Hindawi
Journal of Mathematics
Volume 2024, Article ID 5521979, 28 pages
https://doi.org/10.1155/2024/5521979

Research Article

Mathematical Modelling of Tuberculosis and Hepatitis C Coinfection Dynamics with No Intervention

Mohammed Kizito,[1] **Hasifa Nampala**,[2] **and Pius Ariho**[3]

[1] *Department of Mathematics, Mbarara University of Science and Technology, Mbarara, Uganda*
[2] *Department of Mathematics and Statistics, Kyambogo University, Kyambogo, Uganda*
[3] *Faculty of Computing and Informatics, Mbarara University of Science and Technology, Mbarara, Uganda*

Correspondence should be addressed to Mohammed Kizito; kmohammed@must.ac.ug

Received 28 November 2023; Revised 16 April 2024; Accepted 23 April 2024; Published 21 May 2024

Academic Editor: Yongqiang Fu

In this study, a deterministic tuberculosis (TB)-hepatitis C (HCV) coinfection mathematical model with no intervention is analysed purposely to examine the dynamics of TB-HCV coinfection so as to find conditions for reducing the transmission of both TB and HCV. A unique solution to the model exists; it is positive and is bounded. The analytical and numerical analysis show that for basic reproduction number, $R_0 = \max\{R_T, R_H\} < 1$, the TB and HCV disease-free equilibrium points are stable. Further analysis shows that when the TB-HCV coinfection basic reproduction number is greater than 1, the endemic equilibrium point is stable. Sensitivity analysis reveals that interventions to reduce TB or HCV infection need to aim and concentrate on minimizing the numbers of the effective contact rate with TB- or HCV-infected humans and the rate of progress from latent TB or acute HCV to infectious TB or chronic HCV stage. Numerical simulations reveal that over time, the number of TB latent humans, acute HCV humans, and the number of dually infected humans have a linear relationship with the effective contact and the progression rates for both TB- and HCV-infected humans. We recommend that health education campaigns to communities aimed at reducing the transmission rates of TB and HCV be conducted. These could include screening and isolation, wearing of face masks for TB cases and screening, sterilization of surgical instruments, and use of condoms for HCV-infected humans.

1. Introduction

As stated by the World Health Organisation (WHO) report 2022, a record of 1.6 million humans died from tuberculosis (TB) in 2021, out of which 187000 humans had human immunodeficiency virus (HIV). TB is an infectious disease that is a predominant cause of unhealthiness and among the primary causes of mortality worldwide. TB has always been ranked topmost as the major killer from a single infectious agent well above human immunodeficiency virus (HIV), not including the COVID-19 pandemic [1].

Tuberculosis (TB) is majorly an infection of the lungs. It is caused by *Mycobacterium tuberculosis* bacteria (Mtb). The disease is commonly spread when an infected person coughs, sneezes, or spits in air. The infectious tuberculosis symptoms in the lungs include a cough that lasts more than three weeks, fever, coughing up blood, night sweats, and loss of weight [2]. A TB infection does not always mean one will get sick. Humans infected with latent tuberculosis show no symptom of TB infection and neither do they transmit it. However, these humans can have the germs multiply and hence become sick after a latency period [3]. Parts of the world with the most TB cases include South East Asia, Africa, and the West Pacific.

Across the globe, approximately 328 million humans are infected with chronic hepatitis C (HCV) or hepatitis B (HBV) with the predominant part of the humans undetected and thus not on medication [4]. In 2019, approximately three million humans contracted chronic HCV and HBV infections in spite of availableness of intervention to curb transmission. In the same year, an estimate of 1.1 million humans died from HCV/HBV-induced chronic liver disease and liver cancer [4].

Hepatitis C is a viral infection brought about by hepatitis C virus that affects the liver. The virus leads to liver inflammation and occasionally, it causes serious liver damage. HCV is a predominant cause of chronic liver diseases. It is approximated that 33% of the HCV-infected humans progress to develop hepatitis cirrhosis (scarring of the liver), steatosis, and liver cancer [5].

Human population is the source for hepatitis C. The transmission of HCV is essentially through blood. At the moment, drug injection into the bloodstream is regarded as a major contributor to almost all newly diagnosed HCV infections. However, transmission through vertical means is another usual route of transmission. Other uncommon transmission routes include sexual contact and via blood transfusion (rarely occurs since donated blood that might contain antibody to HCV is abandoned) [6].

HCV could be diagnosed by detecting antibodies to HCV in an enzyme-linked immunosorbent assay (ELISA). However, no symptoms are shown until several years after infection. Thus, screening of high-risk humans for HCV antibodies needs to be done.

HCV is treatable and curable. It is also noted that currently there is no vaccine for HCV.

A coinfection means simultaneous infection of the same host with two or multiple pathogen species, leading to coexistence of the species within the host or at the population level. Humans infected with TB tend to register more new cases of HCV chronic infection than the general population [7] and this increases the risk of the occurrence of drug-induced hepatotoxicity [8]. HCV chronic infection makes the earlier complicated administration of multidrug-resistant TB (MDR-TB) patients even more strenuous. TB coinfection with HCV activates latent TB and increases the risk of death and drug-induced liver damage [9].

The use of mathematical models to capture the causes and the control of infectious diseases has been studied widely [10–13]. Mathematical deterministic models have considerably been applied to comprehend the behaviour of infections both at population and within host levels including proposing strategic intervention approaches. Various researchers have analysed mathematical models for dual infection of a number of infections to ascertain the effect of a given infection on the behaviour of the other and contrariwise. For instance, Bhunu et al. [14] studied the coinfection of HIV and TB; Bowong and Kurths [15] studied the coinfection of TB and HBV; Mayanja et al. [16], Carvalho et al. [17], and Bhunu and Mushayabasa [18] studied the coinfection of HIV and HCV; Nampala et al. [19] studied the coinfection of HIV and HBV; Sanga et al. [20] studied the coinfection of HIV and cervical cancer; and Nannyonga et al. [21] studied the coinfection of HIV and malaria.

Mayanja et al. [16] developed and analysed a deterministic mathematical model of the HIV and HCV codynamics behaviour without medication. They concluded that HIV and HCV latently infected humans need to pursue prompt medication so as to curb the advancement of HIV to AIDS and HCV latent to HCV.

Bhunu and Mushayabasa [18] developed and analysed a mathematical model of the coinfection of HCV and HIV/AIDS with the view to rate their influence on the transmission dynamics of each infection with therapy. They concluded that there is need to reinforce the control of HCV since it has long term negative impact on the wellbeing of humans.

Bowong and Kurths [15] considered a mathematical model of the coinfection of HBV and TB. Using numerical analysis, they realized that the two infections do cooccur at any time their effective reproduction number is greater than one. They also observed that the prevalence levels of TB and hepatitis B were greatly influenced by the rate of progression of latent to active TB in dually infected humans and acute HBV to chronic HBV infection.

The existing mathematical models for coinfection do not consider the TB and HCV coinfection, yet the two infections are a great threat, especially where they are endemic. Therefore, this study aims at mathematically analysing the transmission dynamics of TB and HCV coinfection in order to find the conditions for coinfection interruption.

In our study, a deterministic mathematical model is derived and analysed with an aim of investigating the dynamics of TB infection as a result of HCV infection and vice versa in absence of treatment. Without treatment, the death rate from TB disease is high (about 50%) [1]. Despite the availability of antibiotics, TB-infected humans may not be under medication in the view of the fact that they may not have been detected or if detected, they could just choose to delay to start the antibiotics. In some developing countries like Uganda, and in general, those below the poverty line, some TB-infected humans may have no access to antibiotics. Others may decide to drop out since the treatment takes a long period of time and rather expensive. Comparably, HCV-infected humans, especially in the chronic stage, may be undetected and, therefore, cannot quest medication. Furthermore, screening, detection, and medication of HCV-infected humans continue to remain a challenge globally [18]. Therefore, there is a need to investigate the TB-HCV coinfection dynamics in absence of intervention. The current model will inform policymakers of factors that fuel the transmission of both TB and HCV infections and hence the measures that can be put to curtail the transmission rate.

2. Model Description

Our model partitions the population of individuals into the subsequent subgroups as follows: susceptible humans at a risk of contracting TB or HCV $(S(t))$, tuberculosis latently infected but not infectious of TB $(I_L(t))$, infectious TB humans assumed not under medication $(I_T(t))$, acute HCV humans without symptoms but infectious of HCV $(I_a(t))$, chronic HCV humans with symptoms and infectious of HCV $(I_C(t))$, coinfected humans with acute HCV in the TB latent stage non-TB infectious but infectious of HCV $(I_{aL}(t))$, coinfected humans with HCV chronic infection in the TB latent stage infectious of HCV and not TB $(I_{CL}(t))$, coinfected humans with HCV acute infection in the TB infectious stage infectious of both HCV and TB $(I_{aT}(t))$, and coinfected humans with HCV chronic infection in the TB infectious stage infectious of both HCV and TB ($I_{CT}(t)$), all as summarised in Table 1.

It is supposed that humans in the susceptible subgroup increase at a rate Λ. All humans in different subgroups go through natural death at a constant rate μ. Susceptible humans contract TB infection after a contact with TB infectious human at a rate λ_T and acquire HCV infection after contact with an HCV infectious human at a rate λ_H.

The total number of humans at time t is $N(t)$ and is given by

$$N(t) = S(t) + I_L(t) + I_T(t) + I_a(t) + I_C(t) + I_{aL}(t) + I_{CL}(t) + I_{aT}(t) + I_{CT}(t). \quad (1)$$

The rate at which humans acquire TB infection is given by

$$\lambda_T(t) = \frac{\xi_1 q_1 \left(I_T(t) + a_1 I_{aT}(t) + a_2 I_{CT}(t)\right)}{N(t)}, \quad (2)$$

where ξ_1 is the effective rate of contact with TB infected human. q_1 is the likelihood of the contact being well efficient to give rise to a TB infection. a_1 and a_2 are enhancement factors for the threat of easily contracting TB from a coinfected human in I_{aT} and I_{CT} classes, respectively. Both a_1 and a_2 represent the fact that coinfected humans easily spread the infection compared to those that are not dually infected [18].

Comparably, the incident rate of HCV infection in the population is as shown in the following equation:

$$\lambda_H(t) = \frac{\xi_2 q_2 \left(\phi I_a(t) + I_C(t) + b_1 I_{aL}(t) + b_2 I_{CL}(t) + b_3 I_{aT}(t) + b_4 I_{CT}(t)\right)}{N(t)}, \quad (3)$$

where ξ_2 is the effective contact rate with HCV-infected human. q_2 is the likelihood of the contact being well efficient to give rise to an HCV infection. b_1, b_2, b_3, and b_4 are enhancement factors for the threat of easily contracting HCV from a coinfected human in I_{aL}, I_{CL}, I_{aT}, and I_{CT} classes, respectively. We note that $b_2, b_4 > b_1, b_3 > 1$, with the assumption that an HCV chronically infected human is more infectious than the HCV acutely infected human.

Susceptible humans, once infected with TB, enter TB latently infected class $I_L(t)$. Some humans in $I_L(t)$ subgroup progress to the infectious class $I_T(t)$ at a rate θ while the rest gain natural recovery at a rate η. Humans in the TB infectious class die from both natural and TB-induced death at a rate σ.

Conversely, on contracting HCV, susceptible humans join the acute HCV human class $I_a(t)$. Here, we have some humans in $I_a(t)$ class who recover from the acute HCV instinctively at a rate π where as the rest join the chronic HCV class $I_C(t)$ at a rate α. Humans in acute HCV class only die from natural death with an assumption that acute HCV is not fatal whereas humans in chronic HCV class die from both natural death and chronic HCV at a per capita rate of δ.

When humans in classes $I_L(t)$ and $I_a(t)$ interact, they become dually infected with latent TB and acute HCV and enter a class of the humans coinfected with latent TB and acute HCV $I_{aL}(t)$. The humans in $I_{aL}(t)$ class die from natural death, at a rate μ. Similarly, when humans in class $I_L(t)$ and $I_C(t)$ interact, they are projected to become dually infected with both latent TB and chronic HCV, thus entering a class of the humans coinfected with latent TB and chronic HCV $I_{CL}(t)$. These humans not only die from natural death at a rate μ but also die due to the coinfection at a rate δ.

Humans in class $I_{aL}(t)$ progress to $I_{aT}(t)$ class at a rate τ_1 and the progress to $I_{CL}(t)$ at a rate β_1. Humans in $I_{CL}(t)$ and $I_{aT}(t)$ classes progress to $I_{CT}(t)$ class at rates τ_2 and β_2, respectively. The humans in classes $I_{aT}(t)$ and $I_{CT}(t)$ die from both natural death and coinfection induced death at rates d_1 and d_2, respectively. We note that $d_1, d_2 > \delta$ because the disease-induced death rate from the coinfection is expected to be greater than that of monoinfection.

In the formulation of the TB-HCV coinfection model, it is assumed that all susceptible humans are equally susceptible to TB and HCV; both TB and HCV are transmitted by contact (direct or indirect) between an infected human and a susceptible human. It is also assumed that all the infected HCV humans first develop acute HCV and later progress to chronic form of HCV and both groups are infectious and humans with the acute form of HCV either progress to the chronic form or recover naturally [4]. By [1], all the infected TB humans first become latently infected before developing infectious TB. However, TB latently infected humans can recover without medication and those who are actively infected cannot naturally recover. The parameters used in the explanation of TB-HCV coinfection transmission dynamics are summarized in Table 2.

Based on the description of the TB-HCV coinfection dynamics and assumptions made, Figure 1 presents the TB-HCV coinfection compartmental diagram. From the compartmental diagram, in Figure 1, the associated epidemiological model is as in the following equation system.

Table 1: Variables used in the TB-HCV coinfection dynamics.

Variable	Description
$S(t)$	Susceptible humans
$I_L(t)$	Tuberculosis latently infected but not infectious humans
$I_T(t)$	Infectious TB humans assumed not under medication
$I_a(t)$	Infectious acute HCV humans
$I_C(t)$	Infectious chronic HCV humans
$I_{aL}(t)$	Coinfected humans with HCV acute infection in the TB latent stage
$I_{CL}(t)$	Coinfected humans with HCV chronic infection in the TB latent stage
$I_{aT}(t)$	Coinfected humans with HCV acute infection in the TB active stage
$I_{CT}(t)$	Coinfected humans with HCV chronic infection in the TB active stage

Table 2: Parameters used in the TB-HCV coinfection dynamics and their definition.

Parameter	Description
Λ	Rate of increase in the natural population
μ	Rate at which humans die naturally
σ	Death rate for humans with active TB
δ	Death rate for humans with the chronic form of HCV
d_1	Death rate for humans with acute HCV and active TB dual infection
d_2	Death rate for humans with chronic HCV and active TB dual infection
θ	Rate of progress from the latent stage to the active stage of TB
α	Rate of progress from acute class to chronic HCV
τ_1	Rate of progress from I_{aL} class to I_{aT} class
τ_2	Rate of progress from I_{CL} class to I_{CT} class
β_1	Rate of progress from I_{aL} class to I_{CL} class
β_2	Rate of progress from I_{aT} class to I_{CT} class
η	Natural recovery rate of TB latent humans
π	Natural recovery rate of HCV acute humans
ϕ	Transmission coefficient for the HCV acute humans

$$
\begin{aligned}
\frac{dS(t)}{dt} &= \Lambda + \pi I_a(t) + \eta I_L(t) - (\lambda_T + \lambda_H + \mu)S(t),\\
\frac{dI_L(t)}{dt} &= \lambda_T S(t) - (\theta + \eta + \mu + \lambda_H)I_L(t),\\
\frac{dI_T(t)}{dt} &= \theta I_L(t) - (\mu + \sigma + \lambda_H)I_T(t),\\
\frac{dI_a(t)}{dt} &= \lambda_H S(t) - (\pi + \alpha + \mu + \lambda_T)I_a(t),\\
\frac{dI_C(t)}{dt} &= \alpha I_a(t) - (\mu + \delta + \lambda_T)I_C(t),\\
\frac{dI_{aL}(t)}{dt} &= \lambda_T I_a(t) + \lambda_H I_L(t) - (\tau_1 + \mu + \beta_1)I_{aL}(t),\\
\frac{dI_{CL}(t)}{dt} &= \beta_1 I_{aL}(t) + \lambda_T I_C(t) - (\tau_2 + \mu + \delta)I_{CL}(t),\\
\frac{dI_{aT}(t)}{dt} &= \tau_1 I_{aL}(t) + \lambda_H I_T(t) - (\beta_2 + \mu + d_1)I_{aT}(t),\\
\frac{dI_{CT}(t)}{dt} &= \tau_2 I_{CL}(t) + \beta_2 I_{aT}(t) - (\mu + d_2)I_{CT}(t),
\end{aligned}
\tag{4}
$$

where λ_T and λ_H are described as in (2) and (3), respectively. The starting values of the variables of the model are as follows:

$$S(0) > 0, I_L(0) \geq 0, I_T(0) \geq 0, I_a(0) \geq 0, I_C(0) \geq 0, I_{aL}(0) \geq 0, I_{CL}(0) \geq 0, I_{aT}(0) \geq 0 \text{ and } I_{CT}(0) \geq 0. \tag{5}$$

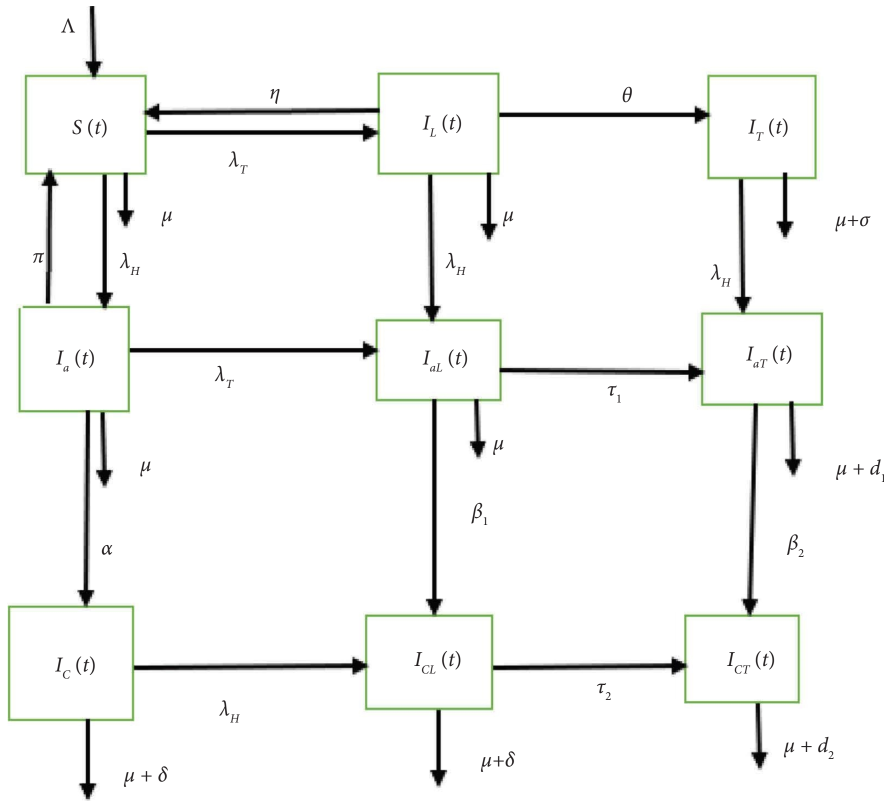

FIGURE 1: A compartmental diagram for TB-HCV coinfection dynamics.

3. Analysis of the Model

3.1. Positivity and Boundedness of Solutions. The model system (4) defines the population of humans. Thus, it is imperative to show that all the variables $S(t), I_L(t), I_T(t), I_a(t), I_C(t), I_{aL}(t), I_{CL}(t), I_{aT}(t)$ and $I_{CT}(t)$ are non-negative for all time.

Theorem 1. *Positivity of solutions.*

Solutions of the model system (4) with non-negative starting values remain non-negative for all $t \geq 0$.

Proof. Let the starting values of the model system (4) be positive. We prove that each solution component of the system remains positive. Otherwise, we assume the following contradiction:

that there exists a first time t_1: $S(t_1) = 0, S'(t_1) < 0$ and $S(t) > 0, I_L(t) > 0, I_T(t) > 0, I_a(t) > 0, I_C(t) > 0, I_{aL}(t) > 0, I_{CL}(t) > 0, I_{aT}(t) > 0, I_{CT}(t) > 0$ for $0 < t < t_1$

or there exists a t_2: $I_L(t_2) = 0, I_L'(t_2) < 0$ and $S(t) > 0, I_L(t) > 0, I_T(t) > 0, I_a(t) > 0, I_C(t) > 0, I_{aL}(t) > 0, I_{CL}(t) > 0, I_{aT}(t) > 0, I_{CT}(t) > 0$ for $0 < t < t_2$

or there exists a t_3: $I_T(t_3) = 0, I_T'(t_3) < 0$ and $S(t) > 0, I_L(t) > 0, I_T(t) > 0, I_a(t) > 0, I_C(t) > 0, I_{aL}(t) > 0, I_{CL}(t) > 0, I_{aT}(t) > 0, I_{CT}(t) > 0$ for $0 < t < t_3$

or there exists a t_4: $I_a(t_2) = 0, I_a'(t_4) < 0$ and $S(t) > 0, I_L(t) > 0, I_T(t) > 0, I_a(t) > 0, I_C(t) > 0, I_{aL}(t) > 0, I_{CL}(t) > 0, I_{aT}(t) > 0, I_{CT}(t) > 0$ for $0 < t < t_4$

or there exists a t_5: $I_C(t_5) = 0, I_C'(t_5) < 0$ and $S(t) > 0, I_L(t) > 0, I_T(t) > 0, I_A(t) > 0, I_C(t) > 0, I_{AL}(t) > 0, I_{CL}(t) > 0, I_{aT}(t) > 0, I_{CT}(t) > 0$ for $0 < t < t_5$

or there exists a t_6: $I_{aL}(t_6) = 0, I_{aL}'(t_6) < 0$ and $S(t) > 0, I_L(t) > 0, I_T(t) > 0, I_a(t) > 0, I_C(t) > 0, I_{aL}(t) > 0, I_{CL}(t) > 0, I_{aT}(t) > 0, I_{CT}(t) > 0$ for $0 < t < t_6$

or there exists a t_7: $I_{CL}(t_7) = 0, I_{CL}'(t_7) < 0$ and $S(t) > 0, I_L(t) > 0, I_T(t) > 0, I_a(t) > 0, I_C(t) > 0, I_{aL}(t) > 0, I_{CL}(t) > 0, I_{aT}(t) > 0, I_{CT}(t) > 0$ for $0 < t < t_7$

or there exists a t_8: $I_{aT}(t_8) = 0, I_{aT}'(t_8) < 0$ and $S(t) > 0, I_L(t) > 0, I_T(t) > 0, I_a(t) > 0, I_C(t) > 0, I_{aL}(t) > 0, I_{CL}(t) > 0, I_{aT}(t) > 0, I_{CT}(t) > 0$ for $0 < t < t_8$

or there exists a t_9: $I_{CT}(t_9) = 0, I_{CT}'(t_9) < 0$ and $S(t) > 0, I_L(t) > 0, I_T(t) > 0, I_a(t) > 0, I_C(t) > 0, I_{aL}(t) > 0, I_{CL}(t) > 0, I_{aT}(t) > 0, I_{CT}(t) > 0$ for $0 < t < t_9$.

From the first equation of model system (4), we have

$$\begin{aligned}\frac{dS(t_1)}{dt} &= \Lambda + \pi I_a(t_1) + \eta I_L(t_1) - (\lambda_T(t_1) + \lambda_H(t_1) \\ &\quad + \mu)S(t_1), \\ &= \Lambda + \pi I_a(t_1) + \eta I_L(t_1) > 0,\end{aligned} \tag{6}$$

thus a contradiction, implying that $S(t)$ shall be non-negative.

From the second equation of model system (4), we have

$$\begin{aligned}\frac{dI_L(t_2)}{dt} &= \lambda_T(t_2)S(t_2) - (\theta + \eta + \mu + \lambda_H(t_2))I_L(t_2),\\ &= \lambda_T(t_2)S(t_2) > 0,\end{aligned} \tag{7}$$

thus a contradiction, implying that $I_L(t)$ shall be non-negative.

From the third equation of model system (4), we have

$$\begin{aligned}\frac{dI_T(t_3)}{dt} &= \theta I_L(t_3) - (\mu + \sigma + \lambda_H(t_3))I_T(t_3),\\ &= \theta I_L(t_3) > 0,\end{aligned} \tag{8}$$

thus a contradiction, implying that $I_T(t)$ shall remain non-negative.

From the fourth equation of model system (4), we have

$$\begin{aligned}\frac{dI_a(t_4)}{dt} &= \lambda_H(t_4)S(t_4) - (\pi + \alpha + \mu + \lambda_T(t_4))I_a(t_4),\\ &= \lambda_H(t_4)S(t_4) > 0,\end{aligned} \tag{9}$$

thus a contradiction, implying that $I_a(t)$ shall be non-negative.

From the fifth equation of model system (4), we have

$$\begin{aligned}\frac{dI_C(t_5)}{dt} &= \alpha I_a(t_5) - (\mu + \delta + \lambda_T(t_5))I_C(t_5),\\ &= \alpha I_A(t_5) > 0,\end{aligned} \tag{10}$$

thus a contradiction, implying that $I_C(t)$ remains non-negative.

From the sixth equation of model system (4), we have

$$\begin{aligned}\frac{dI_{aL}(t_6)}{dt} &= \lambda_T(t_6)I_a(t_6) + \lambda_H(t_6)I_L(t_6)\\ &\quad - (\tau_1 + \mu + \beta_1)I_{aL}(t_6),\\ &= \lambda_T(t_6)I_a(t_6) + \lambda_H(t_6)I_L(t_6) > 0,\end{aligned} \tag{11}$$

giving a contradiction, implying that $I_{AL}(t)$ remains non-negative.

From the seventh equation of model system (4), we have

$$\begin{aligned}\frac{dI_{CL}(t_7)}{dt} &= \beta_1 I_{aL}(t_7) + \lambda_T(t_7)I_C(t_7)\\ &\quad - (\tau_2 + \mu + \delta)I_{CL}(t_7),\\ &= \beta_1 I_{aL}(t_7) + \lambda_T(t_7)I_C(t_7) > 0,\end{aligned} \tag{12}$$

giving a contradiction, implying that $I_{CL}(t)$ remains non-negative.

From the eighth equation of model system (4), we have

$$\begin{aligned}\frac{dI_{aT}(t_8)}{dt} &= \tau_1 I_{aL}(t_8) + \lambda_H(t_8)I_I(t_8)\\ &\quad - (\beta_2 + \mu + d_1)I_{AT}(t_8),\\ &= \tau_1 I_{aL}(t_8) + \lambda_H(t_8)I_I(t_8) > 0,\end{aligned} \tag{13}$$

giving a contradiction, implying that $I_{aT}(t)$ remains non-negative.

From the ninth Equation of model system (4), we have

$$\begin{aligned}\frac{dI_{CT}(t_9)}{dt} &= \tau_2 I_{CL}(t_9) + \beta_2 I_{aT}(t_9) - (\mu + d_2)I_{CT}(t_9),\\ &= \tau_2 I_{CL}(t_9) + \beta_2 I_{aT}(t_9) > 0,\end{aligned} \tag{14}$$

which is a contradiction, meaning that $I_{CT}(t)$ remains positive.

Thus, in all cases, $S(t), I_L(t), I_T(t), I_a(t), I_C(t), I_{aL}(t), I_{CL}(t), I_{aT}(t), I_{CT}(t)$ remain positive for $t \geq 0$.

Theorem 2. *Invariant region.*

The region

$$\begin{aligned}\Omega = \{&S(t), I_L(t), I_T(t), I_a(t), I_C(t), I_{aL}(t), I_{CL}(t), I_{aT}(t),\\ &I_{CT}(t) \in \mathbb{R}_+^9 \colon 0 \leq N(t) \leq \frac{\Lambda}{\mu}\Big\}\end{aligned} \tag{15}$$

is positively invariant and all solutions starting in Ω approach, enter, or stay in Ω.

Proof. Let

$$\begin{aligned}&(S(t), I_L(t), I_T(t), I_a(t), I_C(t), I_{aL}(t), I_{CL}(t),\\ &I_{aT}(t), I_{CT}(t) \in \mathbb{R}_+^9)\end{aligned} \tag{16}$$

be any solution of the model system (4), with non-negative initial condition given by

$$S(0), I_L(0), I_T(0), I_a(0), I_C(0), I_{aL}(0), I_{CL}(0), I_{aT}(0), I_{CT}(0). \tag{17}$$

The total population is given by $N(t)$.

Adding all the differential equations in the model system (4), we have

$$\begin{aligned}\frac{dN(t)}{dt} &= \Lambda - \mu N - \sigma I_T - \delta(I_C + I_{CL})\\ &\quad - d_1 I_{aT} - d_2 I_{CT}.\end{aligned} \tag{18}$$

Since all parameter values are greater than zero and

$$\begin{aligned}&I_T(t) > 0, I_a(t) > 0, I_{aL}(t) > 0, I_{CL}(t) > 0, I_{CT}(t) > 0,\\ &I_C(t) > 0 \text{ and } I_{aT}(t) > 0 \text{ for all } t \geq 0,\end{aligned} \tag{19}$$

then equation (18) yields the inequality

$$\frac{dN(t)}{dt} + \mu N(t) \leq \Lambda. \tag{20}$$

On solving the above inequality by integrating factor method with initial conditions $N(0) = N_0$, we have

$$0 \leq N(t) \leq \frac{\Lambda}{\mu} + \left(N_0 - \frac{\Lambda}{\mu}\right)e^{-\mu t}, \tag{21}$$

so that as $t \longrightarrow \infty$,

$$0 \leq N(t) \leq \frac{\Lambda}{\mu}. \tag{22}$$

Consequently, all possible feasible solutions of the model system (4) that begin in the region

$$\Omega = \{S(t), I_L(t), I_T(t), I_a(t), I_C(t), I_{aL}(t), I_{CL}(t), I_{aT}(t), I_{CT}(t) \in \mathbb{R}_+^9 : 0 \leq N(t) \leq \frac{\Lambda}{\mu}\right\}, \tag{23}$$

remain in the region for all values of t. Thus, the region Ω is positively invariant, that is, for all values of t, the solution is well-posed and biologically meaningful.

3.2. Analysis of the TB-Only Submodel. The TB-only submodel is obtained by equating all the variables concerning HCV in the model system (24) to zero, that is,

$$\begin{aligned} I_a(t) = I_C(t) = I_{aL}(t) = I_{CL}(t) = I_{aT}(t) \\ = I_{CT}(t) = \lambda_H = 0. \end{aligned} \tag{24}$$

Hence, the TB-only submodel is as in the following equation.

$$\begin{aligned} \frac{dS}{dt} &= \Lambda + \eta I_L - (\lambda_T + \mu)S, \\ \frac{dI_L}{dt} &= \lambda_T S - (\theta + \eta + \mu)I_L, \\ \frac{dI_T}{dt} &= \theta I_L - (\mu + \sigma)I_T, \end{aligned} \tag{25}$$

with

$$S(0) = S_0 \geq 0, I_L(0) = I_{L0} \geq 0, I_T(0) = I_{T0} \geq 0 \tag{26}$$

as the starting values,

$$\lambda_T = \frac{\xi_1 q_1 I_T(t)}{N_T(t)} \tag{27}$$

is the force of infection and the total population is given by

$$N_T(t) = S(t) + I_L(t) + I_T(t). \tag{28}$$

Based on biological considerations, the submodel system (24) shall be studied in the following region:

$$\Omega_T = \left\{(S, I_L, I_T) \in \mathbb{R}_+^3 : 0 \leq N_T \leq \frac{\Lambda}{\mu}\right\}. \tag{29}$$

Clearly, the solutions S, I_L, I_T of the submodel system (24) are non-negative for $t \geq 0$ and the region Ω_T is positively invariant and solutions starting in Ω_T approach, enter, or stay in Ω_T.

3.2.1. The TB-Free Equilibrium Point and Reproduction Number for the TB-Only Submodel. To determine the TB-free equilibrium for the TB-only submodel, we suppose that there is no TB infection in the community. Now, equating submodel system (24) to zero gives the TB-free equilibrium point for the TB-only submodel as

$$E_T^0 = \left(S^0, I_L^0, I_T^0\right) = \left(\frac{\Lambda}{\mu}, 0, 0\right). \tag{30}$$

In this instance, we shall define the basic reproduction number R_T as the number of new TB cases given rise to by an infectious TB human during their entire infectious period [22]. Now, the next generation matrix method suggested in [23] is applied to establish the basic reproduction number R_T of system (24).

Let $\mathcal{F}$ represent the matrix of components of new infection and $\mathcal{V}$ the matrix of the rest of transfer components in system (24).

The infected compartments are I_L and I_T. Thus, we have

$$\mathcal{F} = \begin{bmatrix} \lambda_T S \\ 0 \end{bmatrix}, \tag{31}$$

$$\mathcal{V} = \begin{bmatrix} (\theta + \eta + \mu)I_L \\ -\theta I_L + (\mu + \sigma)I_T \end{bmatrix}. \tag{32}$$

The Jacobian matrices of expressions (31) and (32) are computed at the disease-free equilibrium E_T^0, thus yielding matrices F_T and V_T, respectively, as

$$F_T = \begin{bmatrix} 0 & \xi_1 q_1 \\ 0 & 0 \end{bmatrix} \tag{33}$$

and

$$V_T = \begin{bmatrix} (\theta + \eta + \mu) & 0 \\ -\theta & (\mu + \sigma) \end{bmatrix}. \tag{34}$$

Thus, the TB-induced basic reproduction number, R_T, is computed as

$$R_T = \frac{\xi_1 q_1 \theta}{(\theta + \eta + \mu)(\mu + \sigma)}. \tag{35}$$

The decrease of the infection in a human population is influenced by the parameters that will reduce the value of the reproduction number to less than unity. Clearly, it can be observed that when the effective contact rate, ξ_1, with tuberculosis-infected human, the likelihood, q_1, of the contact being well efficient to give rise to a tuberculosis infection and the rate of progression, θ, of latent TB humans to the active TB class become large; this leads to an increase in the TB secondary infections.

Thus, intervention measures to mitigate TB infection should mainly target decreasing ξ_1, q_1, and θ while increasing the natural recovery rate, η, of TB latent humans.

3.2.2. Local Stability of the TB-Only Submodel Disease-Free Equilibrium Point. Using Theorem 2 from [23], the subsequent result is proved.

Lemma 3. *The local and asymptotic stability of the disease-free equilibrium point, E_T^0, of TB-only submodel exists if $R_T < 1$.*

Proof. To establish the local and asymptotic stability of the TB infection-free equilibrium point E_T^0, we use the eigenvalue technique. The approach requires that all the eigenvalues of the Jacobian matrix $J(E_T)$ at E_T^0 have negative real parts as used in [12,16].

The Jacobian matrix, $J(E_T)$, of the TB-only submodel (24) is obtained as follows:

$$J(E_T) = \begin{bmatrix} -\left(\dfrac{\xi_1 q_1 I_T}{N_T} + \mu\right) & \eta & \dfrac{-\xi_1 q_1 S}{N_T} \\ \dfrac{\xi_1 q_1 I_T}{N_T} & -k_1 & \dfrac{\xi_1 q_1 S}{N_T} \\ 0 & \theta & -k_2 \end{bmatrix}, \tag{36}$$

where

$$k_1 = \theta + \eta + \mu \text{ and } k_2 = \mu + \sigma. \tag{37}$$

Evaluating equation (36) at E_T^0 leads to

$$J\left(E_T^0\right) = \begin{bmatrix} -\mu & \eta & -\xi_1 q_1 \\ 0 & -k_1 & \xi_1 q_1 \\ 0 & \theta & -k_2 \end{bmatrix}. \tag{38}$$

The characteristic polynomial of the Jacobian matrix (38) is

$$P(\lambda) = (-\mu + -\lambda)\left(\lambda^2 + (k_1 + k_2)\lambda - \theta\xi_1 q_1\right) = 0. \tag{39}$$

Thus, the eigenvalues of the Jacobian matrix $J(E_T^0)$ are

$$\lambda_1 = -\mu, \lambda_2 = \frac{-(k_1 + k_2) - \sqrt{(k_1 + k_2)^2 + 4\theta\xi_1 q_1}}{2} \text{ and}$$
$$\lambda_3 = \frac{-(k_1 + k_2) + \sqrt{(k_1 + k_2)^2 + 4\theta\xi_1 q_1}}{2}. \tag{40}$$

Now, eigenvalues λ_1 and λ_2 have negative real parts. However, for

$$\lambda_3 = \frac{-(k_1 + k_2) + \sqrt{(k_1 + k_2)^2 + 4\theta\xi_1 q_1}}{2} \tag{41}$$

to have negative real part, the term with a square root should be less than zero, that is,

$$\frac{\sqrt{(k_1 + k_2)^2 + 4\theta\xi_1 q_1}}{2} < 0, \tag{42}$$

from which

$$(k_1 + k_2)^2 + 4\theta\xi_1 q_1 < 0. \tag{43}$$

But from the expression of the basic reproduction number, R_T,

$$\theta\xi_1 q_1 = k_1 k_2 R_T. \tag{44}$$

This implies

$$R_T < \frac{-(k_1 + k_2)^2}{4 k_1 k_2} < 1. \tag{45}$$

Thus, E_T^0 is locally asymptotically stable only if $R_T < 1$.

A local and asymptotic stability of E_T^0 implies that in case some few TB infected humans are introduced into the population, then over time, the system returns to TB-free equilibrium. □

3.2.3. Global and Asymptotic Stability of the TB-Free Equilibrium Point for the TB-Only Submodel. To investigate the global and asymptotic stability of the system of differential equation (24), the approach suggested in [24] and also applied in [12,16] is used.

The TB-only system 6 is rewritten in the following form:

$$\begin{aligned} \frac{dX_T}{dt} &= F(X_T, Z_T), \\ \frac{dZ_T}{dt} &= G(X_T, Z_T), G(X_T, 0) = 0. \end{aligned} \tag{46}$$

where $X_T = (S)$ and $Z_T = (I_L, I_T)$, with $X_T \in \mathbb{R}_+$ indicating the number of uninfected humans and $Z_T \in \mathbb{R}_+^2$ showing the number of humans infected with TB. Let the TB-free equilibrium of this system be denoted by $E_T^0 = (X_T^0, 0) = (\Lambda/\mu, 0)$.

It is necessary to ensure that the following conditions are met to guarantee global asymptotic stability.

$$\begin{aligned} &(H1)\text{: For } \frac{dX_T}{dt} = F(X_T, 0), X_T^0 \text{ is globally asymptotically stable,} \\ &(H2)\text{: } G(X_T, Z_T) = MZ_T - \widehat{G}(X_T, Z_T), \widehat{G}(X_T, Z_T) \geq 0, \end{aligned} \tag{47}$$

for $(X_T, Z_T) \in \Omega_T$, where $M = D_Z G(X_T^0, 0)$ is an Metzler matrix (the off diagonal elements of M are no-negative) and Ω_T is the region where the model makes biological meaning.

Thus, when system (46) satisfies conditions $(H1)$ and $(H2)$, we have the following theorem satisfied.

Theorem 4. *The equilibrium point $E_T^0 = (X_T^0, 0)$ is globally asymptotically stable point of system (46) provided $R_T < 1$ and that conditions $(H1)$ and $(H2)$ are satisfied.*

Proof. By Lemma 3, if $R_T < 1$, then E_T^0 is locally asymptotically stable.

For the first condition $(H1)$, that is, the global asymptotic stability of X_T, we have

$$\frac{dX_T}{dt} = F(X_T, 0) = \Lambda - \mu S, \tag{48}$$

which is a linear differential equation. Solving it, we get

$$S(t) = \frac{\Lambda}{\mu}\left(1 - e^{-\mu t}\right) + S(0)e^{-\mu t}. \tag{49}$$

Now, as $t \longrightarrow \infty$, $S \longrightarrow \Lambda/\mu$ regardless of the value of $S(0)$. Thus, X_T is globally asymptotically stable.

For the second condition $(H2)$, consider

$$\begin{aligned} G(X_T, Z_T) &= \begin{bmatrix} \lambda_T S - k_1 I_L \\ \theta I_L - k_2 I_T \end{bmatrix} \\ &= \begin{bmatrix} \xi_1 q_1 I_T \dfrac{S}{N_T} - k_1 I_L \\ \theta I_L - k_2 I_T \end{bmatrix} \end{aligned} \tag{50}$$

Then,

$$M = D_Z G\left(X_T^0, 0\right) = \begin{bmatrix} -k_1 & \xi_1 q_1 \dfrac{S}{N_T} \\ \theta & -k_2 \end{bmatrix}. \tag{51}$$

At E_T^0,

$$M = \begin{bmatrix} -k_1 & \xi_1 q_1 \\ \theta & -k_2 \end{bmatrix}. \tag{52}$$

Therefore,

$$\widehat{G}(X_T, Z_T) = M Z_T - G(X_T, Z_T) = \begin{bmatrix} \xi_1 q_1 I_T \left(1 - \dfrac{S}{N_T}\right) \\ 0 \end{bmatrix}. \tag{53}$$

Since $0 \le S \le N_T$, then $\widehat{G}(X_T, Z_T) \ge 0$. We also notice that the matrix M is actually a Metzler matrix since both of its off diagonal elements are positive. Thus, it clearly implies that condition $(H2)$ is satisfied. Hence, the TB-free equilibrium point E_T^0 is globally asymptotically stable for $R_T < 1$.

This implies that no matter the number of TB cases that are brought into the population, TB infection shall not persist in the population.

3.2.4. Endemic Equilibrium Point of the TB-Only Submodel. Here, we consider the persistence of TB in the population and determine the TB-endemic equilibrium point. By setting the derivatives of equation (24) of the TB-only submodel to zero, we have the TB-endemic equilibrium point E_T^* as

$$E_T^* = \left(\frac{k_1 k_2 N_T^*}{\theta \xi_1 q_1}, \frac{\Lambda \xi_1 q_1 + \mu k_1 k_2}{\xi_1 q_1 (k_1 - \eta)}, \frac{\theta(\Lambda \xi_1 q_1 + \mu k_1 k_2)}{\xi_1 q_1 k_2 (k_1 - \eta)}\right), \tag{54}$$

where

$$k_1 = \theta + \eta + \mu \text{ and } k_2 = \mu + \sigma. \tag{55}$$

Lemma 5. *The TB-only submodel (24) has a unique endemic equilibrium point if $R_T > 1$.*

Proof. If the infection is consistently present in the population, then $dI_L/dt > 0$ and $dI_T/dt > 0$ as used in [12], that is,

$$\frac{\xi_1 q_1 I_T S}{N_T} - k_1 I_L > 0, \tag{56}$$

$$\theta I_L - k_2 I_T > 0. \tag{57}$$

From inequality (56), we have

$$k_1 I_L < \xi_1 q_1 I_T \frac{S}{N_T}. \tag{58}$$

Using the fact that $S/N_T \le 1$, [18] becomes

$$I_L < \frac{\xi_1 q_1 I_T}{k_1}. \tag{59}$$

From inequality (57), we have

$$I_T < \frac{\theta I_L}{k_2}. \tag{60}$$

Substituting (58) into (59), we get

$$I_L < \frac{\theta \xi_1 q_1 I_L}{k_1 k_2}, \tag{61}$$

$$1 < \frac{\theta \xi_1 q_1}{k_1 k_2} = R_T. \tag{62}$$

Therefore, an endemic equilibrium point E_T^* which is distinctive does exists when $R_T > 1$. □

3.2.5. Local Stability of the TB-Endemic Equilibrium for the TB-Only Submodel

Lemma 6. *The local and asymptotic stability of the TB-endemic equilibrium, E_T^*, exists if $R_T > 1$.*

Proof. The local and asymptotic stability of E_T^* is established using the trace and determinant method. The approach requires that the trace of the Jacobian matrix $J(E_T^*)$ is less than zero and the determinant of the Jacobian matrix $J(E_T^*)$ is greater than zero.

The Jacobian matrix $J(E_T)$ of the TB-only submodel is given by

$$J(E_T) = \begin{bmatrix} -(\lambda_T + \mu) & \eta & -\xi_1 q_1 \dfrac{S}{N_T} \\ \lambda_T & -k_1 & \xi_1 q_1 \dfrac{S}{N_T} \\ 0 & \theta & -k_2 \end{bmatrix}. \quad (63)$$

Evaluating the Jacobian matrix (63) at E_T^* gives

$$J(E_T^*) = \begin{bmatrix} -(\lambda_T^* + \mu) & \eta & \dfrac{-\xi_1 q_1}{R_T} \\ \lambda_T^* & -k_1 & \dfrac{\xi_1 q_1}{R_T} \\ 0 & \theta & -k_2 \end{bmatrix} \quad (64)$$

Now,

$$\operatorname{tr}(J(E_T^*)) = -(\lambda^* + \mu + k_1 + k_2) < 0. \quad (65)$$

Then,

$$\begin{aligned} \det(J(E_T^*)) &= 2\frac{\lambda_T^* q_1 \theta \xi_1}{R_T} + \frac{\mu q_1 \theta \xi_1}{R_T} + \eta k_2 \lambda_T^* - k_1 k_2 \lambda_T^* - k_1 k_2 \mu \\ &= \frac{q_1 \theta \xi_1}{R_T}(2\lambda_T^* + \mu) + \eta k_2 \lambda_T^* - k_1 k_2 (\lambda_T^* + \mu) \\ &= k_1 k_2 (R_T - 1)(\lambda_T^* + \mu) + \lambda_T^* \left(\frac{q_1 \theta \xi_1}{R_T} + \eta k_2 \right). \end{aligned} \quad (66)$$

Since

$$\operatorname{tr}(J(E_T^*)) = -(\lambda^* + \mu + k_1 + k_2) < 0 \quad (67)$$

and the determinant is positive when $R_T > 1$, then the TB-endemic equilibrium, E_T^*, is locally asymptotically stable.

3.2.6. Global Stability of the TB-Endemic Equilibrium Point for the TB-Only Submodel. To investigate the global stability of E_T^*, we proceed with the same approach used in [12].

Lemma 7. *If $R_T > 1$, then the TB-endemic equilibrium E_T^* of TB-only submodel is globally asymptotically stable.*

Proof. The global and asymptotic stability of TB-endemic equilibrium E_T^* is analysed using the following constructed Lyapunov function by Cai et al. [25].

Let the Lyapunov function be

$$\begin{aligned} L(S^*, I_L^*, I_T^*) = L_1\left(S - S^* - S^* In\left(\frac{S^*}{S}\right)\right) \\ + L_2\left(I_L - I_L^* - I_L^* In\left(\frac{I_L^*}{I_L}\right)\right) \\ + L_3\left(I_T - I_T^* - I_T^* In\left(\frac{I_T^*}{I_T}\right)\right). \end{aligned} \quad (68)$$

Taking derivative of the Lyapunov function L with respect to time along the positive solution of the above equation, we obtain

$$\begin{aligned} \frac{dL}{dt} &= L_1\left(1 - \frac{S^*}{S}\right)\frac{dS}{dt} + L_2\left(1 - \frac{I_L^*}{I_L}\right)\frac{dI_L}{dt} + L_3\left(1 - \frac{I_T^*}{I_T}\right)\frac{dI_T}{dt}, \\ &= L_1\left(1 - \frac{S^*}{S}\right)\left(\Lambda + \eta I_L - \frac{\xi_1 q_1 I_T S}{N_T} - \mu S\right) \\ &\quad + L_2\left(1 - \frac{I_L^*}{I_L}\right)\left(\frac{\xi_1 q_1 I_T S}{N_T} - k_1 I_L\right) \\ &\quad + L_3\left(1 - \frac{I_T^*}{I_T}\right)(\theta I_L - k_2 I_T). \end{aligned} \quad (69)$$

At the TB-endemic equilibrium, we have

$$\Lambda = \frac{\xi_1 q_1 I_L^* S^*}{N_T^*} + \mu S^* - \eta I_L^*, \quad (70)$$

$$k_1 = \frac{\xi_1 q_1 I_T^* S^*}{N_T^* I_L^*}, \quad (71)$$

$$k_2 = \frac{\theta I_L^*}{I_T^*}. \quad (72)$$

Substituting (70)–(72) into (69), we get

$$\begin{aligned} \frac{dL}{dt} &= L_1\left(1 - \frac{S^*}{S}\right)\left(\frac{\xi_1 q_1 I_L^* S^*}{N_T^*} + \mu S^* - \eta I_L^* \right. \\ &\quad \left. + \eta I_L - \frac{\xi_1 q_1 I_T S}{N_T} - \mu S\right) \\ &\quad + L_2\left(1 - \frac{I_L^*}{I_L}\right)\left(\frac{\xi_1 q_1 I_T S}{N_T} - \frac{\xi_1 q_1 I_T^* S^*}{N_T^* I_L^*} I_L\right) \\ &\quad + L_3\left(1 - \frac{I_T^*}{I_T}\right)\left(\theta I_L - \frac{\theta I_L^*}{I_T^*} I_T\right). \end{aligned} \quad (73)$$

Expanding and putting together terms of similar signs in equation (73), we have

$$\begin{aligned}\frac{dL}{dt} &= \frac{\xi_1 q_1 I_T^* S^* L_1}{N_T^*} + \mu S^* L_1 + \eta I_L L_1 - \frac{\xi_1 q_1 I_T^* S^{*2} L_1}{N_T S}\\ &- \frac{\mu S^{*2} L_1}{S} - \frac{\eta I_L S^* L_1}{S} + \frac{\xi_1 q_1 I_T S L_2}{N_T}\\ &- \frac{\xi_1 q_1 I_T S I_L^* L_2}{I_L N_T} + \theta I_L L_3 - \frac{\theta I_L I_T^* L_3}{I_T} - L_1 \eta I_L^*\\ &- \frac{\xi_1 q_1 I_T S L_1}{N_T} - \mu S L_1 + \frac{\eta S^* I_L^* L_1}{S} + \frac{\xi_1 q_1 I_T S^* L_1}{N_T}\\ &+ \mu S^* L_1 - \frac{\xi_1 q_1 I_T^* S^*}{N_T^* I_L^*} I_L L_2 +\\ &\frac{\xi_1 q_1 I_T^* S^*}{N_T^*} L_2 - \frac{\theta I_L^*}{I_T^*} I_T L_3 + \theta I_L^* L_3.\end{aligned} \tag{74}$$

$$\frac{dL}{dt} = A - B,$$

where

$$\begin{aligned}A &= \frac{\xi_1 q_1 I_T^* S^* L_1}{N_T^*} + \mu S^* L_1 + \eta I_L L_1 + \frac{\xi_1 q_1 I_T S L_2}{N_T}\\ &+ \theta I_L L_3 + \frac{\eta S^* I_L^* L_1}{S} + \frac{\xi_1 q_1 I_T S^* L_1}{N_T} + \mu S^* L_1\\ &+ \frac{\xi_1 q_1 I_T^* S^*}{N_T^*} L_2 + \theta I_L^* L_3,\end{aligned} \tag{75}$$

and

$$\begin{aligned}B &= \frac{\xi_1 q_1 I_T^* S^{*2} L_1}{N_T S} + \frac{\mu S^{*2} L_1}{S} + \frac{\eta I_L S^* L_1}{S} + \frac{\xi_1 q_1 I_T S I_L^* L_2}{I_L N_T}\\ &+ \frac{\theta I_L I_T^* L_3}{I_T} + L_1 \eta I_L^* + \frac{\xi_1 q_1 I_T S L_1}{N_T} + \mu S L_1\\ &+ \frac{\xi_1 q_1 I_T^* S^*}{N_T^* I_L^*} I_L L_2 + \frac{\theta I_L^*}{I_T^*} I_T L_3.\end{aligned} \tag{76}$$

Thus, if $A < B$, then we obtain that $dL/dt \le 0$, noting that $dL/dt = 0$ if and only if $S = S^*, I_L = I_L^*, I_T = I_T^*$.

Therefore, the largest compact invariant set in $\{(S^*, I_L^*, I_T^*) \in \Omega_T\colon dL/dt = 0\}$ is singleton $\{E_T^*\}$ where E_T^* is the endemic equilibrium point of the system (24). Thus, by Lasalle's invariance principle [26], it implies that E_T^* is globally asymptotically stable in Ω_T if $A < B$.

3.3. Analysis of the HCV-Only Submodel. By setting all the variables concerning TB infection in model system (4) to zero, we obtain the HCV-only submodel.

Let

$$I_L = I_T = I_{aL} = I_{aT} = I_{CL} = I_{CT} = \lambda_T = 0. \tag{77}$$

Then, the HCV-only submodel is given in the following equation.

$$\begin{aligned}\frac{dS}{dt} &= \Lambda + \pi I_a - (\lambda_H + \mu)S,\\ \frac{dI_a}{dt} &= \lambda_H S - (\pi + \alpha + \mu) I_a,\\ \frac{dI_C}{dt} &= \alpha I_a - (\mu + \delta) I_C,\end{aligned} \tag{78}$$

with

$$S(0) = S_0 \ge 0, I_a(0) = I_{a0} \ge 0, I_C(0) = I_{C0} \ge 0, \tag{79}$$

as the initial conditions

$$N_H = S + I_a + I_C \text{ as the total population} \tag{80}$$

and

$$\lambda_H = \frac{\xi_2 q_2 (\phi I_a + I_C)}{N_H} \text{ as the force of infection.} \tag{81}$$

Based on biological considerations, the submodel system (78) shall be studied in the following region:

$$\Omega = \left\{ (S, I_a, I_C) \in \mathbb{R}_+^3\colon 0 \le N_H \le \frac{\Lambda}{\mu} \right\}. \tag{82}$$

It can easily be shown that the solutions S, I_a, I_C of the submodel system (78) are positive for $t \ge 0$ and that the region Ω_H is positively invariant and solutions starting in Ω_H approach, enter, or stay in Ω_H.

3.3.1. The Disease-Free Equilibrium Point and Reproduction Number for the HCV-Only Submodel. To determine the HCV-free equilibrium point, we assume that there is no HCV infection in the community. Now, by equating system (78) to zero, the HCV-free equilibrium for the HCV-only submodel is determined as

$$E_H^0 = \left(S^0, I_a^0, I_C^0\right) = \left(\frac{\Lambda}{\mu}, 0, 0\right). \tag{83}$$

The basic reproduction number, R_H, for the submodel system (78) is defined as the number of secondary HCV cases produced by one HCV positive human during their entire life.

Applying the method of the next generation matrix for calculating the basic reproduction number as proposed in [23], the matrices for the rate of emergence of new infections in compartment i, F_i, and for the rate of movement into and out of compartment i by all other ways, V_i, for the HCV-only submodel in (78) are obtained as follows.

The infected compartments are I_a and I_C. Thus, we have

$$F_i = \begin{bmatrix} \lambda_H S \\ 0 \end{bmatrix} \tag{84}$$

and

$$V_i = \begin{bmatrix} (\pi + \alpha + \mu)I_a \\ -\alpha I_a + (\mu + \delta)I_C \end{bmatrix}. \quad (85)$$

The matrix of linearization of the new HCV infections, F_H, computed at E_H^0 is

$$F_H = \begin{bmatrix} \xi_2 q_2 \phi & \xi_2 q_2 \\ 0 & 0 \end{bmatrix}, \quad (86)$$

and that for the rate of movement into and out of the compartment i by all other ways V_H at E_H^0 is

$$V_H = \begin{bmatrix} (\pi + \alpha + \mu) & 0 \\ -\alpha & (\mu + \delta) \end{bmatrix}. \quad (87)$$

Thus, the basic reproduction number for the HCV-only submodel, R_H, is given as

$$R_H = \frac{\xi_2 q_2 (\phi(\mu + \delta) + \alpha)}{(\pi + \alpha + \mu)(\mu + \delta)}. \quad (88)$$

Therefore, it can be deduced that an increase in HCV transmission probability q_2 and the average number of HCV contact persons, ξ_2, per year, leads to an increase in the HCV secondary infections. An increase in the average period a human remains latently infected with HCV increases the number of secondary HCV infections. Increase in the natural recovery of acute HCV humans, π, leads to reduced number of secondary HCV infections. Thus, intervention measures should target reducing both the average number of HCV contact persons, ξ_2, and HCV transmission probability, q_2, while increasing the natural recovery rate of HCV acute humans, π. This conclusion is in agreement with [16].

3.3.2. Local Stability of the HCV-Free Equilibrium Point. Using Theorem 2 from [23], the subsequent result is proved.

Lemma 8. *The local and asymptotic stability of HCV infection-free equilibrium point E_H^0 exists if $R_H < 1$.*

Proof. The local and asymptotic stability of HCV infection-free equilibrium exists if and only if all the eigenvalues of the Jacobian matrix at E_H^0 have negative real parts. The Jacobian matrix $J(E_H)$ of the HCV-only submodel (78) at E_H^0 is given by

$$J(E_H^0) = \begin{bmatrix} -\mu & \pi - \xi_2 q_2 \phi & -\xi_2 q_2 \\ 0 & \xi_2 q_2 \phi - k_3 & \xi_2 q_2 \\ 0 & \alpha & -k_4 \end{bmatrix}, \quad (89)$$

where

$$k_3 = \pi + \alpha + \mu \text{ and } k_4 = \mu + \delta. \quad (90)$$

The eigenvalues of the characteristic equation are given by

$$\lambda_1 = -\mu, \lambda_2 = \frac{-(k_4 - (\xi_2 q_2 \phi - k_3)) - \sqrt{Q}}{2}$$
$$\text{and } \lambda_3 = \frac{-(k_4 - (\xi_2 q_2 \phi - k_3)) + \sqrt{Q}}{2}, \quad (91)$$

where $Q = (k_4 - (\xi_2 q_2 \phi - k_3))^2 + 4((\xi_2 q_2 \phi - k_3)k_4 + \alpha \xi_2 q_2)$. Clearly, λ_1 and λ_2 have negative real parts. However, the real part of λ_3 is negative when

$$\frac{\sqrt{(k_4 - (\xi_2 q_2 \phi - k_3))^2 + 4((\xi_2 q_2 \phi - k_3)k_4 + \alpha \xi_2 q_2)}}{2} < 0. \quad (92)$$

On simplifying the above inequality, we have

$$\xi_2 q_2 (\phi k_4 + \alpha) - k_3 k_< - \frac{(k_3 + k_4 - \xi_2 q_2 \phi)^2}{4} \quad (93)$$

But

$$\xi_2 q_2 (\phi k_4 + \alpha) = R_H k_3 k_4. \quad (94)$$

Therefore,

$$R_H < \left(1 - \frac{(k_3 k_4 - \xi_2 q_2 \phi)^2}{4 k_3 k_4}\right) < 1. \quad (95)$$

Hence, E_H^0 is locally asymptotically stable if and only if $R_H < 1$.

3.3.3. Global Stability of HCV Infection-Free Equilibrium for the HCV-Only Submodel

Lemma 9. *The HCV-free equilibrium point E_H^0 of the model (78) is globally asymptotically stable if $R_H \leq 1$.*

Proof. Let $L_2 = Q_1 I_a + Q_2 I_C$ be the Lyapunov function that contains humans who participate in proliferation of the HCV infection in the community, with Q_1 and Q_2 being random positive constants. The derivative of the Lyapunov function with respect to time is computed as

$$\begin{aligned} \frac{dL_2}{dt} &= Q_1(\lambda_H S - k_3 I_a) + Q_2(\alpha I_a - k_4 I_c) \\ &= Q_1\left(\xi_2 q_2 (\phi I_a + I_C)\frac{S}{N} - k_3 I_a\right) + Q_2(\alpha I_a - k_4 I_C). \end{aligned} \quad (96)$$

Since $S/N \leq 1$,

$$\frac{dL_2}{dt} \leq ((\xi_2 q_2 \phi - k_3) + \alpha Q_2)I_a + (\xi_2 q_2 Q_1 - k_4 Q_2)I_C. \quad (97)$$

Since constants Q_1 and Q_2 are arbitrarily chosen and are positive, we can let $Q_2 = \xi_2 q_2 Q_1 / k_4$. Thus, inequality (97) becomes

$$\frac{dL_2}{dt} \le \frac{Q_1 I_a}{k_4}\left(\xi_2 q_2\left(\phi k_4 + \alpha\right) - k_3 k_4\right). \tag{98}$$

But

$$\xi_2 q_2\left(\phi k_4 + \alpha\right) = k_3 k_4 R_H. \tag{99}$$

Thus, inequality (98) simplifies to

$$\frac{dL_2}{dt} \le Q_1 k_3 I_a\left(R_H - 1\right). \tag{100}$$

Therefore, $dL_2/dt \le 0$ whenever $R_H \le 1$. In addition, $dL_2/dt = 0$ if either $I_a = I_C = 0$ or $R_H = 1$.

In both cases, the greatest compact invariant set of $\Omega_H = \{(S(t), I_a(t), I_C(t) \in \mathbb{R}_+^3)\colon dL_2/dt = 0\}$ is the singleton E_H^0. Thus, Lasalle's invariance principle suggests that provided $R_H \le 1$, E_H^0 is globally asymptotically stable.

3.3.4. The Endemic Equilibrium Point for the HCV-Only Submodel. By considering the persistence of HCV infection in the population, we determine the HCV-endemic equilibrium point $E_H^* = (S^*, I_a^*, I_C^*)$. Equating the derivatives of sub model system (78) to zero, we get

$$\Lambda + \pi I_a^* - (\lambda_H^* + \mu)S^* = 0, \tag{101}$$

$$\lambda_H^* S^* - (\pi + \alpha + \mu)I_a^* = 0, \tag{102}$$

$$\alpha I_a^* - (\mu + \delta)I_C^* = 0, \tag{103}$$

with

$$\lambda_H^* = \frac{\xi_2 q_2\left(\phi I_a^* + I_C^*\right)}{N_H^*} \tag{104}$$

and

$$N_H^* = S^* + I_a^* + I_C^*. \tag{105}$$

Thus, the endemic equilibrium point for the HCV-only submodel is given by

$$E_H^* = \left(\frac{N^* k_3 k_4}{\xi_2 q_2\left(\phi k_4 + \alpha\right)}, \frac{\mu N^* k_3 k_4 - \Lambda \xi_2 q_2\left(\phi k_4 + \alpha\right)}{\xi_2 q_2\left(\phi k_4 + \alpha\right)\left(\pi - k_3\right)}, \frac{\alpha\mu N^* k_3 k_4 - \alpha\Lambda \xi_2 q_2\left(\phi k_4 + \alpha\right)}{\xi_2 q_2 k_4\left(\phi k_4 + \alpha\right)\left(\pi - k_3\right)}\right). \tag{106}$$

Lemma 10. *Whenever $R_H > 1$, then HCV-only submodel (78) has a distinctive endemic equilibrium point.*

Proof. If the infection stays in the population for some time, then

$$\frac{dI_a}{dt} > 0 \text{ and } \frac{dI_C}{dt} > 0, \tag{107}$$

as done in Lemma 5, that is,

$$\frac{\xi_2 q_2 (\phi I_a + I_C)S}{N_H} - k_3 I_a > 0, \tag{108}$$

and

$$\alpha I_a - k_4 I_C > 0. \tag{109}$$

From inequality (108), we have

$$k_3 I_a < \xi_2 q_2\left(\phi I_a + I_C\right)\frac{S}{N_H}. \tag{110}$$

Using the fact that $S/N_H \le 1$,

$$I_a < \frac{\xi_2 q_2\left(\phi I_a + I_C\right)}{k_3}. \tag{111}$$

From inequality (109), we have

$$I_C < \frac{\alpha I_a}{k_4}. \tag{112}$$

Substituting (112) into (111) and simplifying, we get

$$1 < \frac{\alpha \xi_2 q_2}{k_3 k_4 - k_4 \xi_2 q_2 \phi}. \tag{113}$$

Thus,

$$R_H > 1. \tag{114}$$

Thus, whenever $R_H > 1$, a distinctive endemic equilibrium E_H^* exists.

3.3.5. Local Stability of HCV-Endemic Equilibrium for the HCV-Only Submodel

Lemma 11. *If $R_H > 1$, then the endemic equilibrium E_H^* of the system (78) is locally asymptotically stable in Ω_H.*

Proof. In order to determine the local and asymptotic stability of E_H^*, the Jacobian matrix of HCV-only submodel at E_H^* should have a negative trace and a positive determinant. Evaluating the Jacobian matrix $J(E_H)$ of the HCV submodel (78) at the endemic equilibrium gives

$$J\left(E_H^*\right) = \begin{bmatrix} -(\lambda_H^* + \mu) & \left(\pi - \dfrac{\xi_2 q_2 \phi}{R_H}\right) & \dfrac{\xi_2 q_2}{R_H} \\ \lambda_H^* & \left(\dfrac{\xi_2 q_2 \phi}{R_H} - k_3\right) & \dfrac{\xi_2 q_2}{R_H} \\ 0 & \alpha & -k_4 \end{bmatrix}, \tag{115}$$

where λ_H^* is defined as the rate at which susceptible humans acquire HCV infection, evaluated at the endemic equilibrium point.

Now,

$$\operatorname{tr}\left(J\left(E_H^*\right)\right)=\frac{\xi_2 q_2 \phi}{R_H}-\left(\lambda_H^*+\mu+k_3+k_4\right). \quad (116)$$

For negative trace,

$$\frac{\xi_2 q_2 \phi}{R_H}-\left(\lambda_H^*+\mu+k_3+k_4\right)<0. \quad (117)$$

From the above inequality, we have

$$\frac{R_H\left(\lambda_H^*+\mu+k_3+k_4\right)}{\xi_2 q_2 \phi}>1 \quad (118)$$

Next, we consider

$$\begin{aligned}\det\left(J\left(E_H^*\right)\right)=&\frac{\alpha\mu\xi_2 q_2}{R_H}+\frac{k_4\mu\xi_2 q_2\phi}{R_H}\\&+k_4\pi\lambda_H^*-k_3k_4\left(\lambda_H^*+\mu\right).\end{aligned} \quad (119)$$

Thus, $\det\left(J\left(E_H^*\right)\right)>0$ when

$$\frac{\alpha\mu\xi_2 q_2+k_4\mu\xi_2 q_2\phi+k+4\pi\lambda_H^* R_H}{\xi_2 q_2\left(\phi k_4+\alpha\right)\left(\lambda_H^*+\mu\right)}>1. \quad (120)$$

However, inequalities (118) and (120) hold when $R_H>1$.
Hence, the HCV endemic equilibrium, E_H^*, is locally asymptotically stable whenever $R_H>1$ and unstable otherwise.

3.3.6. Global and Asymptotic Stability of HCV Endemic Equilibrium for the HCV-Only Submodel. To establish the global and asymptotic stability of the HCV-endemic equilibrium point E_H^*, we use the same approach as in Lemma 7.

Lemma 12. *If $R_H>1$, then the global and asymptotic stability of the HCV endemic equilibrium E_H^* of submodel system (78) exists.*

Proof. Let the Lyapunov function $U=U\left(S,I_a,I_C\right)$ be defined as

$$\begin{aligned}U=&U_1\left(S-S^*-S^* In\left(\frac{S^*}{S}\right)\right)\\&+U_2\left(I_a-I_a^*-I_a^* In\left(\frac{I_a^*}{I_a}\right)\right)\\&+U_3\left(I_C-I_C^*-I_C^* In\left(\frac{I_C^*}{I_C}\right)\right).\end{aligned} \quad (121)$$

Taking derivative of the Lyapunov function U with respect to time along the positive solution of the above system, we obtain

$$\begin{aligned}\frac{dU}{dt}=&U_1\left(1-\frac{S^*}{S}\right)\frac{dS}{dt}+U_2\left(1-\frac{I_a^*}{I_a}\right)\frac{dI_a}{dt}\\&+U_3\left(1-\frac{I_C^*}{I_C}\right)\frac{dI_C}{dt},\\=&U_1\left(1-\frac{S^*}{S}\right)\left(\Lambda+\pi I_a+\frac{\xi_2 q_2\left(\phi I_a+I_C\right)S}{N_H}-\mu S\right)\\&+U_2\left(1-\frac{I_a^*}{I_a}\right)\left(\frac{\xi_2 q_2\left(\phi I_a+I_C\right)S}{N_H}-k_3 I_a\right)\\&+U_3\left(1-\frac{I_C^*}{I_C}\right)\left(\alpha I_a-k+4I_C\right).\end{aligned} \quad (122)$$

At the HCV endemic equilibrium, we have

$$\begin{aligned}\Lambda&=-\pi I_a^*+\frac{\xi_2 q_2\left(\phi I_a^*+I_C^*\right)S^*}{N_H^*}+\mu S^*,\\k_3&=\frac{\xi_2 q_2\left(\phi I_a^*+I_C^*\right)S^*}{N_H^* I_a^*},\\k_4&=\alpha\frac{I_a^*}{I_C^*}.\end{aligned} \quad (123)$$

Now, substituting for Λ, k_3, and k_4, we have

$$\begin{aligned}\frac{dU}{dt}=&U_1\left(1-\frac{S^*}{S}\right)\left(-\pi I_a^*+\frac{\xi_2 q_2\left(\phi I_a^*+I_C^*\right)S^*}{N_H^*}+\mu S^*\right.\\&\left.+\pi I_a+\frac{\xi_2 q_2\left(\phi I_a+I_C\right)S}{N_H}-\mu S\right)\\&+U_2\left(1-\frac{I_a^*}{I_A}\right)\left(\frac{\xi_2 q_2\left(\phi I_a+I_C\right)S}{N_H}-\frac{\xi_2 q_2\left(\phi I_a^*+I_C^*\right)S^*}{N_H^* I_a^*}I_a\right)\\&+U_3\left(1-\frac{I_C^*}{I_C}\right)\left(\alpha I_a-\alpha\frac{I_a^*}{I_C^*}I_C\right).\end{aligned} \quad (124)$$

Expanding and collecting the positive terms together and the negative terms together, we have

$$\frac{dU}{dt}=C-D, \quad (125)$$

where

$$C = \frac{\xi_2 q_2 (\phi I_a^* + I_C^*) S^* U_1}{N_H^*} + \mu S^* U_1 + \pi I_a U_1 + \frac{\pi I_a^* S^* U_1}{S} + \frac{\xi_2 q_2 (\phi I_a + I_C) S^* U_1}{S N_H} + \mu S^* U_1 + \frac{\xi_2 q_2 (\phi I_a + I_C) S U_2}{N_H} + \frac{\xi_2 q_2 (\phi I_a^* + I_C^*) S^* U_2}{N_H^*} + \alpha I_a U_3 + \alpha I_a^* U_3, \tag{126}$$

and

$$D = \pi I_a^* U_1 + \frac{\xi_2 q_2 (\phi I_a + I_C) U_1}{N_H} + \mu S U_1 + \frac{\xi_2 q_2 (\phi I_a^* + I_C^*) S^* U_1}{S N_H^*} + \frac{\mu S^{*2} U_1}{S} + \frac{\pi I_a S^* U_1}{S} + \frac{\xi_2 q_2 (\phi I_a^* + I_C^*) S^* I_a U_2}{N_H^* I_a^*} + \frac{\xi_2 q_2 (\phi I_a + I_C) S^* I_a^* U_2}{N_H I_a} + \frac{\alpha I_a^* I_C U_3}{I_C^*} + \frac{\alpha I_a I_C^* U_3}{I_C}. \tag{127}$$

Hence, if $C < D$, then we obtain that $dU/dt \le 0$, with $dU/dt = 0$ if and only if $S = S^*, I_a = I_a^*, I_C = I_C^*$.

Therefore, the largest compact invariant set in $\{(S^*, I_a^*, I_C^*) \in \Omega_H\colon dU/dt = 0\}$ is singleton $\{E_H^*\}$, where E_H^* is the endemic equilibrium point of the system (78). Hence, Lasalle's invariance principle (Lasalle J, 1976) suggests that E_H^* is globally and asymptotically stable in Ω_H if $C < D$.

3.3.7. Analysis of the TB-HCV Coinfection Model. We then calculate the disease-free equilibrium point E^0 for the TB-HCV coinfection model system (4).

The disease-free equilibrium point, E^0, for the TB-HCV coinfection model is given by

$$E^0 = \left(S^0, I_L^0, I_T^0, I_a^0, I_C^0, I_{aL}^0, I_{CL}^0, I_{aT}^0, I_{CT}^0\right) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0, 0, 0, 0\right). \tag{128}$$

Next, we consider determining the basic reproduction number R_0 for the TB-HCV coinfection model.

Lemma 13. *The basic reproduction number R_0 for TB-HCV coinfection model is given by:*

$$R_0 = \max\{R_T, R_H\} \tag{129}$$

Proof. The basic reproduction number is calculated using the method of the next generation matrix proposed by [23] for model system (4). By determining the matrix, F_i, for the rate of emergence of new cases in component i and the matrix, V_i, for the rate of movement into and out of component i by all other means, we get the following:

$$F_i = \begin{bmatrix} \lambda_T S \\ 0 \\ \lambda_H S \\ 0 \\ \lambda_T I_a + \lambda_H I_L \\ \lambda_T I_C \\ \lambda_H I_T \\ 0 \end{bmatrix} \tag{130}$$

and

$$V_i = \begin{bmatrix} (\theta + \eta + \mu + \lambda_H) I_L \\ -\theta I_L + (\mu + \sigma + \lambda_H) I_T \\ (\pi + \alpha + \mu + \lambda_T) I_a \\ -\alpha I_a + (\mu + \delta + \lambda_T) I_C \\ (\tau_1 + \mu + \beta_1) I_{aL} \\ -\beta_1 I_{aL} + (\tau_2 + \mu + \delta) I_{CL} \\ -\tau_1 I_{aL} + (\beta_2 + \mu + d_1) I_{aT} \\ -\tau_2 I_{CL} - \beta_2 I_{aT} + (\mu + d_2) I_{CT} \end{bmatrix}. \tag{131}$$

Now, the Jacobian matrix, F, of the new cases at the disease-free equilibrium, E^0, is determined as

$$F(E^0) = \begin{bmatrix} 0 & \xi_1 q_1 & 0 & 0 & 0 & 0 & \xi_1 q_1 a_1 & \xi_1 q_1 a_2 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \xi_2 q_2 \phi & \xi_2 q_2 & \xi_2 q_2 b_1 & \xi_2 q_2 b_2 & \xi_2 q_2 b_3 & \xi_2 q_2 b_4 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}. \tag{132}$$

Then, the Jacobian matrix, V, for the rate of movement from one compartment to another at disease-free equilibrium point, E^0, is determined as

$$V\left(E^0\right)=\begin{bmatrix}(\theta+\eta+\mu) & 0 & 0 & 0 & 0 & 0 & 0 & 0\\ -\theta & (\mu+\sigma) & 0 & 0 & 0 & 0 & 0 & 0\\ 0 & 0 & (\pi+\alpha+\mu) & 0 & 0 & 0 & 0 & 0\\ 0 & 0 & -\alpha & (\mu+\delta) & 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0 & (\tau_1+\mu+\beta_1) & 0 & 0 & 0\\ 0 & 0 & 0 & 0 & -\beta & (\tau_2+\mu+\delta) & 0 & 0\\ 0 & 0 & 0 & 0 & -\tau_1 & 0 & (\beta_2+\mu+d_1) & 0\\ 0 & 0 & 0 & 0 & 0 & -\tau_2 & -\beta_2 & (\mu+d_2)\end{bmatrix}. \tag{133}$$

Then,

$$FV^{-1}=\begin{bmatrix}\frac{\xi_1 q_1\theta}{(\theta+\eta+\mu)(\mu+\sigma_1)} & \frac{\xi_1 q_1}{(\mu+\sigma)} & 0 & 0 & \mathrm{K} & \frac{\xi_1 q_1 a_2\tau_2}{(\tau_2+\mu+\delta)(\mu+d_2)} & \frac{\xi_1 q_1(a_1(\mu+d_2)+a_2\beta_2)}{(\eta_2+\mu+d_1)(\mu+d_2)} & \frac{\xi_1 q_1 a_2}{(\mu+d_2)}\\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\ 0 & 0 & \frac{\xi_2 q_2(\phi(\mu+\delta)+\alpha)}{(\pi+\alpha+\mu)(\mu+\delta)} & \frac{\xi_2 q_2}{(\mu+\delta)} & \mathrm{T} & \frac{\xi_2 q_2(b_2(\mu+d_2)+b_4\tau_2)}{(\tau_2+\mu+\delta)(\mu+d_2)} & \frac{\xi_2 q_2(b_3(\mu+d_2)+b_4\beta_2)}{(\beta_2+\mu+d_1)(\mu+d_2)} & \frac{\xi_2 q_2 b_4}{(\mu+d_2)}\\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\end{bmatrix}, \tag{134}$$

where $\mathrm{K} = \tau_1\xi_1 q_1 a_1(\tau_2+\mu+\delta)(\mu+d_2)+\xi_1 q_1 a_2(\beta_2\tau_1(\tau_2+\mu+\delta)+\beta_1\tau_2(\beta_2+\mu+d_1))/(\tau_1+\mu+\beta_1)(\tau_2+\mu+\delta)\quad(\beta_2+\mu+d_1)(\mu+d_2)$, and

$$\mathrm{T}=\frac{\xi_2 q_2(\beta_2+\mu+d_1)(\mu+d_1)(b_1(\tau_2+\mu+\delta)+b_2\beta_1)+\xi_2 q_2(b_3\tau_1(\tau_2+\mu+\delta)(\mu+d_2)+b_4(\beta_2\tau_1(\beta_2\tau_1(\tau_2+\mu+\delta)+\beta_1\tau_2(\beta_2+\mu+d_1))))}{(\tau_1+\mu+\beta_1)(\tau_2+\mu+\delta)(\beta_2+\mu+d_2)(\mu+d_2)} \tag{135}$$

On solving for the eigenvalues, the dominant eigenvalues for the matrix FV^{-1} are

$$\lambda_1=\frac{\xi_1 q_1\theta}{(\mu+\sigma)(\theta+\eta+\mu)}\ \text{and}\ \lambda_2=\frac{\xi_2 q_2(\phi(\mu+\delta)+\alpha)}{(\pi+\alpha+\mu)(\mu+\delta)}. \tag{136}$$

However, these correspond to the reproduction numbers for the TB infection submodel and HCV infection submodel, respectively. Thus, the basic reproduction number, R_0, for the TB-HCV coinfection model is given by

$$R_0=\max\{R_T,R_H\}. \tag{137}$$

This implies that if $R_T > R_H$, then the dynamics of the coinfection is dependent on TB and vice versa. It is noted that in absence of TB, $R_0 = R_H$ and in absence of HCV, $R_0 = R_T$.

Using Theorem 2 from [23], the subsequent result is proved.

Lemma 14. *The local and asymptotic stability of the disease-free equilibrium point, E^0, of model system (4) exists if $R_0 < 1$.*

Proof. The local and asymptotic stability of the disease-free equilibrium, E^0, is determined by computing the Jacobian matrix of the TB-HCV coinfection model system (4) and establishing the signs of the eigenvalues of its submatrices, in the upper left corner, J_{11}, the inner submatrix, J_{22}, and the lower right hand corner submatrix, J_{33}.

The local and asymptotically stability exists if and only if all the eigenvalues of $J_{11}, J_{22,}$ and J_{33} have negative real parts [16, 27].

The Jacobian matrix of the TB-HCV coinfection model at E^0 is given by

$$J\left(E^0\right) = \begin{bmatrix} -\mu & \eta & \xi_1 q_1 & (\pi - \xi_2 q_2 \phi) & -\xi_2 q_2 & -\xi_2 q_2 b_1 & -\xi_2 q_2 b_2 & -(\xi_1 q_1 a_1 + \xi_2 q_2 b_3) & -(\xi_1 q_1 a_2 + \xi_2 q_2 b_4) \\ 0 & -(\theta + \eta + \mu) & \xi_1 q_1 & 0 & 0 & 0 & 0 & \xi_1 q_1 a_1 & \xi_1 q_1 a_2 \\ 0 & \theta & -(\mu + \sigma) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & x_1 & \xi_2 q_2 & \xi_2 q_2 b_1 & \xi_2 q_2 b_2 & \xi_2 q_2 b_3 & \xi_2 q_2 b_4 \\ 0 & 0 & 0 & \alpha & -(\mu + \delta) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -x_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \beta_1 & -(\tau_2 + \mu + \delta) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \tau_1 & 0 & -(\beta_2 + \mu + d_1) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \tau_2 & \beta_2 & -(\mu + d_2) \end{bmatrix}, \tag{138}$$

where

$$x_1 = (\xi_2 q_2 \phi - (\pi + \alpha + \mu)) \text{ and } x_2 = (\tau_1 + \mu + \beta_1). \tag{139}$$

We now rewrite the Jacobian matrix, J, at E^0 as

$$J\left(E^0\right) = \begin{bmatrix} J_{11} & J_{12} & J_{13} \\ J_{21} & J_{22} & J_{23} \\ J_{31} & J_{32} & J_{33}, \end{bmatrix} \tag{140}$$

where

$$\begin{aligned} J_{11} &= \begin{bmatrix} -\mu & \eta & \xi_1 q_1 \\ 0 & -(\theta + \eta + \mu) & \xi_1 q_1 \\ 0 & \theta & -(\mu + \sigma) \end{bmatrix}, \\ J_{12} &= \begin{bmatrix} (\pi - \xi_2 q_2 \phi) & -\xi_2 q_2 & -\xi_2 q_2 b_1 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \\ J_{13} &= \begin{bmatrix} -\xi_2 q_2 b_2 & -(\xi_1 q_1 a_1 + \xi_2 q_2 b_3) & -(\xi_1 q_1 a_2 + \xi_2 q_2 b_4) \\ 0 & \xi_1 q_1 a_1 & \xi_1 q_1 a_2 \\ 0 & 0 & 0 \end{bmatrix} \\ J_{21} &= \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \\ J_{22} &= \begin{bmatrix} \kappa & \xi_2 q_2 & \xi_2 q_2 b_1 \\ \alpha & -(\mu + \delta) & 0 \\ 0 & 0 & (\tau_1 + \mu + \beta_1) \end{bmatrix}, \\ J_{23} &= \begin{bmatrix} \xi_2 q_2 b_2 & \xi_2 q_2 b_3 & \xi_2 q_2 b_4 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \end{aligned} \tag{141}$$

where $\kappa = (\xi_2 q_2 \phi - (\pi + \alpha + \mu))$ and

$$J_{31} = \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, J_{32} = \begin{bmatrix} (0 & 0 & \beta_1 \\ 0 & 0 & \tau_1 \\ 0 & 0 & 0 \end{bmatrix},$$
$$J_{33} = \begin{bmatrix} -(\tau_2 + \mu + \delta) & 0 & 0 \\ 0 & -(\beta_2 + \mu + d_1) & 0 \\ \tau_2 & \tau_2 & -(\mu + d_2) \end{bmatrix}. \quad (142)$$

Then, we continue to determine the eigenvalues of the submatrices $J_{11}, J_{22,}$ and J_{33} to establish their signs.

For J_{33}, the corresponding eigenvalues are

$$-(\tau_2 + \mu + \delta), -(\beta_2 + \mu + d_1) \text{ and } -(\mu + d_2). \quad (143)$$

We observe that all the eigenvalues of J_{33} are negative.

For J_{22}, the eigenvalues are

$$\xi_2 q_2 \phi - (\pi + \alpha + \mu), -(\mu + \delta) \text{ and } -(\tau_1 + \mu + \beta_1). \quad (144)$$

We observe that the eigenvalues of J_{22} are all negative when

$$\xi_2 q_2 \phi < (\pi + \alpha + \mu) \quad (145)$$

From which,

$$\frac{\xi_2 q_2 \phi}{(\pi + \alpha + \mu)} = \frac{(\mu + \delta) R_H}{\phi(\mu + \delta) + \alpha} < 1 \quad (146)$$

since

$$(\pi + \alpha + \mu) = \frac{\xi_2 q_2 (\phi(\mu + \delta) + \alpha)}{R_H (\mu + \delta)}. \quad (147)$$

However, inequality (146) is satisfied only if $R_H < 1$.

Finally, the corresponding eigenvalues of J_{11} are

$$-\mu, \frac{-(y_1 + y_2) - \sqrt{(y_1 + y_2)^2 - 4(y_1 y_2 - \xi_1 q_1 \theta)}}{2} \text{ and }$$
$$\frac{-(y_1 + y_2) + \sqrt{(y_1 + y_2)^2 - 4(y_1 y_2 - \xi_1 q_1 \theta)}}{2}, \quad (148)$$

where

$$y_1 = (\theta + \eta + \mu) \text{ and } y_2 = (\mu + \sigma). \quad (149)$$

The third eigenvalue is a negative when

$$\sqrt{(y_1 + y_2)^2 - 4(y_1 y_2 - \xi_1 q_1 \theta)} < 0$$
$$4\xi_1 q_1 \theta < 2 y_1 y_2 - (y_1^2 + y_2^2). \quad (150)$$

But

$$\xi_1 q_1 \theta = y_1 y_2 R_T. \quad (151)$$

Thus,

$$R_T < \frac{-(y_1 - y_2)^2}{4 y_1 y_2} < 1. \quad (152)$$

Therefore, if inequalities (146) and (152) are satisfied, then $R_0 < 1$ and hence the disease-free equilibrium point E^0 for model system (4) is locally asymptotically stable.

3.3.8. Global Stability of the Disease-Free Equilibrium Point for TB-HCV Coinfection. To understand the global behaviour of the system (4), we deploy an approach used by [24].

Our model system (4) is now expressed in the form

$$\frac{dX}{dt} = F(X, Y),$$
$$\frac{dY}{dt} = G(X, Y), G(X, 0) = 0, \quad (153)$$

where $X = (S)$ with $X \in \mathbb{R}_+$ denoting the number of uninfected humans and $Y = (I_L, I_T, I_a, I_C, I_{aL}, I_{CL}, I_{aT}, I_{CT})$ with $Y \in \mathbb{R}_+^8$ whose components denote the number of humans infected with TB-only or HCV-only or both TB and HCV.

Let the disease-free equilibrium of our system (4) be denoted by $E^0 = (X^0, 0) = (\Lambda/\mu, 0)$.

We have to establish that the subsequent conditions are fulfilled to guarantee global asymptotic stability.

$$(C1): \text{ For } \frac{dX}{dt} = F(X, 0), X^0 \text{ is globally asymptotically stable,}$$
$$(C2): G(X, Y) = AY - \widehat{G}(X, Y), \widehat{G}(X, Y) \geq 0, \text{for } (X, Y) \in \Omega, \quad (154)$$

where $A = D_Y G(X^0, 0)$ is a Metzler matrix (the off diagonal elements of Metzler are non-negative) and Ω is the region where the model makes biological meaning.

Thus, when system (153) satisfies conditions $(C1)$ and $(C2)$, we have the following theorem satisfied.

Theorem 15. *The equilibrium point* $E^0 = (X^0, 0)$ *is globally asymptotically stable point of system (153) provided* $R_0 < 1$ *and that conditions* $(C1)$ *and* $(C2)$ *are satisfied.*

Proof. From Lemma 14, E^0 is locally asymptotically stable if $R_0 < 1$.

For the first condition $(C1)$, that is, the global asymptotic stability of X^0, we have

$$F(X,Y) = [\Lambda + \pi I_a + \eta I_L - (\lambda_T + \lambda_H + \mu)S] \tag{155}$$

and

$$\frac{dX}{dt} = F(X,0) = \Lambda - \mu S, \tag{156}$$

which is a linear differential equation. Solving it, we get

$$S(t) = \frac{\Lambda}{\mu} - \frac{\Lambda}{\mu}e^{-\mu t} + S(0)e^{-\mu t}. \tag{157}$$

Now, as $t \longrightarrow \infty$, $S \longrightarrow \Lambda/\mu$ regardless of the value of $S(0)$. Thus, there is convergence in Ω implying that $(C1)$ holds.

For the second condition $(C2)$, consider

$$G(X,Y) = \begin{bmatrix} \lambda_T S - (\theta + \eta + \mu + \lambda_H) I_L \\ \theta I_L - (\mu + \sigma + \lambda_H) I_T \\ \lambda_H S - (\pi + \alpha + \mu + \lambda_T) I_a \\ \alpha I_a - (\mu + \delta + \lambda_T) I_C \\ \lambda_T I_a + \lambda_H I_L - (\tau_1 + \mu + \beta_1) I_{aL} \\ \beta_1 I_{aL} + \lambda_T I_C - (\tau_2 + \mu + \delta) I_{CL} \\ \tau_1 I_{aL} + \lambda_H I_T - (\beta_2 + \mu + d_1) I_{aT} \\ \tau_2 I_{CL} + \beta_2 I_{aT} - (\mu + d_2) I_{CT} \end{bmatrix}, \tag{158}$$

and

$$A = \begin{bmatrix} -(\theta+\eta+\mu) & \xi_1 q_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ \theta & -(\mu+\sigma) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & (\xi_2 q_2 \phi - (\pi+\alpha+\mu)) & \xi_2 q_2 & \xi_2 q_2 b_1 & \xi_2 q_2 b_2 & \xi_2 q_2 b_3 & \xi_2 q_2 b_4 \\ 0 & 0 & \alpha & -(\mu+\delta) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\tau_1+\mu+\beta_1) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta_1 & -(\tau_2+\mu+\delta) & 0 & 0 \\ 0 & 0 & 0 & 0 & \tau_1 & 0 & -(\beta_2+\mu+d_1) & 0 \\ 0 & 0 & 0 & 0 & 0 & \tau_2 & \beta_2 & -(\mu+d_2) \end{bmatrix}. \tag{159}$$

Now,

$$\widehat{G}(X,Y) = AY - G(X,Y). \tag{160}$$

Therefore,

$$\widehat{G}(X,Y) = \begin{bmatrix} \xi_1 q_1 I_T - \lambda_T S + \lambda_H I_L \\ \lambda_H I_T \\ \xi_2 q_2 (\phi I_a + I_C + I_{aL} + I_{CL} + b_3 I_{aT} + b_4 I_{CT}) - \lambda_H S + \lambda_T I_a \\ \lambda_T I_C \\ -\lambda_T I_a - \lambda_H I_L \\ -\lambda_T I_C \\ -\lambda_H I_T \\ 0 \end{bmatrix}. \tag{161}$$

Since $\widehat{G}_5(X,Y) < 0, \widehat{G}_6(X,Y) < 0$ and $\widehat{G}_7(X,Y) < 0$, then $\widehat{G}(X,Y) \ngeq 0$. This implies that condition $C2$ is not satisfied. Therefore, the disease-free equilibrium, $E^0 = (X^0, 0)$, may not be globally asymptotically stable for $R_0 < 1$. This indicates that a backward bifurcation will occur at $R_0 = 1$ as proved by Feng et al. [28]. Backward bifurcation in biological sense implies that a stable endemic equilibrium point shall exist at the same time with a stable infection-free equilibrium point whenever the basic reproduction number is less than one. Furthermore, when backward bifurcation exists, it explains a phenomenon due to the disease cannot be completely exterminated by merely decreasing the basic reproduction number to less than unity.

3.4. Existence of TB-HCV Coinfection Endemic Equilibrium. The endemic equilibrium point for TB-HCV coinfection model (4) does exist if $R_T > 1$ and $R_H > 1$, that is, $R_0 = \max\{R_T, R_H\} > 1$. However, the explicit computation of the endemic equilibrium for the TB-HCV coinfection in terms of the model parameters is analytically clumsy. Thus, the existence and stability are investigated through numerical simulations.

The starting values of the variables $S, I_L, I_T, I_a, I_C, I_{aL}, I_{CL}, I_{aT}$, *and* I_{CT} are changed to establish whether they settle to the same values greater than zero with time, regardless of the dissimilar starting values of the variables. In the numerical analysis, the values of the parameters used are shown in Table 3.

In Figures 2–4, the starting values of humans who are at a risk of contracting TB and HCV infections, $S(0)$; TB latently infected humans, $I_L(0)$; infectious TB humans, $I_T(0)$; acute HCV infectious humans, $I_a(0)$; chronic HCV infectious humans, $I_C(0)$; TB latent and acute HCV coinfected humans, $I_{aL}(0)$; TB latent and chronic HCV coinfected humans, $I_{CL}(0)$; TB infectious and acute HCV dually infected humans, $I_{aT}(0)$; and TB infectious and chronic HCV dually infected humans, $I_{CT}(0)$, are changed for each variable at a time while maintaining starting values of the other variables.

Figure 2 shows that over time, regardless of the starting value of humans at a risk of contracting TB and HCV infections, the number of humans that remain likely to contract TB and HCV infections is identical. Similarly, in Figures 3 and 4, initial values of each of the respective infected and coinfected variables are changed while maintaining values of other state variables. Over time, it is revealed that the number of humans left infected and coinfected is the same. This can be concluded that there is a globally stable endemic equilibrium for the TB-HCV coinfection model.

4. Sensitivity and Numerical Analysis

4.1. Sensitivity Analysis. With the aim of establishing how to decrease human death and morbidity rates due to TB infection, HCV infection, and their coinfection, it is imperative to be aware of the significance of the given parameters in the dynamics of the infection. This helps us to know the suitable intervention plan of action that can be taken to curb the infection. Here, we compute the sensitivity indices of the basic reproduction number, $R_0 = \max\{R_T, R_H\}$, with respect to the parameters in TB-HCV coinfection model (4). This is done using the normalized forward sensitivity index method (Chitnis et al. [29]).

Table 3: The TB-HCV coinfection model parameter values.

Parameter	Value	Source
μ	$0.02\,\text{yr}^{-1}$	[51]
σ	$0.0575\,\text{yr}^{-1}$	[51]
δ	$0.82\,\text{yr}^{-1}$	[59]
d_1	$0.6\,\text{yr}^{-1}$	Assumed
d_2	$0.8\,\text{yr}^{-1}$	Assumed
θ	$0.25\,\text{yr}^{-1}$	[60]
α	$2\,\text{yr}^{-1}$	[56]
τ_1	$0.00013\,\text{yr}^{-1}$	Assumed
τ_2	$0.00015\,\text{yr}^{-1}$	Assumed
β_1	$0.00014\,\text{yr}^{-1}$	Assumed
β_2	$0.00016\,\text{yr}^{-1}$	Assumed
η	$0.4405\,\text{yr}^{-1}$	[61]
π	$0.27\,\text{yr}^{-1}$	[56]
ϕ	$0.20\,\text{yr}^{-1}$	Assumed
ξ_1, ξ_2	4, 2 people yr^{-1}	[60], [59]
q_1, q_2	$0.08,\ 0.07\,\text{yr}^{-1}$	[60]
a_1, a_2	$1.002,\ 1.003\,\text{yr}^{-1}$	Assumed
b_1, b_2, b_3 and b_4	$1.001,\ 1.003,\ 1.002,\ 1.005\,\text{yr}^{-1}$	Assumed

Definition 16. The normalized forward sensitivity index of a variable, V, that depends differentiably on a parameter, p, is defined as a ratio of relative change in V to the relative change in parameter, p, that is,

$$i_p^V = \frac{\partial V}{\partial p} \times \frac{p}{V}. \tag{162}$$

Now, since $R_0 = \max\{R_T, R_H\}$, the sensitivity analysis of R_0 with respect to each of the parameters is analysed by way of the sensitivity indices of R_T and R_H. Thus, implicitly, the decisive parameters shall entirely be dependent on the predominant infection.

4.1.1. Sensitivity Indices of R_T and R_H. These are computed with parameter values from Table 3 using the formula

$$i_p^{R_T} = \frac{\partial R_T}{\partial p} \times \frac{p}{R_T} \text{ and } i_p^{R_H} = \frac{\partial R_H}{\partial p} \times \frac{p}{R_H}. \tag{163}$$

For example, the sensitivity index of R_T and R_H with respect to ξ_1 and ξ_2 are calculated as follows:

$$\begin{aligned} i_{\xi_1}^{R_T} &= \frac{\partial R_T}{\partial \xi_1} \times \frac{\xi_1}{R_T} = \frac{q_1 \theta}{(\mu + \sigma_1)(\theta + \eta + \mu)} \times \frac{\xi_1}{R_T} = 1, \\ i_{\xi_2}^{R_H} &= \frac{\partial R_H}{\partial \xi_2} \times \frac{\xi_2}{R_H} = \frac{q_2(\phi(\mu + \delta_2) + \alpha)}{(\pi + \alpha + \mu + \delta_1)(\mu + \delta_2)} \times \frac{\xi_2}{R_H} = 1. \end{aligned} \tag{164}$$

Other sensitivity indices for both R_T and R_H with respect to the particular parameter are calculated in a similar manner. Sensitivity indices for both R_T and R_H are presented in Table 4 where the parameters are arranged from the most sensitive to the least ones.

4.1.2. Sensitivity Indices and Their Interpretation. From Table 4, for a parameter with a positive index, it signifies that the corresponding basic reproduction number decreases (or

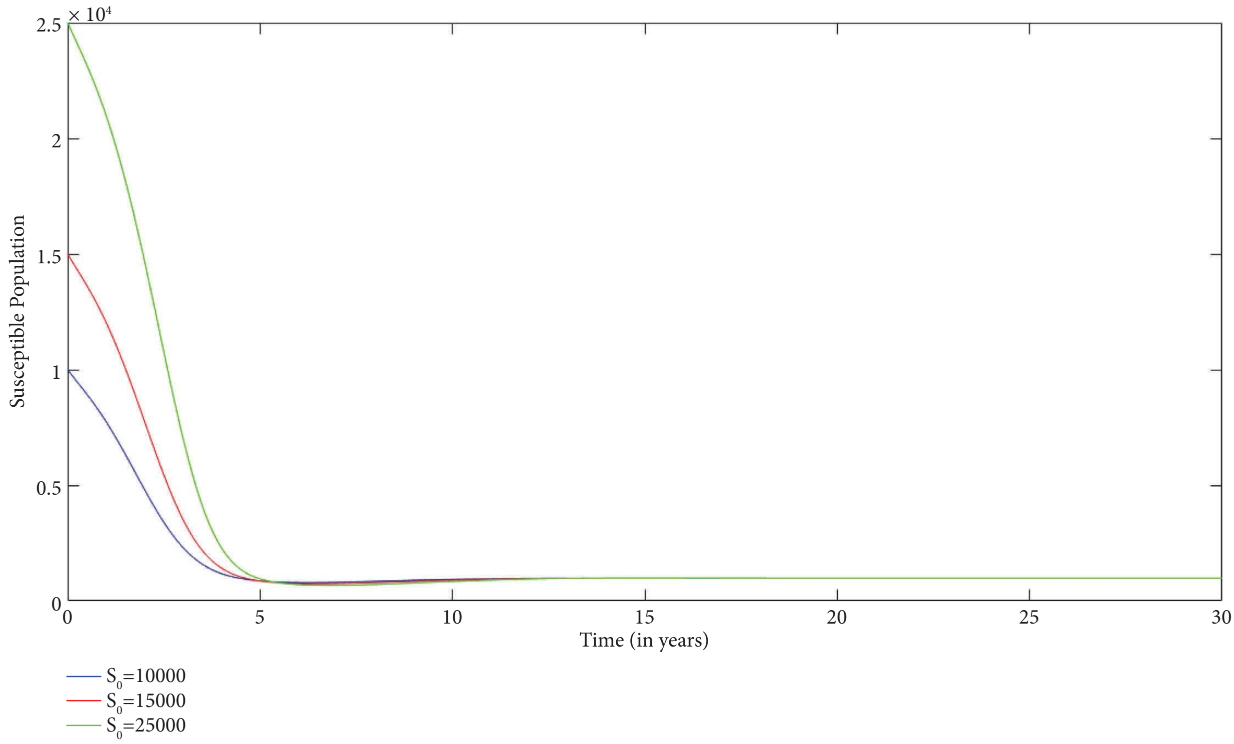

Figure 2: A graph of susceptible humans against time with only values of susceptible varied.

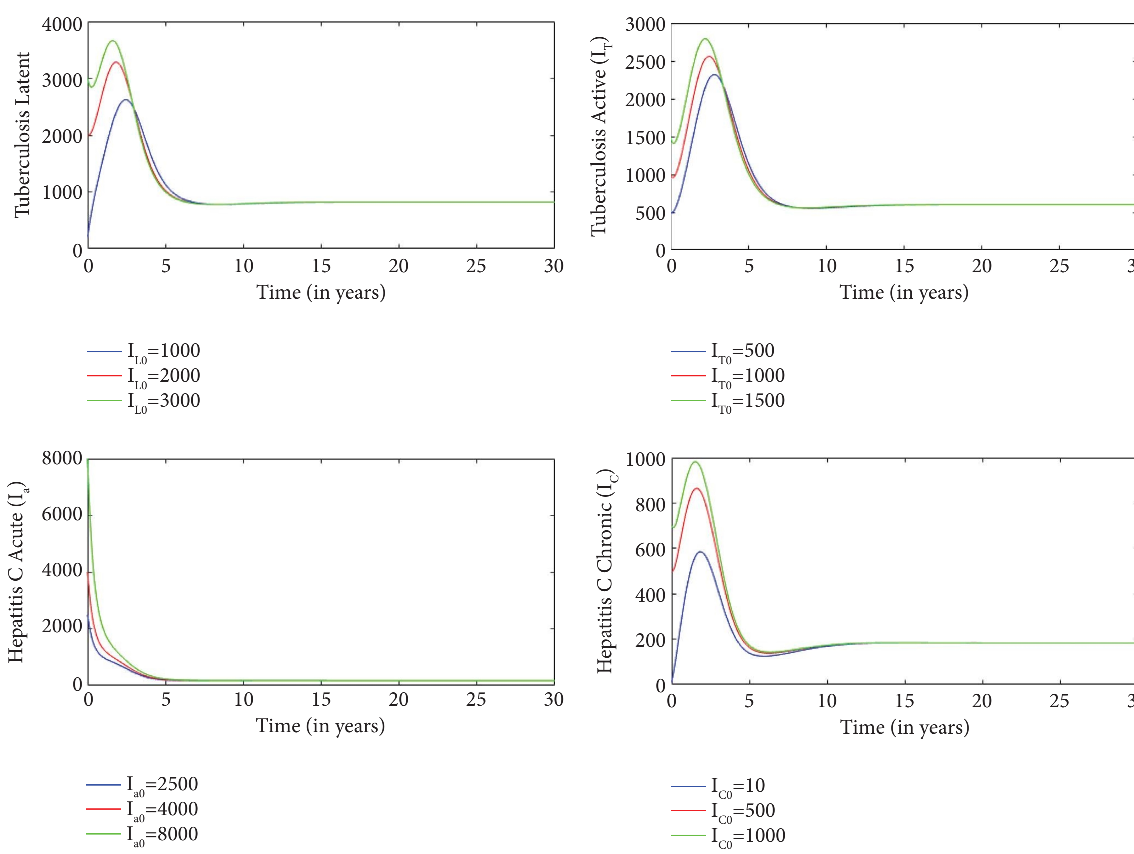

Figure 3: A graph of infected humans against time with only values of respective infectives varied.

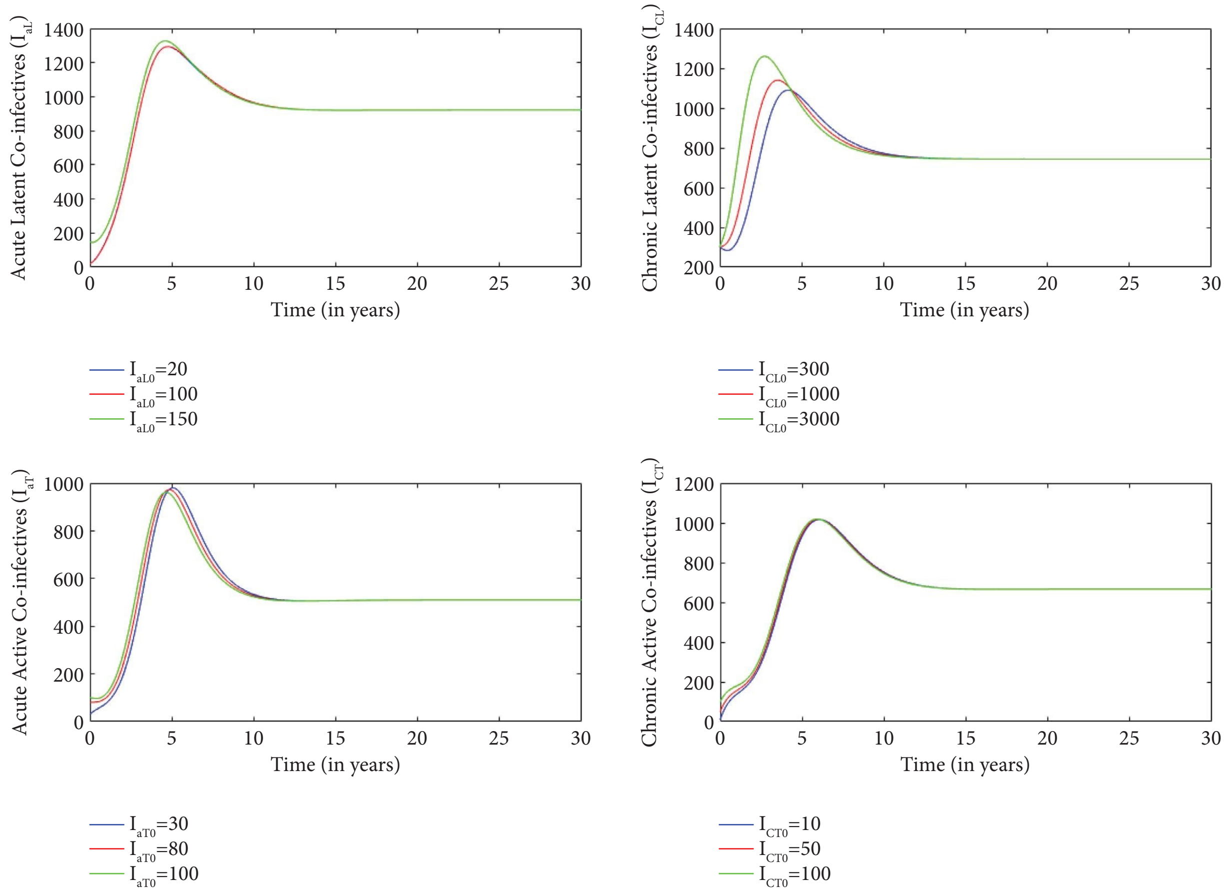

FIGURE 4: A graph of coinfected humans against time with only values of respective coinfectives varied.

TABLE 4: Numerical values for the sensitivity indices of R_T and R_H with respect to parameters.

Basic reproduction number	Parameter	Sensitivity index
	ξ_1	+1.0000
	q_1	+1.0000
R_T	σ	−0.7877
	η	−0.6239
	θ	+0.3219
	μ	−0.0949
	ξ_2	+1.0000
	q_2	+1.0000
	δ	−0.7568
R_H	α	+0.1640
	π	−0.0973
	ϕ	+0.0771
	μ	−0.0227

increases) with decrease (or increase) in that parameter, while keeping other parameters unchanged. For example, $i_{\xi_1}^{R_T} = 1$ means that decreasing (or increasing) the value of effective contact rate with TB-infected humans, ξ_1 by say, 10%, while keeping the other parameter values constant, decreases (or increases) the value of R_T by 10%. Similarly, $i_{\theta}^{R_T} = 0.3219$ means that increasing (or decreasing) of θ by 10% increases (or decreases) R_T by 3.219%.

On the other hand, the negative sign of the sensitivity index of say R_T with respect to $\sigma, \eta,$ and μ means an inverse relationship between the parameters and R_T. For instance, a 20% decrease (or increase) in the value of the natural recovery rate of TB latent humans, η, while maintaining the value of the other parameters increases (or decreases) the value of R_T by about 12.4%.

It is noted that the spread of TB infection rises when the values of ξ_1, q_1, and θ are increased and the ones of $\sigma, \eta,$ and μ are decreased. The most sensitive parameters in TB infection are the effective contact rate with TB-infected human ξ_1 and the likelihood of the contact being well efficient to give rise to a TB infection, q_1 followed by the disease induced death rate, σ, for humans infected with active TB. Therefore, the interventions need to target and aim at reducing the values of ξ_1, q_1, and the rate of progression, θ, from latent to infectious TB stage.

It is also noted that the endemicity of HCV infection increases when the values of the effective contact rate with HCV-infected human, ξ_2, the likelihood of the contact being well efficient to give rise to HCV infection, q_2, and the progression rate, α, from acute HCV to chronic stage are increased and those of $\delta, \pi,$ and μ are decreased.

The most sensitive parameters in HCV infection are ξ_2 and q_2, followed by α. Thus, interventions to reduce HCV

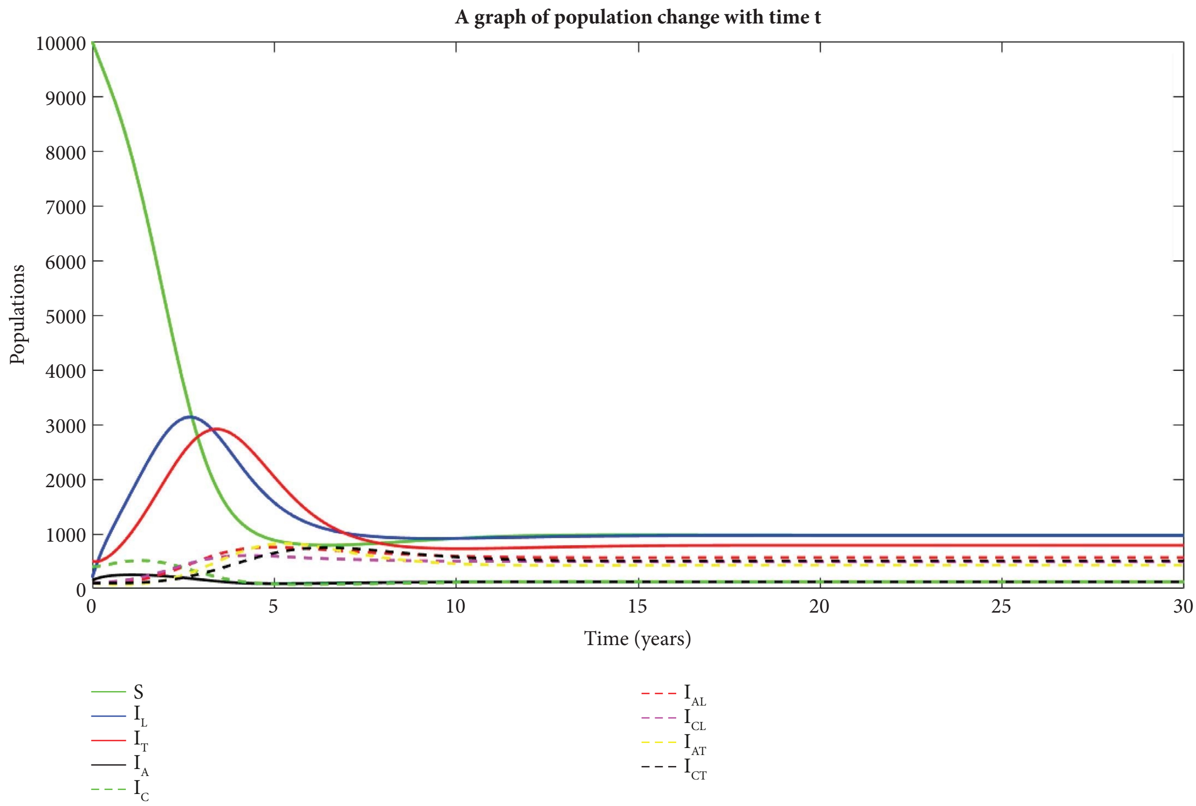

Figure 5: Simulation results showing susceptible, monoinfected, and coinfected humans against time.

infection should aim and concentrate on decreasing values of ξ_2, q_2, and α.

Therefore, strategies for TB and HCV control should target behaviours that reduce the contact rates for both diseases. Such strategies include screening and isolation, wearing of face masks for TB-infected humans and screening, sterilization of surgical instruments, and use of condoms for HCV cases.

Now, substituting for the parameter values in Table 3 into R_T and R_H, we have $R_T = 3.816$ and $R_H = 1.378$. We observe that the basic reproduction number R_0 of the TB-HCV coinfection model is concluded as

$$R_0 = \max\{R_T, R_H\} = \max\{3.816, 1.378\} = 3.816. \qquad (165)$$

Thus, the dynamics of TB-HCV coinfection is majorly influenced by TB.

4.2. Numerical Simulations. In the above sections, we have discussed the analytical behaviours of the TB-HCV coinfection model as well as the submodels. Here, we carry out numerical simulations to support the analytical solutions by studying the TB-HCV coinfection dynamics without intervention. We assume initial values of the state variables to be $S(0) = 10000, I_L(0) = 200, I_T(0) = 500$, $I_a(0) = 150$, $I_C(0) = 400, I_{aL}(0) = 100, I_{CL}(0) = 100$, $I_{aT}(0) = 100$ and $I_{CT}(0) = 100$ and parameter values as described in Table 3; the model was simulated using ODE 45 solver coded in MATLAB computer software. Both situations for $R_0 > 1$ and $R_0 < 1$ are considered with parameter modifications.

5. Discussion

It is noted from sensitivity analysis that the effective contact rate with TB- or HCV-infected humans together with the likelihood of a contact is well efficient to give rise to TB or HCV infection are equally likely to increase TB or HCV infection. Besides, their increase leads to increase in almost all other parameters.

Therefore, efforts such as screening and isolation, wearing face masks by TB-infected persons, and avoiding sharing surgical instruments during blood transfusion should be undertaken. Also, early treatment needs to be sought by the latent TB humans and acute HCV humans to avoid progression to infectious TB and chronic HCV stages, respectively. In Figure 5, we note that the local stability of the endemic equilibrium of the TB-HCV coinfection model does exist since the system goes to equilibria after about 10 years. This happens for $R_T = 3.816 > 1$ and $R_H = 1.378 > 1$.

In Figure 6, we realize that without treatment for both TB and HCV, the number of humans susceptible to TB and HCV decrease asymptotically to the low level in about 5 years. This is because more humans continue contracting TB and HCV with no intervention.

In Figure 7, the number of humans infected with latent TB, I_L, starts increasing and over time decline to a steady

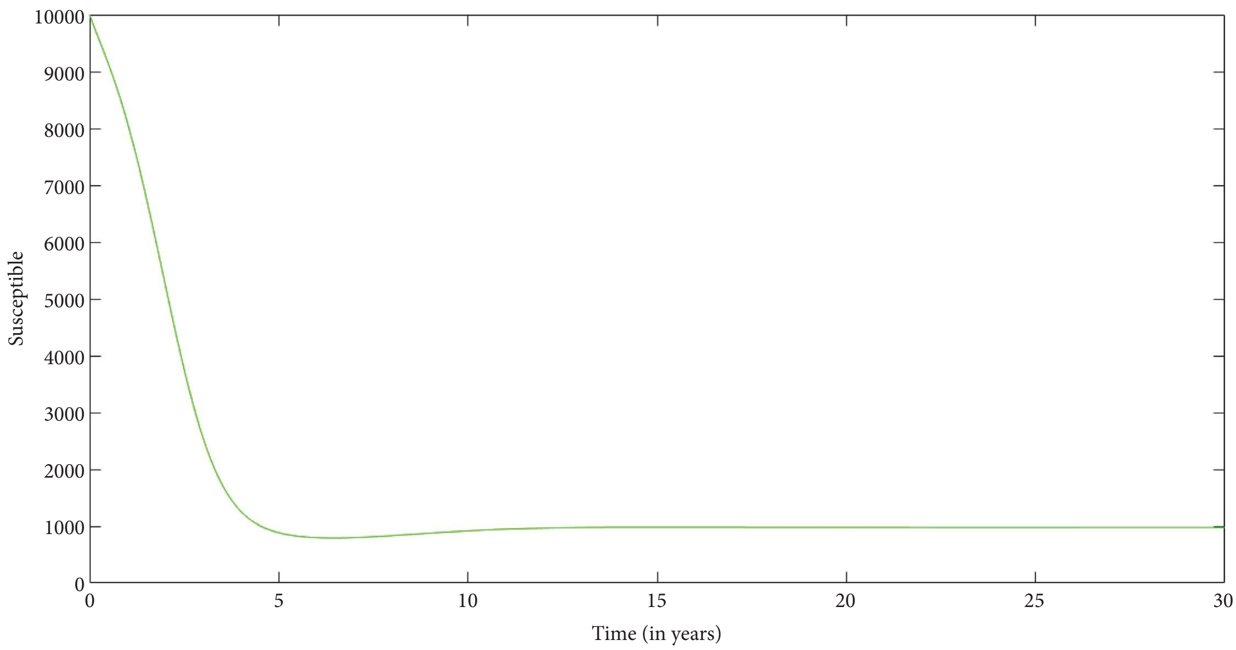

FIGURE 6: Simulation results showing susceptible humans against time.

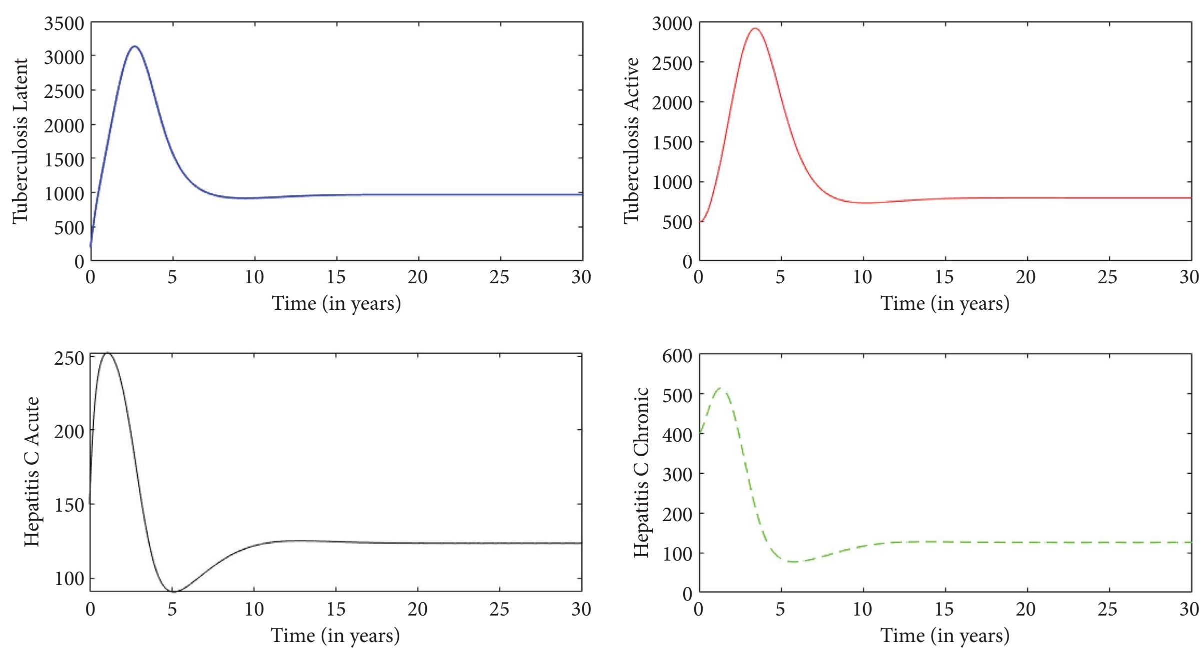

FIGURE 7: Simulation results showing monoinfected humans against time.

state at a low level. This is due to progression to infectious TB and natural recovery to susceptible class. The number of infectious TB humans, I_T, starts by increasing and in the process decline to a steady state at a low level. This is due to progression to dually infected classes.

The number of acute HCV humans, I_a, is seen to increase at the start and over time reduce to the steady state. Due to progression of acute HCV humans to chronic HCV class, the number of chronic HCV humans, I_C, increase at first and later on reduce to the steady state.

From Figure 8, the number of humans coinfected with latent TB and acute HCV begins to rise and later on reduce asymptotically to a slightly low steady level. Similarly, the number of infected humans from the dually infected classes I_{CL}, I_{aT}, and I_{CT} increases in the beginning and over time reduce asymptotically to slightly lower steady levels.

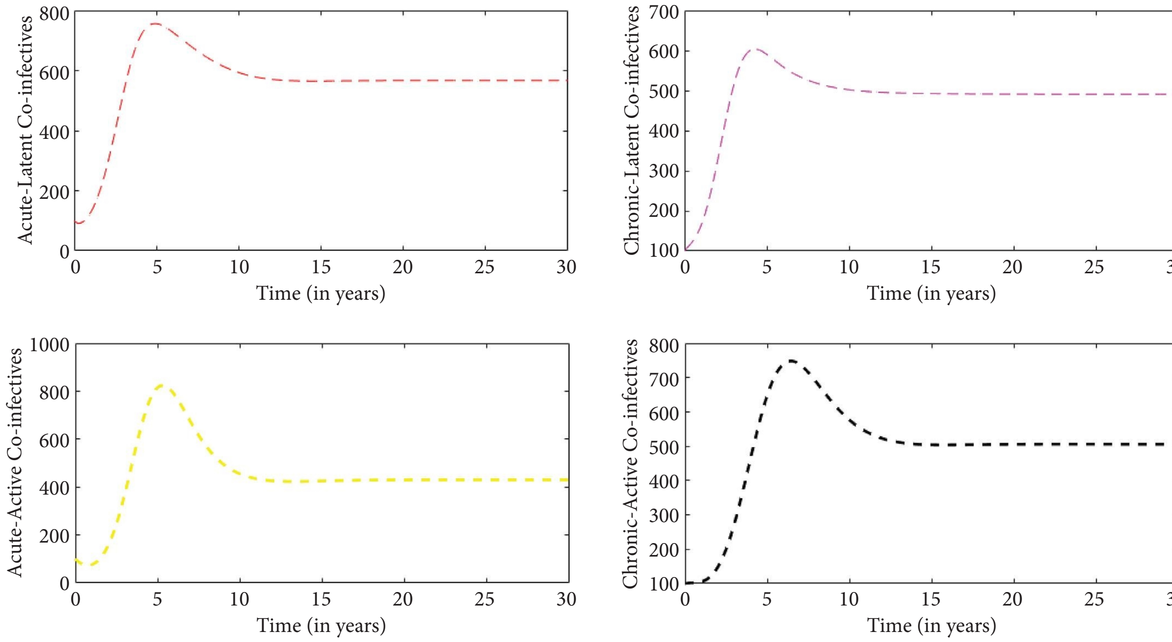

FIGURE 8: Simulation results showing coinfected humans against time.

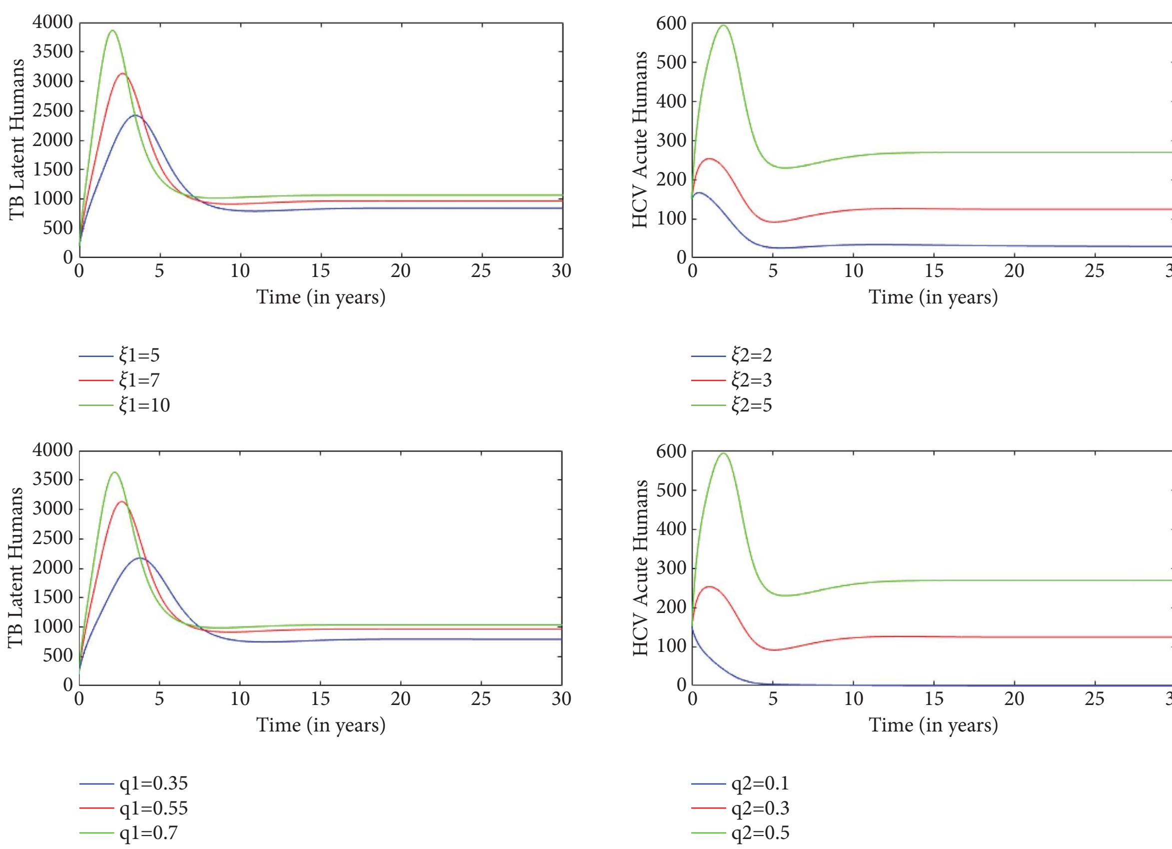

FIGURE 9: Simulation results showing TB latent humans and HCV acute humans against time with $\xi_1, \xi_2, q_1,$ and q_2, respectively, varied.

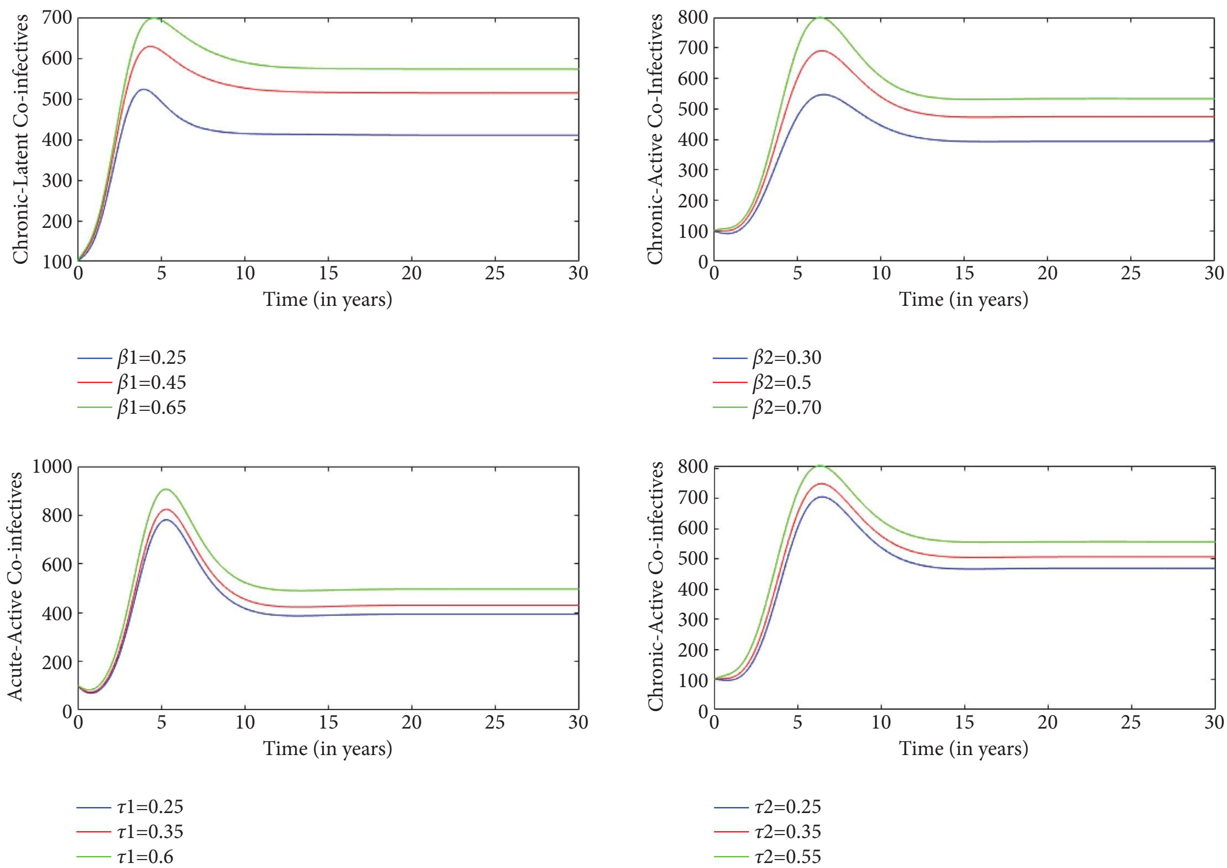

FIGURE 10: Graphs of humans coinfected with chronic HCV and latent TB, I_{CL}, chronic HCV and active TB, I_{CT}, acute HCV and active TB, I_{aT}, and chronic HCV and active TB, I_{CT}, against time when β_1, β_2, τ_1, and τ_2 are, respectively, varied.

From Figures 7 and 8, it is observed that

(1) Coinfected classes, I_{aL}, I_{CL}, I_{aT} and I_{CT}, over time decline asymptotically to attain stability to levels higher than those at which monoinfected classes I_L, I_T, I_a, and I_C attain stability.

(2) Coinfected classes over time take longer to attain steady state compared to monoinfected classes. Whereas monoinfected classes take about 5 years, the dually infected classes take about 10 years to attain stability.

Figure 9 explains the effect of effective contact rate with TB- or HCV-infected human on the number of TB latent or HCV acute humans. It shows that an increase in the effective contact rate with TB- or HCV-infected humans leads to the increase in the number of TB latent or HCV acute humans and vice versa. Relatedly, an increase in the likelihood of the contact being well efficient to cause a TB or HCV infection leads to increase in the rate of transmission of TB-HCV infection and vice versa.

Figure 10 shows the dynamics of humans coinfected with chronic HCV and latent TB, I_{CL}, chronic HCV and active TB, I_{CT}, acute HCV and active TB I_{aT}, and chronic HCV and active TB, I_{CT}, with changing values of β_1, β_2, τ_1, and τ_2, respectively.

Generally, the graphs indicate that an increase in the rates of progression, say β_1, β_2, τ_1 or τ_2 from one coinfected class leads to increase in the number of coinfected humans in another class. This leads to escalation of the disease among the humans. However, as time passes by, all the graphs begin to fly horizontally regardless of the dissimilar values of the individual parameters. Thus, there is dire need to introduce the dually infected humans to some interventions, say treatment.

6. Conclusion

In our study, a TB-HCV coinfection model with no intervention was developed and analysed. The positivity and boundedness properties of the model solutions in a biologically feasible region were verified. The steady states of the submodels and their stability with respect to the basic reproduction numbers were analysed.

In both submodels, the disease-free equilibrium points are found to be locally asymptotically stable provided their respective reproduction numbers are less than unity. The unique endemic equilibrium points, E_T for the TB submodel and E_H for the HCV submodel, exist whenever their corresponding reproduction numbers R_T and R_H are greater than unity.

The disease-free equilibrium, $E^0 = (X^0, 0)$, may not be globally asymptotically stable for $R_0 < 1$, indicating a backward bifurcation will occur at $R_0 = 1$ as proved in Feng et al.

[28]. However, the equilibrium points, E^0 and E^*, for the TB-HCV coinfection model are both locally asymptotically stable.

From numerical simulations, the number of TB latent humans and HCV acute humans and the number of dually infected humans have a linear relationship with the effective contact rate of TB or HCV infected humans. The number is also proportional to the likelihood of the contact being well efficient to give rise to TB or HCV infection and the progression rate from TB latent or HCV acute to TB active or HCV chronic stage. Therefore, it is necessary to mitigate and eradicate the infections.

From sensitivity analysis, decreasing the rate of contact between TB or HCV infected and susceptible humans is the major effective way to manage the spread of TB or HCV infection. Hence, strategies such as health education campaigns to communities targeting reducing the transmission rates of TB and HCV could help to reduce the progression of latent TB and acute HCV humans to infectious TB and chronic HCV humans, respectively. These could include screening and isolation, wearing of face masks for TB cases and screening, sterilization of surgical instruments, and use of condoms for HCV-infected humans.

The current model comes with limitations in accessing real data since there are no mathematical studies that deeply explore the coinfection burden of TB and HCV. Including intervention strategies against both TB and HCV infections in the current model could further improve our understanding of the control dynamics of both diseases.

Data Availability

The data used to support the findings of this study are included within the article.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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