rate with the probability of infection per contact with an infected human through biting. The exposed mosquito vectors thus progress to been infected at the rate of  $\theta_V$ . The natural death rate of the mosquito vectors is given as  $\mu_V$  and the disease induced death rate of the infected malaria vectors is given as  $\delta_V$ .

# $\omega_M$



Figure 1. Schematic diagram for the model.

# 2.1. Model Assumptions

The assumptions used in the formulation of the model are:

- No transmission of the infections occurs vertically from mother to unborn child [28].
- There is homogeneous mixing, meaning that all susceptible individuals are equally at risk of infection upon contact with those who are infectious.
- Disease-induced fatalities occur exclusively in the infectious compartments, with a consistent natural death rate across all compartments.
- The rodent population can spread only tuberculosis (TB) and not malaria [29].
- Individuals who have recovered from malaria or tuberculosis can become susceptible to the diseases once more [30].

# 2.2. The Model Equations

The differential equations that describe the above illustrations are.

$$\frac{dS_H}{dt} = \Lambda_H - \left(\lambda_M + \lambda_T + \mu_H\right)S_H + \omega_M R$$

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$$\frac{dE_{M}}{dt} = \lambda_{M}S_{H} - (\theta_{M} + K_{M} + \mu_{H})E_{M}$$

$$\frac{dE_{T}}{dt} = \lambda_{T}S_{H} - (\theta_{T} + K_{T} + \mu_{H})E_{T}$$

$$\frac{dE_{MT}}{dt} = K_{M}E_{M} + K_{T}E_{T} - (\theta_{MT} + \mu_{H})E_{MT}$$

$$\frac{dI_{M}}{dt} = \theta_{M}E_{M} - (\varepsilon_{1}\lambda_{T} + \varepsilon_{3}\lambda_{T} + \gamma_{M} + \delta_{M} + \mu_{H})I_{M}$$

$$\frac{dA_{T}}{dt} = \theta_{T}E_{T} - (\varepsilon_{2}\lambda_{M} + \tau + \gamma_{T} + \delta_{T} + \mu_{H})A_{T}$$

$$\frac{dC_{T}}{dt} = \tau A_{T} - (\varepsilon_{4}\lambda_{M} + \gamma_{c} + \delta_{T} + \mu_{H})C_{T}$$

$$\frac{dI_{AM}}{dt} = \theta_{MT}E_{MT} + \varepsilon_{1}\lambda_{T}I_{M} + \varepsilon_{2}\lambda_{M}A_{T} - (\rho_{AM} + \delta_{AM} + \mu_{H})I_{AM}$$

$$\frac{dI_{CM}}{dt} = \varepsilon_{3}\lambda_{T}I_{M} + \varepsilon_{4}\lambda_{M}C_{T} + \rho_{AM}I_{AM} - (\gamma_{CM} + \delta_{CM} + \mu_{H})I_{CM}$$

$$\frac{dT_{M}}{dt} = \gamma_{T}A_{T} + \gamma_{C}C_{T} - (\alpha_{T} + \delta_{T} + \mu_{H})T_{T}$$

$$\frac{dT_{T}}{dt} = \gamma_{CM}I_{CM} - (\alpha_{CM} + \delta_{CM} + \mu_{H})T_{CM}$$

$$\frac{dT_{R}}{dt} = \alpha_{M}T_{M} + \alpha_{T}T_{T} + \alpha_{CM}T_{CM} - (\omega_{M} + \mu_{H})R$$

$$\frac{dS_{V}}{dt} = \lambda_{V} - (\lambda_{V} + \mu_{V})S_{V}$$

$$\frac{dE_{V}}{dt} = \delta_{V}E_{V} - (\delta_{V} + \mu_{V})I_{V}$$
The form of the triangle of the triangle

The force of infection for the malaria disease transmission in the human population is given as:

$$\lambda_{M} = \frac{(1-\phi)m\beta_{M}I_{V}}{N_{H}}, \ \lambda_{T} = \frac{\beta_{T}(A_{T}+C_{T}+I_{AM}+I_{CM})}{N_{H}}, \ \lambda_{V} = \frac{m\beta_{V}(I_{M}+I_{AM}+I_{CM})}{N_{H}}$$

## 2.3. Description of Variables and Model parameters

Table 1.

Variables and parameters used in the model formulation Variable Description Susceptible Humans  $S_{H}$ Exposed Humans to Malaria only  $E_M$ Exposed Humans to TB only  $E_T$  $E_{MT}$ Co-exposed humans to malaria and TB Infected humans with malaria only  $I_{M}$ Acutely infected humans with TB only  $A_T$ Chronically infected humans with TB only.  $C_T$ Co-infected humans with malaria and acute TB  $I_{\underline{AM}}$ Co-infected humans with chronic TB and malaria  $I_{CM}$ Treated Humans due to malaria only  $T_M$ Treated Humans to TB only  $T_T$ Treated humans due to TB and malaria  $T_{CM}$ **Recovered Humans** R Susceptible vectors  $S_{V}$ Exposed Vectors  $E_{V}$ Infected Vectors  $I_V$ 

Parameter	Description
$\Lambda_{H}$	Recruitment rate of humans
$\beta_{_M}$	Contact of susceptible humans and infected mosquitoes
$\beta_T$	The effective contact rate with the probability of infection per contact with infected human with TB
m	Biting ate of vectors
K <sub>M</sub>	Co-exposure rate of malaria with TB
K <sub>T</sub>	Co-exposure rate of TB with malaria
$\theta_{_M}$	Progression rate of exposed malaria to infected malaria class
$\theta_{T}$	Progression rate of exposed TB to acutely infected TB class
$\theta_{_{MT}}$	Progression rate from co-exposed malaria – TB class into co-infected acute TB – malaria class
$\mu_{H}$	Natural death rate of humans
$\mu_{v}$	Natural death rate of mosquito vectors
$\varepsilon_i(i=1,2,3,4)$	Modification parameters that account for reduced rate of co-infections
$\gamma_M, \gamma_T, \gamma_{CM}$	Treatment rate of infected humans with malaria only, TB and malaria – TB co- infection respectively.

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1000

0	Progression rate from co-infected Acute TB - malaria into chronic TB- malaria co-
PAM	infection.
τ	Progression rate from acute TB to chronic TB class
8888	Disease induced death rate of infected malaria, TB, acute TB - malaria, chronic TB -
$\mathcal{O}_M, \mathcal{O}_T, \mathcal{O}_{AM}, \mathcal{O}_{CM}$	Malaria classes respectively.
aaa	Recovery rate of individuals in malaria only, TB only and TB- malaria co-infection
$\alpha_{_M}, \alpha_{_T}, \alpha_{_{CM}}$	individuals in their respective treatment classes.
$\omega_{_M}$	Re-infection rate of recovered malaria individuals.
$\Lambda_V$	Recruitment rate of malaria vectors
$\beta_{V}$	Contact rate susceptible mosquitoes and infected humans with malaria
$\theta_{V}$	Progression rate from exposed to infected vector classes.
$\delta_{V}$	Disease induced death of malaria vectors.
$\phi$	Rate of compliance to the usage of treated bed nets.

## 3. Fractional Order of the Malaria-TB Model

The Caputo derivative is measured as a differential operator in our model. We present in this segment some well-known definitions and effects that we shall be using throughout this research.

Definition 1 The Caputo fractional order derivative of a function (f) on the interval [0,T] is defined by:

$$\left[{}^{C}D_{0}^{\beta}f(t)\right] = \frac{1}{\Gamma(n-\beta)} \int_{0}^{t} (t-s)^{n-\beta-1} f^{(n)}(s) ds,$$
<sup>(2)</sup>

Where  $n = [\beta] + 1$  and  $[\beta]$  represents the integer part of  $\beta$ . In particular, for  $0 < \beta < 1$ , the Caputo derivative becomes:

$$\left[{}^{C}D_{0}^{\beta}f(t)\right] = \frac{1}{\Gamma(1-\beta)} \int_{0}^{t} \frac{f(s)}{(t-s)^{\beta}} ds,$$
(3)

Definition 2 Laplace transform of Caputo derivatives is defined as

$$\mathcal{L}[{}^{C}D^{\beta}q(t)] = S^{\beta}h(S) - \sum_{K=0}^{n} S^{\beta-i-1}y^{k}(0), \quad n-1 < \beta < n, \ n \in N,$$
(4)

For arbitrary  $c_i \in R, i = 0, 1, 2, ..., n-1$ ,  $n = \lfloor \beta \rfloor + 1$  and  $\lfloor \beta \rfloor$  represents the non-integer part of  $\beta$ . Lemma 1. [26] The following results hold for fractional differentiation equations

$$I^{\beta}[^{c}D^{\beta}h](t) = h(t) + \sum_{i=0}^{n-1} \frac{h^{(i)}(0)}{i!} t^{i},$$
(5)

For arbitrary  $\beta > 0, i = 0, 1, 2, ..., n-1$ , where  $n = \lfloor \beta \rfloor + 1$  and  $\lfloor \beta \rfloor$  represents the integer part of  $\beta$  Introducing fractional-order into the model, we now present a new model described by the following Introducing fractional order derivative into the model we present new mathematical model describe by set of fractional difference of order  $\beta$  for  $0 < \beta < 1$ .

$$D^{\beta}(S_{H}) = \Lambda_{H} + \omega_{M}R - (\lambda_{M} + \lambda_{T} + \mu_{H})S_{H},$$

$$D^{\beta}(E_{M}) = \lambda_{M}S_{H} - (\theta_{H} + K_{M} + \mu_{H})E_{M},$$

$$D^{\beta}(E_{T}) = \lambda_{T}S_{H} - (\theta_{T} + K_{T} + \mu_{H})E_{T},$$

$$D^{\beta}(E_{MT}) = K_{M}E_{M} + K_{T}E_{T} - (\theta_{MT} + \mu_{H})E_{MT},$$

$$D^{\beta}(I_{M}) = \theta_{M}E_{M} - (\varepsilon_{1}\lambda_{T} + \varepsilon_{3}\lambda_{T} + \gamma_{M} + \delta_{M} + \mu_{H})I_{M},$$

$$D^{\beta}(A_{T}) = \theta_{T}E_{T} - (\varepsilon_{2}\lambda_{M} + \tau + \gamma_{T} + \delta_{T} + \mu_{H})A_{T},$$

$$D^{\beta}(C_{T}) = \tau A_{T} - (\varepsilon_{4}\lambda_{M} + \gamma_{c} + \delta_{T} + \mu_{H})C_{T},$$

$$D^{\beta}(I_{AM}) = \theta_{MT}E_{MT} + \varepsilon_{1}\lambda_{T}I_{M} + \varepsilon_{2}\lambda_{M}A_{T} - (\rho_{AM} + \delta_{AM} + \mu_{H})I_{AM},$$

$$D^{\beta}(I_{CM}) = \varepsilon_{3}\lambda_{T}I_{M} + \varepsilon_{4}\lambda_{M}C_{T} + \rho_{AM}I_{AM} - (\gamma_{CM} + \delta_{CM} + \mu_{H})I_{CM},$$

$$D^{\beta}(T_{M}) = \gamma_{M}I_{M} - (\alpha_{M} + \delta_{M} + \mu_{H})T_{M}.$$

$$D^{\beta}(T_{T}) = \gamma_{T}A_{T} + \gamma_{C}C_{T} - (\alpha_{T} + \delta_{T} + \mu_{H})T_{T}$$

$$D^{\beta}(R) = \alpha_{M}T_{M} + \alpha_{T}T_{T} + \alpha_{CM}T_{CM} - (\omega_{M} + \mu_{H})R$$

$$D^{\beta}(S_{V}) = \Lambda_{V} - (\lambda_{V} + \mu_{V})S_{V}$$

$$D^{\beta}(E_{V}) = \lambda_{V}S_{V} - (\theta_{V} + \mu_{V})I_{V}$$
(6)

## 3.1. The Laplace-Adomian Decomposition Method (LADM) Implementation

We considered the general procedure of this method with the initial conditions. Applying Laplace transforms to both sides of the equation (1), and then we have:

$$\begin{split} S^{\beta} \mathcal{L}(S_{H}) - S^{\beta-1}S_{H}(0) &= \mathcal{L} \Big[ \Lambda_{H} + \omega_{M}R - \left( \lambda_{M} + \lambda_{T} + \mu_{H} \right) S_{H} \Big] \\ S^{\beta} \mathcal{L}(E_{M}) - S^{\beta-1}E_{H}(0) &= \mathcal{L} \Big[ \lambda_{M}S_{H} - \left( \theta_{M} + K_{M} + \mu_{H} \right) E_{M} \Big] \\ S^{\beta} \mathcal{L}(E_{T}) - S^{\beta-1}E_{T}(0) &= \mathcal{L} \Big[ \lambda_{T}S_{H} - \left( \theta_{T} + K_{T} + \mu_{H} \right) E_{T} \Big] \\ S^{\beta} \mathcal{L}(E_{MT}) - S^{\beta-1}E_{MT}(0) &= \mathcal{L} \Big[ \theta_{M}E_{M} - \left( \varepsilon_{1}\lambda_{T} + \varepsilon_{3}\lambda_{T} + \gamma_{M} + \delta_{M} + \mu_{H} \right) I_{M} \Big] \\ S^{\beta} \mathcal{L}(I_{M}) - S^{\beta-1}I_{M}(0) &= \mathcal{L} \Big[ \theta_{T}E_{T} - \left( \varepsilon_{2}\lambda_{M} + \tau + \gamma_{T} + \delta_{T} + \mu_{H} \right) A_{T} \Big] \\ S^{\beta} \mathcal{L}(A_{T}) - S^{\beta-1}I_{C}(0) &= \mathcal{L} \Big[ \tau A_{T} - \left( \varepsilon_{4}\lambda_{M} + \gamma_{c} + \delta_{T} + \mu_{H} \right) A_{T} \Big] \\ S^{\beta} \mathcal{L}(I_{M}) - S^{\beta-1}I_{AM}(0) &= \mathcal{L} \Big[ \theta_{M}TE_{MT} + \varepsilon_{1}\lambda_{T}I_{M} + \varepsilon_{2}\lambda_{M}A_{T} - \left( \rho_{AM} + \delta_{AM} + \mu_{H} \right) I_{AM} \Big] \\ S^{\beta} \mathcal{L}(I_{CM}) - S^{\beta-1}I_{CM}(0) &= \mathcal{L} \Big[ \varepsilon_{3}\lambda_{T}I_{M} + \varepsilon_{4}\lambda_{M}C_{T} + \rho_{AM}I_{AM} - \left( \gamma_{CM} + \delta_{CM} + \mu_{H} \right) I_{CM} \Big] \\ S^{\beta} \mathcal{L}(T_{T}) - S^{\beta-1}T_{C}(0) &= \mathcal{L} \Big[ \gamma_{T}A_{T} + \gamma_{C}C_{T} - \left( \alpha_{T} + \delta_{T} + \mu_{H} \right) T_{T} \Big] \\ S^{\beta} \mathcal{L}(T_{T}) - S^{\beta-1}T_{C}(0) &= \mathcal{L} \Big[ \gamma_{T}M_{T} + \alpha_{C}T_{T} - \left( \alpha_{CM} + \delta_{CM} + \mu_{H} \right) T_{CM} \Big] \\ S^{\beta} \mathcal{L}(S_{V}) - S^{\beta-1}R(0) &= \mathcal{L} \Big[ \gamma_{CM}I_{CM} - \left( \alpha_{CM} + \delta_{CM} + \mu_{H} \right) T_{CM} \Big] \\ S^{\beta} \mathcal{L}(V_{V}) - S^{\beta-1}R(0) &= \mathcal{L} \Big[ \Lambda_{V} - \left( \lambda_{V} + \mu_{V} \right) S_{V} \Big] \\ S^{\beta} \mathcal{L}(V_{V}) - S^{\beta-1}R(0) &= \mathcal{L} \Big[ \Lambda_{V} - \left( \lambda_{V} + \mu_{V} \right) S_{V} \Big] \\ S^{\beta} \mathcal{L}(V_{V}) - S^{\beta-1}I_{V}(0) &= \mathcal{L} \Big[ \Lambda_{V} - \left( \lambda_{V} + \mu_{V} \right) S_{V} \Big] \\ S^{\beta} \mathcal{L}(V_{V}) - S^{\beta-1}I_{V}(0) &= \mathcal{L} \Big[ \lambda_{V} V_{V} - \left( \lambda_{V} + \mu_{V} \right) S_{V} \Big] \\ S^{\beta} \mathcal{L}(V_{V}) - S^{\beta-1}I_{V}(0) &= \mathcal{L} \Big[ \lambda_{V} V_{V} - \left( \lambda_{V} + \mu_{V} \right) S_{V} \Big] \\ S^{\beta} \mathcal{L}(V_{V}) - S^{\beta-1}I_{V}(0) &= \mathcal{L} \Big[ \theta_{V} E_{V} - \left( \delta_{V} + \mu_{V} \right) I_{V} \Big] \end{aligned}$$

With initial conditions

$$\begin{split} S_{H}(0) &= n_{1}, \quad E_{H}(0) = n_{2}, E_{T}(0) = n_{3}, \quad E_{MT}(0) = n_{4}, \quad I_{M}(0) = n_{5}, A_{T}(0) = n_{6}, \quad C_{T}(0) = n_{7}, \\ I_{AM}(0) &= n_{8}, \quad I_{CM}(0) = n_{9}, \quad T_{M}(0) = n_{10}, \\ T_{T}(0) &= n_{11}, \quad T_{CM}(0) = n_{12}, \quad R(0) = n_{13}, \quad S_{V}(0) = n_{14}, \\ E_{V}(0) &= n_{15}, \quad I_{V}(0) = n_{16}, \\ \text{Dividing equation (7) by } (S^{\beta}) \text{ we have:} \end{split}$$

Dividing equation (7) by (5 ) we have

$$\begin{split} \mathcal{L}(S_{H}) &= \frac{n_{1}}{S} + \frac{1}{S^{\beta}} \mathcal{L} \Big[ \Lambda_{H} + \omega_{M} R - \left( \lambda_{M} + \lambda_{T} + \mu_{H} \right) S_{H} \Big] \\ \mathcal{L}(E_{w}) &= \frac{n_{2}}{S} + \frac{1}{S^{\beta}} \mathcal{L} \Big[ \lambda_{M} S_{H} - \left( \theta_{M} + K_{M} + \mu_{H} \right) E_{M} \Big] \\ \mathcal{L}(E_{T}) &= \frac{n_{3}}{S} + \frac{1}{S^{\beta}} \mathcal{L} \Big[ \lambda_{T} S_{H} - \left( \theta_{T} + K_{T} + \mu_{H} \right) E_{T} \Big] \\ \mathcal{L}(E_{MT}) &= \frac{n_{3}}{S} + \frac{1}{S^{\beta}} \mathcal{L} \Big[ K_{M} E_{M} + K_{T} E_{T} - \left( \theta_{MT} + \mu_{H} \right) E_{MT} \Big] \\ \mathcal{L}(I_{M}) &= \frac{n_{5}}{S} + \frac{1}{S^{\beta}} \mathcal{L} \Big[ \theta_{M} E_{M} - \left( \varepsilon_{1} \lambda_{T} + \varepsilon_{3} \lambda_{T} + \gamma_{M} + \delta_{M} + \mu_{H} \right) I_{M} \Big] \\ \mathcal{L}(A_{T}) &= \frac{n_{5}}{S} + \frac{1}{S^{\beta}} \mathcal{L} \Big[ \theta_{T} E_{T} - \left( \varepsilon_{2} \lambda_{M} + \tau + \gamma_{T} + \delta_{T} + \mu_{H} \right) A_{T} \Big] \\ \mathcal{L}(C_{T}) &= \frac{n_{7}}{S} + \frac{1}{S^{\beta}} \mathcal{L} \Big[ \theta_{MT} E_{MT} + \varepsilon_{1} \lambda_{T} I_{M} + \varepsilon_{2} \lambda_{M} A_{T} - \left( \rho_{AM} + \delta_{AM} + \mu_{H} \right) I_{AM} \Big] \\ \mathcal{L}(I_{AM}) &= \frac{n_{5}}{S} + \frac{1}{S^{\beta}} \mathcal{L} \Big[ \varepsilon_{3} \lambda_{T} I_{M} + \varepsilon_{4} \lambda_{M} C_{T} + \rho_{AM} I_{AM} - \left( \gamma_{CM} + \delta_{CM} + \mu_{H} \right) I_{CM} \Big] \\ \mathcal{L}(I_{CM}) &= \frac{n_{9}}{S} + \frac{1}{S^{\beta}} \mathcal{L} \Big[ \gamma_{T} A_{T} + \gamma_{C} C_{T} - \left( \alpha_{T} + \delta_{T} + \mu_{H} \right) T_{M} \Big] \\ \mathcal{L}(T_{T}) &= \frac{n_{11}}{S} + \frac{1}{S^{\beta}} \mathcal{L} \Big[ \gamma_{T} A_{T} + \gamma_{C} C_{T} - \left( \alpha_{T} + \delta_{CM} + \mu_{H} \right) T_{CM} \Big] \\ \mathcal{L}(R) &= \frac{n_{12}}{S} + \frac{1}{S^{\beta}} \mathcal{L} \Big[ \alpha_{M} T_{M} - \left( \alpha_{CM} + \delta_{CM} + \mu_{H} \right) T_{CM} \Big] \\ \mathcal{L}(R) &= \frac{n_{13}}{S} + \frac{1}{S^{\beta}} \mathcal{L} \Big[ \alpha_{M} T_{M} + \alpha_{T} T_{T} + \alpha_{CM} T_{CM} - \left( \omega_{M} + \mu_{H} \right) R \Big] \\ \mathcal{L}(R) &= \frac{n_{13}}{S} + \frac{1}{S^{\beta}} \mathcal{L} \Big[ \Lambda_{V} - \left( \lambda_{V} + \mu_{V} \right) S_{V} \Big] \\ \mathcal{L}(R_{V}) &= \frac{n_{16}}{S} + \frac{1}{S^{\beta}} \mathcal{L} \Big[ \lambda_{V} S_{V} - \left( \theta_{V} + \mu_{V} \right) E_{V} \Big] \\ \mathcal{L}(R_{V}) &= \frac{n_{16}}{S} + \frac{1}{S^{\beta}} \mathcal{L} \Big[ \delta_{V} S_{V} - \left( \delta_{V} + \mu_{V} \right) I_{V} \Big] \end{aligned}$$

$$\tag{8}$$

Decomposing the non-linear term of equation (6) whereby we assume the solution of  $S_H(t), E_M(t), E_T(t), E_{MT}(t), I_M(t), A_T(t), C_T(t), I_{AM}(t), I_{CM}(t), T_M(t), T_T(t), T_{CM}(t), R(t), S_V(t), E_V(t), I_V(t)$  are in the form of infinite series given by:

$$S_{H}(t) = \sum_{n=0}^{\infty} S_{H}(n), \ E_{M}(t) = \sum_{n=0}^{\infty} E_{M}(n), \ E_{T}(t) = \sum_{n=0}^{\infty} E_{T}(n), \ E_{MT}(t) = \sum_{n=0}^{\infty} E_{MT}(n), \ I_{M}(t) = \sum_{n=0}^{\infty} I_{M}(n), \ A_{T}(t) = \sum_{n=0}^{\infty} A_{T}(n), \ C_{T}(t) = \sum_{n=0}^{\infty} C_{T}(n), \ I_{AM}(t) = \sum_{n=0}^{\infty} I_{AM}(n), \ I_{CM}(t) = \sum_{n=0}^{\infty} I_{CM}(n), \ T_{M}(t) = \sum_{n=0}^{\infty} T_{M}(n), \ T_{T}(t) = \sum_{n=0}^{\infty} T_{T}(n), \ T_{CM}(t) = \sum_{n=0}^{\infty} T_{CM}(n), \ R(t) = \sum_{n=0}^{\infty} R(n), \ S_{V}(t) = \sum_{n=0}^{\infty} S_{V}(n), \ E_{V}(t) = \sum_{n=0}^{\infty} E_{V}(n), \ I_{V}(t) = \sum_{n=0}^{\infty} I_{V}(n).$$

We have three (5) non-linear terms. The non-linear term in equation (6) are decomposed by Adomian polynomial as follows:

$$I_{V}(t)S_{H}(t) = \sum_{n=0}^{\infty} A(n), \ A_{T}(t)S_{H}(t) = \sum_{n=0}^{\infty} B(n), \ C_{T}(t)S_{H}(t) = \sum_{n=0}^{\infty} C(n), \ I_{AM}(t)S_{H}(t) = \sum_{n=0}^{\infty} D(n), \ I_{CM}(t)S_{H}(t) = \sum_{n=0}^{\infty} E(n), \ A_{T}(t)I_{M}(t) = \sum_{n=0}^{\infty} F(n), \ C_{T}(t)I_{M}(t) = \sum_{n=0}^{\infty} G(n), \ I_{AM}(t)I_{M}(t) = \sum_{n=0}^{\infty} H(n), \ I_{CM}(t)I_{M}(t) = \sum_{n=0}^{\infty} I(n), \ I_{M}(t)A_{T}(t) = \sum_{n=0}^{\infty} J(n), \ I_{M}(t)C_{T}(t) = \sum_{n=0}^{\infty} K(n), \ I_{M}(t)S_{V}(t) = \sum_{n=0}^{\infty} L(n), \ I_{AM}(t)S_{V}(t) = \sum_{n=0}^{\infty} M(n), \ I_{CM}(t)S_{V}(t) = \sum_{n=0}^{\infty} N(n)$$
(10)

Where

$$A(n), B(n), C(n), D(n), E(n), F(n), G(n), H(n), I(n), J(n), K(n), L(n), M(n), N(n)$$
  
are Adomian polynomials given by

$$\begin{split} &A(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_V(k) \sum_{k=0}^n \lambda^k S_H(k) \right]_{\lambda=0} \\ &B(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k A_T(k) \sum_{k=0}^n \lambda^k S_H(k) \right]_{\lambda=0} \\ &C(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k C_T(k) \sum_{k=0}^n \lambda^k S_H(k) \right]_{\lambda=0} \\ &D(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_{AM}(k) \sum_{k=0}^n \lambda^k S_H(k) \right]_{\lambda=0} \\ &E(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_{CM}(k) \sum_{k=0}^n \lambda^k S_H(k) \right]_{\lambda=0} \\ &F(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_{CM}(k) \sum_{k=0}^n \lambda^k I_M(k) \right]_{\lambda=0} \\ &G(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_{CM}(k) \sum_{k=0}^n \lambda^k I_M(k) \right]_{\lambda=0} \\ &H(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_{AM}(k) \sum_{k=0}^n \lambda^k I_M(k) \right]_{\lambda=0} \\ &I(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_{AM}(k) \sum_{k=0}^n \lambda^k I_M(k) \right]_{\lambda=0} \\ &I(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_M(k) \sum_{k=0}^n \lambda^k I_M(k) \right]_{\lambda=0} \\ &I(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_M(k) \sum_{k=0}^n \lambda^k I_M(k) \right]_{\lambda=0} \\ &L(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_M(k) \sum_{k=0}^n \lambda^k S_V(k) \right]_{\lambda=0} \\ &L(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_M(k) \sum_{k=0}^n \lambda^k S_V(k) \right]_{\lambda=0} \\ &M(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_{AM}(k) \sum_{k=0}^n \lambda^k S_V(k) \right]_{\lambda=0} \\ &M(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_{AM}(k) \sum_{k=0}^n \lambda^k S_V(k) \right]_{\lambda=0} \\ &M(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_{AM}(k) \sum_{k=0}^n \lambda^k S_V(k) \right]_{\lambda=0} \\ &M(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_{AM}(k) \sum_{k=0}^n \lambda^k S_V(k) \right]_{\lambda=0} \\ &M(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_{AM}(k) \sum_{k=0}^n \lambda^k S_V(k) \right]_{\lambda=0} \\ &M(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_{AM}(k) \sum_{k=0}^n \lambda^k S_V(k) \right]_{\lambda=0} \\ &M(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_{AM}(k) \sum_{k=0}^n \lambda^k S_V(k) \right]_{\lambda=0} \\ &M(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_{AM}(k) \sum_{k=0}^n \lambda^k S_V(k) \right]_{\lambda=0} \\ &M(n) = \frac{1}{\Gamma(n+1)} \frac{d^n}{d\lambda^n} \left[ \sum_{k=0}^n \lambda^k I_{AM}(k) \sum_{k=0}^n \lambda^k S_V(k) \right]_{\lambda=0} \\ &M(n) = \frac{1}{\Gamma(n+$$

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(11)

The polynomials are given by

$$\begin{split} A(0) &= I_{V}(0)S_{H}(0), \\ A(1) &= I_{V}(0)S_{H}(1) + I_{V}(1)S_{H}(0), \\ A(2) &= I_{V}(0)S_{H}(2) + I_{V}(1)S_{H}(1) + I_{V}(2)S_{H}(0). \\ B(0) &= A_{T}(0)S_{H}(0), \\ B(1) &= A_{T}(0)S_{H}(1) + A_{T}(1)S_{H}(1) + A_{T}(2)S_{H}(0). \\ C(0) &= C_{T}(0)S_{H}(0), \\ C(1) &= C_{T}(0)S_{H}(1) + C_{T}(1)S_{H}(1) + A_{T}(2)S_{H}(0). \\ C(2) &= C_{T}(0)S_{H}(2) + C_{T}(1)S_{H}(1) + C_{T}(2)S_{H}(0). \\ D(0) &= I_{AH}(0)S_{H}(0), \\ D(1) &= I_{AH}(0)S_{H}(2) + I_{AH}(1)S_{H}(0), \\ E(0) &= I_{CH}(0)S_{H}(2) + I_{AH}(1)S_{H}(0), \\ E(1) &= I_{CH}(0)S_{H}(2) + I_{CH}(1)S_{H}(0), \\ E(2) &= I_{CH}(0)S_{H}(2) + I_{CH}(1)S_{H}(1) + I_{CM}(2)S_{H}(0). \\ F(0) &= A_{T}(0)I_{M}(0), \\ F(1) &= A_{T}(0)I_{M}(0), \\ F(2) &= A_{T}(0)I_{M}(0), \\ G(1) &= C_{T}(0)I_{M}(0), \\ G(1) &= C_{T}(0)I_{M}(0), \\ H(0) &= I_{AH}(0)I_{M}(0), \\ H(1) &= I_{AH}(0)I_{M}(2) + I_{AH}(1)I_{M}(0), \\ H(2) &= I_{AH}(0)I_{M}(0), \\ H(1) &= I_{AH}(0)I_{M}(0), \\ I(1) &= I_{AH}(0)I_{M}(0), \\ I(2) &= I_{CH}(0)I_{M}(0), \\ I(2) &= I_{M}(0)A_{T}(1) + I_{M}(1)A_{T}(0), \\ I(2) &= I_{M}(0)A_{T}(1) + I_{M}(1)A_{T}(0), \\ I(2) &= I_{M}(0)A_{T}(2) + I_{M}(1)A_{T}(1) + I_{M}(2)A_{T}(0). \\ I(2) &= I_{M}(0)A_{T}(2) + I_{M}(1)A_{T}(1) + I_{M}(2)A_{T}(0). \\ I(2) &= I_{M}(0)A_{T}(2) + I_{M}(1)A_{T}(1) + I_{M}(2)A_{T}(0). \\ I(3) &= I_{M}(0)A_{T}(2) + I_{M}(1)A_{T}(1) + I_{M}(2)A_{T}(0). \\ I(3) &= I_{M}(0)A_{T}(2) + I_{M}(1)A_{T}(1) + I_{M}(2)A_{T}(0). \\ I(3) &= I_{M}(0)A_{T}(2) + I_{M}(1)A_{T}(0), \\ I(3) &= I_{M}(0)A$$

$$\begin{split} &K(0) = I_{M}(0)C_{T}(0), \\ &K(1) = I_{M}(0)C_{T}(1) + I_{M}(1)C_{T}(0), \\ &K(2) = I_{M}(0)C_{T}(2) + I_{M}(1)C_{T}(1) + I_{M}(2)C_{T}(0). \\ &L(0) = I_{M}(0)S_{V}(0), \\ &L(1) = I_{M}(0)S_{V}(1) + I_{M}(1)S_{V}(0), \\ &L(2) = I_{M}(0)S_{V}(2) + I_{M}(1)S_{V}(1) + I_{M}(2)S_{V}(0). \\ &M(0) = I_{AM}(0)S_{V}(0), \\ &M(1) = I_{AM}(0)S_{V}(1) + I_{AM}(1)S_{V}(0), \\ &M(2) = I_{AM}(0)S_{V}(2) + I_{AM}(1)S_{V}(1) + I_{AM}(2)S_{V}(0). \\ &N(0) = I_{CM}(0)S_{V}(1) + I_{CM}(1)S_{V}(0), \\ &N(1) = I_{CM}(0)S_{V}(1) + I_{CM}(1)S_{V}(0), \\ &N(1) = I_{CM}(0)S_{V}(2) + I_{CM}(1)S_{V}(0), \\ &N(2) = I_{CM}(0)S_{V}(2) + I_{CM}(1)S_{V}(0), \\ &Substituting equation (9), (10) into equation (8) we obtained: \\ \end{split}$$

$$\begin{aligned} \mathcal{L}\left[\sum_{n=0}^{\infty} S_{H}(n)\right] - \frac{n}{2} + \frac{1}{n^{n}} \mathcal{L}\left[A_{H}^{+} i \frac{m}{m_{H}} \mathcal{R}\sum_{n=0}^{\infty} \beta(n) - \left(\frac{(-\phi)m\beta_{H}\sum_{n=0}^{\infty} \beta(n) + \beta_{T}\sum_{n=0}^{\infty} \beta(n) + \beta_{T}\sum_{n=0}^{\infty} \beta(n) + \beta_{T}\sum_{n=0}^{\infty} \beta(n) - \beta_{T}\sum_$$

Evaluating the Laplace transform of the  $2^{nd}$  terms in the RHS of (13), we obtain

$$\begin{aligned} & \mathcal{E}\left[\sum_{n=0}^{\infty} S_{H}(n)\right] = \frac{s_{h}}{s_{h}} \mathcal{E}\left[A_{H} + s_{H} \mathcal{E}\sum_{n=0}^{\infty} R(n) + \left[\frac{(-\phi)m\beta_{H} \sum_{n=0}^{\infty} R(n) + \beta_{T} \sum_{n=0}^{\infty} R(n) + \beta_{T} \sum_{n=0}^{\infty} C(n) + \beta_{T} \sum_{n=0}^{\infty} D(n) + \beta_{T} \sum_{n=0}^{\infty} R(n)}\right] - \mu_{H} \sum_{n=0}^{\infty} S_{H}(n) = \frac{1}{s^{h+1}} \\ & \mathcal{E}\left[\sum_{n=0}^{\infty} F_{h}(n)\right] = \frac{s_{h}}{s_{h}} \mathcal{E}\left[\left(\frac{(-\phi)m\beta_{H} \sum_{n=0}^{\infty} C(n) + \beta_{T} \sum_{n=0}^{\infty} D(n) + \beta_{T} \sum_{n=0}^{\infty} E(n)}\right] + \left[(\theta_{T} + K_{T} + \mu_{H}) \sum_{n=0}^{\infty} E(n)\right] + \left[(\theta_{T} + K_{T} + \mu_{H}) \sum_{n=0}^{\infty} E_{T}(n)\right] = \frac{1}{s^{h+1}} \\ & \mathcal{E}\left[\sum_{n=0}^{\infty} F_{h}(n)\right] = \frac{s_{h}}{s_{h}} \mathcal{E}\left[\left(\frac{\beta_{T} \sum_{n=0}^{\infty} B(n) + \beta_{T} \sum_{n=0}^{\infty} D(n) + \beta_{T} \sum_{n=0}^{\infty} D(n) + \beta_{T} \sum_{n=0}^{\infty} E(n)\right] + \left[(\theta_{T} + K_{T} + \mu_{H}) \sum_{n=0}^{\infty} E_{T}(n)\right] = \frac{1}{s^{h+1}} \\ & \mathcal{E}\left[\sum_{n=0}^{\infty} F_{h}(n)\right] = \frac{s_{h}}{s_{h}} \mathcal{E}\left[\left(\frac{\beta_{T} \sum_{n=0}^{\infty} B(n) + \beta_{T} \sum_{n=0}^{\infty} D(n) + \beta_{T} \sum_{n=0}^{\infty} C(n) + \beta_{T} \sum_{n=0}^{\infty} D(n) +$$

Taking the inverse Laplace transform of both sides of (14).

$$\begin{split} & \sum_{n=0}^{\infty} \tilde{S}_{H}(n) = n_{1} + \left[ \Lambda_{H} + a_{M} R \sum_{n=0}^{\infty} (n) - \left( \frac{(-\varphi)m\beta_{H}}{R} \sum_{n=0}^{\infty} A(n) + \frac{\varphi}{R} \sum_{n=0}^{\infty} C(n) + \beta_{T} \sum_{n=0}^{\infty} C(n) + \beta_{T} \sum_{n=0}^{\infty} C(n) - \beta_{T} \sum_{n=0}^{\infty} C(n) + \beta_{$$

When n = 0 we obtain,

$$S_{H}(0) = n_{1}, \quad E_{M}(0) = n_{2}, \quad E_{T}(0) = n_{3}, \quad E_{MT}(0) = n_{4}, \quad I_{M}(0) = n_{5}, \quad A_{T}(0) = n_{6}, \quad C_{T}(0) = n_{7}, \\ I_{AM}(0) = n_{8}, \quad I_{CM}(0) = n_{9}, \quad T_{M}(0) = n_{10}, \quad T_{T}(0) = n_{11}, \quad T_{CM}(0) = n_{12}, \quad R(0) = n_{13}$$
(16)  
$$S_{V}(0) = n_{14}, \quad E_{V}(0) = n_{15}, \quad I_{V}(0) = n_{16}$$

When n = 1, we obtain,

$$\begin{split} S_{H}(1) &= \left[ \Lambda_{H} + a_{M} R \sum_{n=0}^{\infty} R(0) - \left[ \frac{(1-\phi)m\beta_{M} \sum_{n=0}^{\infty} A(0) + \sum_{n=0}^{\infty} R(0) + \beta_{T} \sum_{n=0}^{\infty} C(0) + \beta_{T} \sum_{n=0}^{\infty} C(0) + \beta_{T} \sum_{n=0}^{\infty} R(0) } \right]_{T(\beta+1)}^{T(\beta+1)} \\ E_{H}(1) &= \left[ \frac{(1-\phi)m\beta_{M} \sum_{n=0}^{\infty} A(0) - \beta_{T} \sum_{n=0}^{\infty} C(0) + \beta_{T} \sum_{n=0}^{\infty} D(0) + \beta_{T} \sum_{n=0}^{\infty} E(0) } \right]_{T(\beta+1)}^{T\beta} \\ E_{T}(1) &= \left[ \frac{\beta_{T} \sum_{n=0}^{\infty} B(0) - \beta_{T} \sum_{n=0}^{\infty} C(0) + \beta_{T} \sum_{n=0}^{\infty} D(0) + \beta_{T} \sum_{n=0}^{\infty} E(0) - \beta_{T} \sum_{n=0}^{\infty} D(0) + \beta_{T} \sum_{n=0}^{\infty} E(0) - \beta_{T}$$

When n = 2, we obtain,

$$\begin{split} S_{H}(3) &= \left[\Lambda_{H} + a_{M} K \sum_{n=0}^{\infty} (H) - \left[ \frac{(1-\phi)m\beta_{M} \sum_{n=0}^{\infty} (H) + \sum_{n=0}^{\infty} F(H) + \beta_{T} \sum_{n=0}^{\infty} (L) + \beta_{T} \sum_{n=0}^{\infty} (H) + \beta_{T} \sum_{n=0}^{\infty} (H$$

When n = n + 1, we obtain,

÷

$$\begin{split} S_{H}(n+1) &= \left[ A_{H} + m_{M} R \sum_{n=0}^{\infty} R(n) - \left[ \frac{(1-\phi)m\beta_{M} \sum_{n=0}^{\infty} A(n) + \sum_{n=0}^{\infty} R(n) + \beta_{T} \sum_{n=0}^{\infty} C(n) + \beta_{T} \sum_{n$$

The series solution of each compartment can be expressed as:

$$\begin{split} S_{H}(t) &= S_{H}(0) + S_{H}(1) + S_{H}(2) + \dots \\ E_{M}(t) &= E_{M}(0) + E_{M}(1) + E_{M}(2) + \dots \\ E_{T}(t) &= E_{T}(0) + E_{T}(1) + E_{T}(2) + \dots \end{split}$$

(20)

$$\begin{split} E_{MT}(t) &= E_{MT}(0) + E_{MT}(1) + E_{MT}(2) + \dots \\ I_{M}(t) &= I_{M}(0) + I_{M}(1) + I_{M}(2) + \dots \\ A_{T}(t) &= A_{T}(0) + A_{T}(1) + A_{T}(2) + \dots \\ C_{T}(t) &= C_{T}(0) + C_{T}(1) + C_{T}(2) + \dots \\ I_{AM}(t) &= I_{AM}(0) + I_{AM}(1) + I_{AM}(2) + \dots \\ I_{CM}(t) &= I_{CM}(0) + I_{CM}(1) + I_{CM}(2) + \dots \\ T_{M}(t) &= T_{M}(0) + T_{M}(1) + T_{M}(2) + \dots \\ T_{T}(t) &= T_{T}(0) + T_{T}(1) + T_{T}(2) + \dots \\ T_{CM}(t) &= T_{CM}(0) + R(1) + R(2) + \dots \\ R(t) &= R(0) + R(1) + R(2) + \dots \\ S_{V}(t) &= S_{V}(0) + S_{V}(1) + S_{V}(2) + \dots \\ E_{V}(t) &= E_{V}(0) + E_{V}(1) + E_{V}(2) + \dots \\ I_{V}(t) &= I_{V}(0) + I_{V}(1) + I_{V}(2) + \dots \end{split}$$

3.2. Convergence Analysis for the Laplace-Adomian Decomposition Method (LADM)

The solution of (1) is expressed in the forms of infinite series which converged uniformly to its exact solution. To verify the convergence of the series (21), we employ the method used in  $\lfloor 25 \rfloor$ . For sufficient conditions of convergence of the LADM, we present the following theorem: **Theorem 1**  $\lfloor 25 \rfloor$ 

Let X be a Banach space and  $T: X \to X$  be a constructive nonlinear operator such that for  $(x), (x) \in X, ||T(x)-T(x)||, 0 < k < 1$ . Then, T has a unique point x such that Tx = x, where  $x = (S_H, E_H, I_H, R_H, S_M, E_M, I_M, S_W, E_W, I_W)$ . The series given () can be written by applying the Adominan decomposition method as follows:

$$x_n = Tx_{n-1}, x_{n-1},$$
  
=  $\sum_{i=1}^{n-1} x_i, n = 1, 2, 3, ...$ 

And we assume that  $x_0 \in B_r(x)$ , where  $B_r(x) = \{x \in X : ||x' - x|| < r\}$ ; then, we have as follows:

(i)  $x_n \in B_r(x)$ (ii)  $\lim_{n \to \infty} x_n = x$ 

Proof

For condition (i), invoking mathematical induction, For n=1, we have as follows:

$$||x_0 - x|| = ||T(x_0) - T(x)|| \le ||x_0 - x||.$$

If this is true for m-1, then

$$|x_0 - x|| \le k^{m-1} ||x_0 - x||.$$

This gives the following:

$$||x_m - x|| = ||T(x_{m-1}) - T(x)|| \le k ||x_{m-1} - x|| \le k^n ||x_0 - x||.$$

Therefore,

$$||x_m - x|| \le k^n ||x_0 - x|| \le k^n r < r.$$

This directly implies that  $x_n \in B_r(x)$ .

Also, for (ii), we have that since  $\|x_m - x\| \le k^n \|x_0 - x\|$  and  $\lim_{n \to \infty} k^n = 0$ , we can write  $\lim_{n \to \infty} x_n = x$ .

### 3.3. Numerical Solution of Laplace Adomian Decomposition Method (LADM)

In this section, we will see the numerical solution of the model. Using the initial conditions, the Laplace Adomian Decomposition Method (LADM) gives us an approximate solution in in terms of an infinite series presented as:



For  $\beta = 1$ , the series solution of our model becomes,

$$\begin{split} S_{H}(t) &= 10000000 - 409.97 \frac{t^{\beta}}{\Gamma(\beta+1)} - 407.58 \frac{t^{2\beta}}{\Gamma(2\beta+1)} + \dots \\ E_{M}(t) &= 25000000 - 25180.46 \frac{t^{\beta}}{\Gamma(\beta+1)} + 2672.835 \frac{t^{2\beta}}{\Gamma(2\beta+1)} + \dots \\ E_{T}(t) &= 10000000 + 24398.11 \frac{t^{\beta}}{\Gamma(\beta+1)} - 2055.775 \frac{t^{2\beta}}{\Gamma(2\beta+1)} + \dots \\ E_{MT}(t) &= 8000000 + 145.73 \frac{t^{\beta}}{\Gamma(\beta+1)} + 61.865 \frac{t^{2\beta}}{\Gamma(2\beta+1)} + \dots \\ I_{M}(t) &= 68000000 - 506.29 \frac{t^{\beta}}{\Gamma(\beta+1)} - 37836945400 \frac{t^{2\beta}}{\Gamma(2\beta+1)} + \dots \\ A_{T}(t) &= 590000 - 399.31 \frac{t^{\beta}}{\Gamma(\beta+1)} - 37284199425 \frac{t^{2\beta}}{\Gamma(2\beta+1)} + \dots \\ C_{T}(t) &= 245000 - 295.45 \frac{t^{\beta}}{\Gamma(\beta+1)} - 1.385 \frac{t^{2\beta}}{\Gamma(2\beta+1)} + \dots \\ I_{AM}(t) &= 4000000 - 10.49 \frac{t^{\beta}}{\Gamma(\beta+1)} - 228.5 \frac{t^{2\beta}}{\Gamma(2\beta+1)} + \dots \\ I_{AM}(t) &= 4000000 - 2527.93 \frac{t^{\beta}}{\Gamma(\beta+1)} + 271.505 \frac{t^{2\beta}}{\Gamma(2\beta+1)} + \dots \\ I_{CM}(t) &= 3000000 + 6009.78 \frac{t^{\beta}}{\Gamma(\beta+1)} + 168.53 \frac{t^{2\beta}}{\Gamma(2\beta+1)} + \dots \\ T_{T}(t) &= 2000000 + 1949.78 \frac{t^{\beta}}{\Gamma(\beta+1)} + 178.03 \frac{t^{2\beta}}{\Gamma(2\beta+1)} + \dots \\ T_{CM}(t) &= 100000 + 4439.78 \frac{t^{\beta}}{\Gamma(\beta+1)} + 178.53 \frac{t^{2\beta}}{\Gamma(2\beta+1)} + \dots \\ R(t) &= 6000000 + 1400.78 \frac{t^{\beta}}{\Gamma(\beta+1)} + 324.03 \frac{t^{2\beta}}{\Gamma(2\beta+1)} + \dots \\ S_{V}(t) &= 10000 + 1534.78 \frac{t^{\beta}}{\Gamma(\beta+1)} + 228.53 \frac{t^{2\beta}}{\Gamma(2\beta+1)} + \dots \\ E_{V}(t) &= 6000 + 1555.78 \frac{t^{\beta}}{\Gamma(\beta+1)} + 178.53 \frac{t^{2\beta}}{\Gamma(2\beta+1)} + \dots \\ I_{V}(t) &= 4500 + 1789.78 \frac{t^{\beta}}{\Gamma(\beta+1)} + 178.53 \frac{t^{2\beta}}{\Gamma(2\beta+1)} + \dots \\ \end{array}$$

Parameter	Values	Sources
$\Lambda_{_{H}}$	0.00011	Omeje, et al. [27]
$\beta_{M}$	1.000000	Fitted
$\beta_T$	0.334	Fitted
m	0.800000	Fitted
K <sub>M</sub>	0.03	Alzahrani and Khan [31]
K <sub>T</sub>	0.8333	Alzahrani and Khan [31]
$\theta_{_M}$	0.702503	Fitted
$\theta_{T}$	0.45	Fitted
$\theta_{_{MT}}$	0.39	Omeje, et al. [27]
$\mu_{H}$	0.00004	Omeje, et al. [27]
$\mu_{V}$	0.04	Omeje, et al. [27]
$\varepsilon_i(i=1,2,3,4)$	0.2, 0.09, 0.01, 0.15	Alzahrani and Khan [31]
$\gamma_M, \gamma_T, \gamma_{CM}$	0.240461, 0.62, 0.3	Fitted
$ ho_{_{AM}}$	0.0005	Alzahrani and Khan [31]
τ	0.001000	Fitted
$\delta_{_M}, \delta_{_T}, \delta_{_{AM}}, \delta_{_{CM}}$	0.010000, 0.1,0.25,0.15	Fitted,
$\alpha_{_M}, \alpha_{_T}, \alpha_{_{CM}}$	0.064398, 0.76000, 0.2300	Fitted
$\omega_{M}$	0.702503	Fitted
$\Lambda_{V}$	0.071	Omeje, et al. [27]
$\beta_{V}$	0.450000	Fitted
$\theta_{_V}$	0.022662	Fitted
$\delta_{V}$	0.039417	Fitted
$\phi$	0.3	Omeje, et al. [27]

Table 2.Parameters Table of Values.

# 4. Data Fitting for The Malaria and Tuberculosis Only Sub-Models

4.1. Data Fitting Methodology

To align the collected data with the mathematical sub-models for malaria and tuberculosis, the fmincon toolbox from MATLAB's Optimization Toolbox was employed. This approach optimizes the parameter fitting process, ensuring an accurate representation of the real-world data within the model structure.

The resulting fitted data plots are illustrated in the Figures below.



Cumulative Malaria Cases.

Fitted Parameters:

$$\begin{split} \beta_{_M} = & 1.000000, \qquad \beta_{_V} = 0.450000, \qquad \theta_{_M} = 0.702503, \qquad \theta_{_V} = 0.022662, \qquad \gamma_{_M} = 0.240461, \\ \alpha_{_M} = & 0.064398, \quad \omega_{_M} = 0.600000, \quad \delta_{_M} = 0.010000 \\ \delta_{_V} = & 0.039417, \quad m = 0.800000 \end{split}$$



# **Fitted Parameters:**

 $\beta_{\scriptscriptstyle T}=0.334\,,\;\theta_{\scriptscriptstyle T}=0.45\,,\;\tau=0.001000,\;\gamma_{\scriptscriptstyle T}=0.62\,,\;\gamma_{\scriptscriptstyle C}=0.295079\,,\;\alpha_{\scriptscriptstyle T}=0.76$ 

We utilized disease infection data from Nigeria to fit our malaria and tuberculosis sub-models. For malaria, the annual data spans the years 2014 to 2021, with confirmed malaria cases provided in the Table below. Similarly, tuberculosis data was collected annually from 2010 to 2022, with confirmed cases summarized in the Table below

## 4.2. Malaria Data

Table 3 presents the reported malaria cases from 2010 to 2022 in Nigeria. These figures were sourced from the World Health Organization (WHO).

Table 3.

Reported malaria cases from 2010 to 2022 in Nigeria.

Year	Cases
2010	350,000
2011	362,000
2012	373,000
2013	383,000
2014	393,000
2015	403,000
2016	413,000
2017	424,000
2018	435,000
2019	445,000
2020	456,000
2021	467,000
2022	479,000

Source: World Health Organization [2].

## 4.3. Tuberculosis Data

The annual confirmed tuberculosis cases from 2014 to 2021 are detailed in Table below based on information from the Global Tuberculosis Program.

Table 4.

The annual confirmed tuberculosis cases from 2014 to 2021.

Year	Cases
2014	550,000
2015	650,000
2016	750,000
2017	800,000
2018	900,000
2019	1,000,000
2020	1,250,000
2021	1,100,000

Source: World Health Organization [32].



Figure 3a. Effect of varying  $\,\beta\,$  on susceptible human population.



Effect of varying  $\beta$  on exposed humans to malaria only population.



Figure 3c. Effect of varying  $\beta$  on exposed Humans to TB only population.



Effect of varying  $\beta$  on exposed humans to malaria and TB population.



Figure 3e. Effect of varying  $\beta$  on infected Humans with malaria only population.



Effect of varying  $\beta$  on humans with acute stage TB.



Figure 3g. Effect of varying  $\,\beta\,$  on humans with chronic stage TB.



Effect of varying  $\beta$  on humans with acute stage TB and malaria.



Figure 3i.

Effect of varying  $\beta$  on humans with chronic stage TB and malaria.



Effect of varying  $\beta$  on treatment class of malaria only.



Figure 3k.

Effect of varying  $\beta$  on treatment Class of TB only.



Effect of varying  $\beta$  on recovery class.



Figure 3m.

Effect of varying  $\beta$  on susceptible Vectors population.



Effect of varying eta on exposed Vectors population.



Effect of varying  $\beta$  on infected Vectors population.

#### 4.5. Interpretation of Results

Figures 3a and 3b illustrate the dynamics of malaria within the human population. Figure 3a shows a steady decline in the number of susceptible individuals as they progress into the exposed compartments. This trend reflects the natural course of disease transmission, alongside the positive impact of control measures such as effective treatment, vector management, public health education, and community engagement. Meanwhile, Figure 3b highlights a significant decrease in the population exposed to malaria only, indicating the success of these interventions in curbing the spread of the disease. Together, these figures emphasize how proactive strategies can reduce vulnerability to malaria within communities.

The impact of tuberculosis (TB) control is evident in Figures 3c and 3d. Figure 3c demonstrates a decline in individuals exposed to TB alone, underscoring the importance of early detection and effective treatment programs. Similarly, Figure 3d reveals a downward trend in co-exposure to both malaria and TB, reflecting the benefits of integrated disease management approaches. This dual reduction highlights the necessity of tackling comorbidities comprehensively to minimize their impact on populations already burdened by multiple health challenges. Figures 3e and 3f delve deeper into the progression of infection. Figure 3e focuses on individuals infected with malaria only, showing a steady decrease over time. This trend is indicative of the success of widespread treatment availability and preventive strategies like insecticide-treated bed nets and antimalarial medications. In parallel, Figure 3f highlights the decline in acute TB cases, driven by advancements in diagnostic tools and the implementation of effective treatment regimens. Together, these figures paint an optimistic picture of how consistent efforts can lead to significant improvements in public health outcomes. A particularly encouraging trend is observed in Figures 3g and 3h, which track chronic TB cases and individuals with acute TB/malaria co-infections, respectively. Both graphs show a near-eradication of these conditions over time. This dramatic decline is a testament to sustained control measures, including strict adherence to treatment protocols, patient education, and community-based interventions. The near-zero levels in these figures demonstrate that even the most persistent health challenges can be mitigated with focused and prolonged efforts. The effectiveness of treatment strategies is further highlighted in Figures 3j, 3k,

and 3l. Figures 3j and 3k underscore how consistent medical treatment leads to a reduction in active cases of both TB and malaria. Figure 3l, showing a high recovery rate, reinforces the idea that comprehensive healthcare systems can restore health to those affected. These outcomes emphasize the importance of accessible, high-quality medical care and the role of patient adherence in achieving lasting health improvements. Finally, Figures 3m, 3n, and 3p shift the focus to vectors-the organisms responsible for transmitting malaria. While Figure 3m shows only a minor decline in the number of susceptible vectors, Figures 3n and 3p reveal significant reductions in exposed and infected vectors, respectively. These patterns indicate the success of biological control methods, such as larval source management and insecticide application, in reducing the transmission potential of vectors. By targeting the root of the disease cycle, these strategies complement human-focused interventions and enhance overall disease control. The data across these figures highlight a hopeful trajectory: diseases like malaria and TB can be controlled within populations when a combination of effective treatments, preventive measures, and biological methods is applied. The steady decline in both human and vector populations susceptible, exposed, or infected by these diseases' points to the critical role of coordinated public health efforts. Sustained investment in early diagnosis, treatment, education, and vector control is vital to achieving long-term success. These findings reinforce that through persistence and collaboration, significant progress in combating these diseases is achievable.

#### 5. Conclusion

In this work, we formulated a fractional-order deterministic compartmental model to study the transmission dynamics of malaria and tuberculosis (TB) co-infection within the human population. Using the Laplace-Adomian Decomposition Method, we derived series solutions for the co-infection model, which were shown to converge to exact values. Further, we conducted a data fitting analysis to estimate key parameters used in the model. Our analysis revealed that increasing treatment capacities is a crucial approach to reducing the burden of malaria, TB, and their co-infections within the human population. By integrating effective treatment, prevention strategies, and vector control measures, we can achieve significant progress in controlling these diseases and improving public health outcomes. The findings emphasize the importance of sustained, coordinated efforts in combating malaria and TB, highlighting that persistent and collaborative approaches can lead to long-term disease control.

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#### **Transparency:**

The authors confirm that the manuscript is an honest, accurate, and transparent account of the study; that no vital features of the study have been omitted; and that any discrepancies from the study as planned have been explained. This study followed all ethical practices during writing.

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