

Sensitivity Analysis of HIV and Tuberculosis Co-Infection with Interventions

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Abstract: Human Immunodeficiency Virus (HIV) and Tuberculosis (TB) represent a synergistic syndemic where immunocompromising HIV infection significantly escalates the risk of active TB reactivation and progression. We developed and analysis a co-infection compartmental model which incorporates features such as treatment, vaccination, disease progression, and modification parameters to capture the interaction between single and coinfections. The basic reproduction numbers for the HIV and TB sub-models are derived using the next generation matrix method, and stability analysis shows that the disease-free equilibrium is stable when the reproduction number is less than unity. Sensitivity analysis identifies transmission rates and modification parameters as key drivers of disease spread, while treatment and control measures reduce infection levels. Numerical simulations confirm the analytical results and demonstrate that the interaction between HIV and TB significantly increases the overall disease burden. The findings highlight the importance of integrated intervention strategies for effective disease control.

Keywords: HIV, TB, Basic Reproduction, Intervention, Sensitivity

INTRODUCTION

Human immunodeficiency virus (HIV) and tuberculosis (TB) remain important public health concerns, especially in many low- and middle-income countries. Although they are caused by different pathogens, the two diseases are closely connected because HIV weakens the immune system and makes people more vulnerable to TB infection and the development of active disease. This interaction has contributed considerably to the burden of illness and death associated with the two infections. The World Health Organization (WHO) reported that an estimated 10.8 million people developed TB globally in 2023, including about 662,000 people living with HIV. TB also accounted for a substantial number of deaths among people with HIV during the same period (WHO, 2024). The continued occurrence of both infections, particularly in regions where they overlap, makes their combined transmission an important issue for public health research.

The relationship between HIV and TB is not simply a matter of two diseases occurring in the same population. HIV-related damage to the immune system can increase the likelihood that a person with TB infection will progress to active disease. TB infection, in turn, can further complicate the clinical management of individuals living with HIV. This close relationship means that changes in the transmission or progression of one disease may affect the dynamics of the other. Mathematical studies have shown that the interaction between HIV and TB can produce transmission patterns that may not be adequately represented when the diseases are considered separately (Bhunu et al., 2009; Sharomi et al., 2008). Understanding these interactions is therefore important when evaluating strategies aimed at reducing the combined burden of HIV and TB.

The situation becomes even more challenging with the emergence of drug-resistant TB. Drug-resistant TB develops when the bacteria responsible for TB are no longer effectively controlled by one or more of the medicines normally used for treatment. Multidrug-resistant or rifampicin-resistant TB (MDR/RR-TB) is of particular concern because it requires different treatment approaches and can be more difficult to manage. In 2023, an estimated 400,000 people developed MDR/RR-TB globally, according to the WHO (2024). Inadequate treatment, treatment interruption, and transmission of resistant strains can all contribute to the persistence of drug-resistant TB. When drug-resistant TB occurs alongside HIV and drug-susceptible TB, the resulting transmission process becomes more complicated because individuals may experience different pathways of infection, disease progression, treatment, and recovery. These circumstances highlight the need for approaches that can account for the interactions among the different disease states.

Mathematical modelling offers a useful way of studying such complex disease interactions. By separating a population into different epidemiological groups and describing how individuals move from one group to another, a mathematical model can provide insight into how infections are introduced, transmitted, treated, and controlled within a population. Previous studies have used mathematical models to investigate HIV–TB coinfection and to examine the effects of treatment and other control measures. For instance, Bhunu et al. (2009) developed a model that considered the interaction between HIV/AIDS and TB, including the effects of treatment, while Sharomi et al. (2008) examined HIV and TB coinfection in the presence of treatment. These studies demonstrate how mathematical modelling can be used to investigate important epidemiological quantities such as the basic reproduction number, equilibrium states, and the stability of disease-free conditions. Such information can be useful in understanding which factors contribute most strongly to disease persistence and how control measures may influence transmission.

Building on these considerations, this study develops a deterministic compartmental model for the transmission of HIV, TB, and drug-resistant TB within a population. The model takes into account important processes such as vaccination, treatment, disease progression, and modification of transmission or disease-related parameters, while also allowing for interactions between individuals with single infections and those experiencing coinfection. The basic reproduction numbers associated with the HIV and TB components are obtained using the next-generation matrix approach, and the stability of the disease-free equilibrium is investigated. Sensitivity analysis is then used to examine the influence of selected model parameters on disease transmission, while numerical simulations are carried out to

illustrate the behaviour of the system and the effects of the incorporated control measures. The study is intended to improve understanding of the interaction among HIV, TB, and drug-resistant TB and to provide mathematical evidence that can support the consideration of integrated approaches to controlling these infections.

Sensitivity Analysis of the HIV Submodel

In this subsection, sensitivity analysis of the HIV submodel is carried out using the normalized forward sensitivity index in order to determine the relative influence of each parameter on the basic reproduction number of the HIV submodel. This analysis is important because it shows the extent to which a change in each parameter affects the transmission potential of HIV in the population.

The normalized forward sensitivity index of the basic reproduction number R_{0H} with respect to a parameter p is defined by

$$\Upsilon_p^{R_{0H}} = \frac{\partial R_{0H}}{\partial p} \cdot \frac{p}{R_{0H}}.$$

A positive sensitivity index implies that an increase in the parameter increases the basic reproduction number, while a negative sensitivity index implies that an increase in the parameter decreases the basic reproduction number.

Recall that the basic reproduction number of the HIV submodel is given by

$$R_{0H} = \frac{\beta_H}{\alpha + \mu + \tau_H} \left(1 + \frac{\theta\alpha}{\mu + \delta_H + \tau_H} \right).$$

We now determine the sensitivity indices with respect to the key model parameters.

Sensitivity index with respect to β_H

Differentiating R_{0H} with respect to β_H gives

$$\frac{\partial R_{0H}}{\partial \beta_H} = \frac{1}{\alpha + \mu + \tau_H} \left(1 + \frac{\theta\alpha}{\mu + \delta_H + \tau_H} \right).$$

Therefore,

$$\Upsilon_{\beta_H}^{R_{0H}} = \frac{\partial R_{0H}}{\partial \beta_H} \cdot \frac{\beta_H}{R_{0H}} = \frac{1}{\alpha + \mu + \tau_H} \left(1 + \frac{\theta\alpha}{\mu + \delta_H + \tau_H} \right) \cdot \frac{\beta_H}{\frac{\beta_H}{\alpha + \mu + \tau_H} \left(1 + \frac{\theta\alpha}{\mu + \delta_H + \tau_H} \right)}.$$

Hence,

$$\boxed{\Upsilon_{\beta_H}^{R_{0H}} = 1.}$$

This shows that the basic reproduction number is directly proportional to the HIV transmission rate.

Sensitivity index with respect to θ

Differentiating R_{0H} with respect to θ gives

$$\frac{\partial R_{0H}}{\partial \theta} = \frac{\beta_H}{\alpha + \mu + \tau_H} \cdot \frac{\alpha}{\mu + \delta_H + \tau_H}.$$

Thus,

$$\Upsilon_{\theta}^{R_{0H}} = \frac{\partial R_{0H}}{\partial \theta} \cdot \frac{\theta}{R_{0H}} = \left[\frac{\beta_H}{\alpha + \mu + \tau_H} \cdot \frac{\alpha}{\mu + \delta_H + \tau_H} \right] \cdot \frac{\theta}{\frac{\beta_H}{\alpha + \mu + \tau_H} \left(1 + \frac{\theta \alpha}{\mu + \delta_H + \tau_H} \right)}.$$

Simplifying gives

$$\Upsilon_{\theta}^{R_{0H}} = \frac{\theta \alpha}{\mu + \delta_H + \tau_H} \cdot \frac{1}{1 + \frac{\theta \alpha}{\mu + \delta_H + \tau_H}}.$$

Hence,

$$\boxed{\Upsilon_{\theta}^{R_{0H}} = \frac{\theta \alpha}{\mu + \delta_H + \tau_H + \theta \alpha}.$$

Since all parameters are positive, it follows that

$$\Upsilon_{\theta}^{R_{0H}} > 0.$$

Therefore, an increase in the infectiousness of individuals in the AIDS class increases the reproduction number.

Sensitivity index with respect to α

Differentiating

$$R_{0H} = \frac{\beta_H}{\alpha + \mu + \tau_H} \left(1 + \frac{\theta \alpha}{\mu + \delta_H + \tau_H} \right)$$

with respect to α gives

$$\frac{\partial R_{0H}}{\partial \alpha} = -\frac{\beta_H}{(\alpha + \mu + \tau_H)^2} \left(1 + \frac{\theta \alpha}{\mu + \delta_H + \tau_H} \right) + \frac{\beta_H}{\alpha + \mu + \tau_H} \cdot \frac{\theta}{\mu + \delta_H + \tau_H}.$$

Thus,

$$\Upsilon_{\alpha}^{R_{0H}} = \frac{\partial R_{0H}}{\partial \alpha} \cdot \frac{\alpha}{R_{0H}}$$

$$\Upsilon_{\alpha}^{R_{0H}} = -\frac{\alpha}{\alpha + \mu + \tau_H} + \frac{\theta\alpha}{\mu + \delta_H + \tau_H} \cdot \frac{1}{1 + \frac{\theta\alpha}{\mu + \delta_H + \tau_H}}.$$

Hence,

$$\boxed{\Upsilon_{\alpha}^{R_{0H}} = -\frac{\alpha}{\alpha + \mu + \tau_H} + \frac{\theta\alpha}{\mu + \delta_H + \tau_H + \theta\alpha}.$$

This shows that the effect of the progression rate α depends on the balance between progression to AIDS and the removal processes acting on infected individuals.

Sensitivity index with respect to τ_H

Since τ_H appears in both denominators of R_{0H} , differentiating with respect to τ_H gives

$$\frac{\partial R_{0H}}{\partial \tau_H} = -\frac{\beta_H}{(\alpha + \mu + \tau_H)^2} \left(1 + \frac{\theta\alpha}{\mu + \delta_H + \tau_H}\right) - \frac{\beta_H}{\alpha + \mu + \tau_H} \cdot \frac{\theta\alpha}{(\mu + \delta_H + \tau_H)^2}.$$

Therefore,

$$\Upsilon_{\tau_H}^{R_{0H}} = \frac{\partial R_{0H}}{\partial \tau_H} \cdot \frac{\tau_H}{R_{0H}}$$

Hence,

$$\boxed{\Upsilon_{\tau_H}^{R_{0H}} = -\frac{\tau_H}{\alpha + \mu + \tau_H} - \frac{\tau_H\theta\alpha}{(\mu + \delta_H + \tau_H)(\mu + \delta_H + \tau_H + \theta\alpha)}.$$

Since all parameters are positive, it follows that

$$\Upsilon_{\tau_H}^{R_{0H}} < 0.$$

Thus, increasing the HIV treatment rate reduces the basic reproduction number.

Sensitivity index with respect to δ_H

Differentiating R_{0H} with respect to δ_H gives

$$\frac{\partial R_{0H}}{\partial \delta_H} = -\frac{\beta_H}{\alpha + \mu + \tau_H} \cdot \frac{\theta\alpha}{(\mu + \delta_H + \tau_H)^2}.$$

Thus,

$$\Upsilon_{\delta_H}^{R_{0H}} = \frac{\partial R_{0H}}{\partial \delta_H} \cdot \frac{\delta_H}{R_{0H}} = \left[-\frac{\beta_H}{\alpha + \mu + \tau_H} \cdot \frac{\theta\alpha}{(\mu + \delta_H + \tau_H)^2} \right] \cdot \frac{\delta_H}{\frac{\beta_H}{\alpha + \mu + \tau_H} \left(1 + \frac{\theta\alpha}{\mu + \delta_H + \tau_H} \right)}.$$

Simplifying gives

$$\Upsilon_{\delta_H}^{R_{0H}} = -\frac{\delta_H \theta \alpha}{(\mu + \delta_H + \tau_H)^2} \cdot \frac{1}{1 + \frac{\theta\alpha}{\mu + \delta_H + \tau_H}}.$$

Hence,

$$\boxed{\Upsilon_{\delta_H}^{R_{0H}} = -\frac{\delta_H \theta \alpha}{(\mu + \delta_H + \tau_H)(\mu + \delta_H + \tau_H + \theta\alpha)}}.$$

Therefore,

$$\Upsilon_{\delta_H}^{R_{0H}} < 0.$$

This means that an increase in AIDS-induced mortality reduces the reproduction number.

Sensitivity index with respect to μ

Since μ appears in both denominators of R_{0H} , differentiating with respect to μ gives

$$\frac{\partial R_{0H}}{\partial \mu} = -\frac{\beta_H}{(\alpha + \mu + \tau_H)^2} \left(1 + \frac{\theta\alpha}{\mu + \delta_H + \tau_H} \right) - \frac{\beta_H}{\alpha + \mu + \tau_H} \cdot \frac{\theta\alpha}{(\mu + \delta_H + \tau_H)^2}.$$

Thus,

$$\Upsilon_{\mu}^{R_{0H}} = \frac{\partial R_{0H}}{\partial \mu} \cdot \frac{\mu}{R_{0H}}$$

$$\boxed{\Upsilon_{\mu}^{R_{0H}} = -\frac{\mu}{\alpha + \mu + \tau_H} - \frac{\mu\theta\alpha}{(\mu + \delta_H + \tau_H)(\mu + \delta_H + \tau_H + \theta\alpha)}}.$$

Therefore,

$$\Upsilon_{\mu}^{R_{0H}} < 0.$$

This indicates that an increase in the natural death rate reduces the basic reproduction number.

These results show that the HIV transmission rate β_H has the strongest positive effect on the spread of HIV, while the treatment rate τ_H , the disease-induced death rate δ_H , and the natural death rate μ reduce

the basic reproduction number. This confirms that reducing HIV transmission and increasing treatment coverage are among the most effective strategies for controlling the disease.

Sensitivity Analysis of the TB Submodel

In this subsection, sensitivity analysis of the TB submodel is carried out using the normalized forward sensitivity index in order to determine the relative influence of each parameter on the basic reproduction number of the TB submodel. This analysis is important because it identifies the parameters that contribute most strongly to the spread of tuberculosis and those that help to reduce transmission.

The normalized forward sensitivity index of the basic reproduction number R_{0T} with respect to a parameter p is defined by

$$\Upsilon_p^{R_{0T}} = \frac{\partial R_{0T}}{\partial p} \cdot \frac{p}{R_{0T}}.$$

A positive sensitivity index implies that an increase in the parameter increases the basic reproduction number, while a negative sensitivity index implies that an increase in the parameter decreases the basic reproduction number.

Recall that the basic reproduction number of the TB submodel is

$$R_{0T} = \frac{\beta_T \mu}{(\omega + \mu)(\tau_T + \rho + \mu + \delta_T)} \left(1 + \frac{\eta \rho}{\tau_{DR} + \mu + \delta_{DR}} \right).$$

We now determine the sensitivity indices with respect to the key model parameters.

Sensitivity index with respect to β_T

Differentiating R_{0T} with respect to β_T gives

$$\frac{\partial R_{0T}}{\partial \beta_T} = \frac{\mu}{(\omega + \mu)(\tau_T + \rho + \mu + \delta_T)} \left(1 + \frac{\eta \rho}{\tau_{DR} + \mu + \delta_{DR}} \right).$$

Therefore,

$$\Upsilon_{\beta_T}^{R_{0T}} = \frac{\partial R_{0T}}{\partial \beta_T} \cdot \frac{\beta_T}{R_{0T}}$$

Hence,

$$\boxed{\Upsilon_{\beta_T}^{R_{0T}} = 1.}$$

This shows that the basic reproduction number is directly proportional to the effective TB transmission rate.

Sensitivity index with respect to η

Differentiating R_{0T} with respect to η gives

$$\frac{\partial R_{0T}}{\partial \eta} = \frac{\beta_T \mu}{(\omega + \mu)(\tau_T + \rho + \mu + \delta_T)} \cdot \frac{\rho}{\tau_{DR} + \mu + \delta_{DR}}.$$

$$\Upsilon_{\eta}^{R_{0T}} = \frac{\eta \rho}{\tau_{DR} + \mu + \delta_{DR}} \cdot \frac{1}{1 + \frac{\eta \rho}{\tau_{DR} + \mu + \delta_{DR}}}.$$

Hence,

$$\Upsilon_{\eta}^{R_{0T}} = \frac{\eta \rho}{\tau_{DR} + \mu + \delta_{DR} + \eta \rho}.$$

Since all parameters are positive, it follows that

$$\Upsilon_{\eta}^{R_{0T}} > 0.$$

Therefore, an increase in the modification parameter associated with the infectiousness of drug-resistant TB increases the basic reproduction number.

Sensitivity index with respect to ρ

Differentiating

$$R_{0T} = \frac{\beta_T \mu}{(\omega + \mu)(\tau_T + \rho + \mu + \delta_T)} \left(1 + \frac{\eta \rho}{\tau_{DR} + \mu + \delta_{DR}} \right)$$

with respect to ρ gives

$$\frac{\partial R_{0T}}{\partial \rho} = - \frac{\beta_T \mu}{(\omega + \mu)(\tau_T + \rho + \mu + \delta_T)^2} \left(1 + \frac{\eta \rho}{\tau_{DR} + \mu + \delta_{DR}} \right) + \frac{\beta_T \mu}{(\omega + \mu)(\tau_T + \rho + \mu + \delta_T)} \cdot \frac{\eta}{\tau_{DR} + \mu + \delta_{DR}}.$$

Thus,

$$\Upsilon_{\rho}^{R_{0T}} = \frac{\partial R_{0T}}{\partial \rho} \cdot \frac{\rho}{R_{0T}}$$

Hence,

$$\Upsilon_{\rho}^{R_{0T}} = - \frac{\rho}{\tau_T + \rho + \mu + \delta_T} + \frac{\eta \rho}{\tau_{DR} + \mu + \delta_{DR} + \eta \rho}.$$

This shows that the effect of the progression rate from drug-sensitive TB to drug-resistant TB depends on the balance between removal from the infectious TB class and the added contribution of resistant transmission.

Sensitivity index with respect to τ_T

Differentiating R_{0T} with respect to τ_T gives

$$\frac{\partial R_{0T}}{\partial \tau_T} = -\frac{\beta_T \mu}{(\omega + \mu)(\tau_T + \rho + \mu + \delta_T)^2} \left(1 + \frac{\eta \rho}{\tau_{DR} + \mu + \delta_{DR}}\right)$$

$$\boxed{\Upsilon_{\tau_T}^{R_{0T}} = -\frac{\tau_T}{\tau_T + \rho + \mu + \delta_T}.$$

Since all parameters are positive, it follows that

$$\Upsilon_{\tau_T}^{R_{0T}} < 0.$$

Thus, increasing the treatment rate of drug-sensitive TB reduces the basic reproduction number.

Sensitivity index with respect to τ_{DR}

Differentiating R_{0T} with respect to τ_{DR} gives

$$\frac{\partial R_{0T}}{\partial \tau_{DR}} = -\frac{\beta_T \mu}{(\omega + \mu)(\tau_T + \rho + \mu + \delta_T)} \cdot \frac{\eta \rho}{(\tau_{DR} + \mu + \delta_{DR})^2}.$$

Thus,

$$\boxed{\Upsilon_{\tau_{DR}}^{R_{0T}} = -\frac{\tau_{DR} \eta \rho}{(\tau_{DR} + \mu + \delta_{DR})(\tau_{DR} + \mu + \delta_{DR} + \eta \rho)}.$$

Therefore,

$$\Upsilon_{\tau_{DR}}^{R_{0T}} < 0.$$

This means that increasing the treatment rate of drug-resistant TB reduces the basic reproduction number.

Sensitivity index with respect to ω

Differentiating R_{0T} with respect to ω gives

$$\frac{\partial R_{0T}}{\partial \omega} = -\frac{\beta_T \mu}{(\omega + \mu)^2(\tau_T + \rho + \mu + \delta_T)} \left(1 + \frac{\eta \rho}{\tau_{DR} + \mu + \delta_{DR}}\right).$$

Hence,

$$\Upsilon_{\omega}^{R_{0T}} = -\frac{\omega}{\omega + \mu}.$$

Since all parameters are positive, we have

$$\Upsilon_{\omega}^{R_{0T}} < 0.$$

Thus, increasing the vaccination rate reduces the basic reproduction number.

Sensitivity index with respect to δ_T

Differentiating R_{0T} with respect to δ_T gives

$$\frac{\partial R_{0T}}{\partial \delta_T} = -\frac{\beta_T \mu}{(\omega + \mu)(\tau_T + \rho + \mu + \delta_T)^2} \left(1 + \frac{\eta \rho}{\tau_{DR} + \mu + \delta_{DR}} \right).$$

Thus,

Hence,

$$\Upsilon_{\delta_T}^{R_{0T}} = -\frac{\delta_T}{\tau_T + \rho + \mu + \delta_T}. \quad \Upsilon_{\delta_T}^{R_{0T}} < 0.$$

This implies that increasing TB-induced mortality reduces the basic reproduction number.

Sensitivity index with respect to δ_{DR}

Differentiating R_{0T} with respect to δ_{DR} gives

$$\frac{\partial R_{0T}}{\partial \delta_{DR}} = -\frac{\beta_T \mu}{(\omega + \mu)(\tau_T + \rho + \mu + \delta_T)} \cdot \frac{\eta \rho}{(\tau_{DR} + \mu + \delta_{DR})^2}.$$

Thus,

$$\Upsilon_{\delta_{DR}}^{R_{0T}} = -\frac{\delta_{DR} \eta \rho}{(\tau_{DR} + \mu + \delta_{DR})(\tau_{DR} + \mu + \delta_{DR} + \eta \rho)}.$$

Therefore,

$$\Upsilon_{\delta_{DR}}^{R_{0T}} < 0.$$

This means that increasing the death rate associated with drug-resistant TB reduces the basic reproduction number.

Sensitivity index with respect to μ

Since μ appears in all three denominator terms and also in the numerator of R_{0T} , differentiating with respect to μ give

$$\Upsilon_{\mu}^{R_{0T}} = 1 - \frac{\mu}{\omega + \mu} - \frac{\mu}{\tau_T + \rho + \mu + \delta_T} - \frac{\mu\eta\rho}{(\tau_{DR} + \mu + \delta_{DR})(\tau_{DR} + \mu + \delta_{DR} + \eta\rho)}.$$

This shows that the effect of the natural death rate on the basic reproduction number is determined by the balance between its direct contribution in the numerator and its removal effects in the denominator.

These results show that the transmission rate β_T and the modification parameter η increase the spread of tuberculosis, while the treatment rates τ_T and τ_{DR} , the vaccination rate ω , and the disease-induced death rates δ_T and δ_{DR} reduce the basic reproduction number. This confirms that reducing transmission, strengthening treatment, and increasing vaccination remain essential strategies for controlling TB transmission in the population.

Sensitivity Analysis of the Coinfection Model

In this subsection, sensitivity analysis of the full HIV–TB–DRTB coinfection model is carried out using the normalized forward sensitivity index. The purpose of this analysis is to determine the relative influence of the model parameters on the overall basic reproduction number of the full system. This is important because it helps to identify the epidemiological factors that most strongly drive the persistence of the coinfection dynamics and those that can be targeted for effective disease control.

The normalized forward sensitivity index of the basic reproduction number R_0 with respect to a parameter p is defined by

$$\Upsilon_p^{R_0} = \frac{\partial R_0}{\partial p} \cdot \frac{p}{R_0}.$$

A positive sensitivity index indicates that an increase in the parameter increases the reproduction number, while a negative sensitivity index indicates that an increase in the parameter decreases the reproduction number.

Recall that for the full coinfection model, the overall basic reproduction number is given by

$$R_0 = \max\{R_{0H}, R_{0T}\},$$

where

$$R_{0H} = \frac{\beta_H}{\alpha + \mu + \tau_H} \left(1 + \frac{\theta\alpha}{\mu + \delta_H + \tau_H} \right)$$

is the reproduction number associated with the HIV transmission pathway, and

$$R_{0T} = \frac{\beta_T \mu}{(\omega + \mu)(\tau_T + \rho + \mu + \delta_T)} \left(1 + \frac{\eta \rho}{\tau_{DR} + \mu + \delta_{DR}} \right)$$

is the reproduction number associated with the TB transmission pathway.

Since the overall reproduction number is defined as the maximum of R_{0H} and R_{0T} , the sensitivity analysis is naturally carried out in two cases.

Case 1: If $R_{0H} > R_{0T}$, then

$$R_0 = R_{0H}.$$

In this case, the sensitivity indices of the full coinfection model coincide with those of the HIV submodel. Therefore,

$$\Upsilon_{\beta_H}^{R_0} = \frac{\partial R_0}{\partial \beta_H} \cdot \frac{\beta_H}{R_0} = \frac{\partial R_{0H}}{\partial \beta_H} \cdot \frac{\beta_H}{R_{0H}} = 1,$$

so that

$$\boxed{\Upsilon_{\beta_H}^{R_0} = 1.}$$

Similarly,

$$\Upsilon_{\theta}^{R_0} = \frac{\partial R_{0H}}{\partial \theta} \cdot \frac{\theta}{R_{0H}} = \frac{\theta \alpha}{\mu + \delta_H + \tau_H + \theta \alpha},$$

and hence

$$\boxed{\Upsilon_{\theta}^{R_0} = \frac{\theta \alpha}{\mu + \delta_H + \tau_H + \theta \alpha}.$$

Also,

$$\Upsilon_{\alpha}^{R_0} = \frac{\partial R_{0H}}{\partial \alpha} \cdot \frac{\alpha}{R_{0H}} = -\frac{\alpha}{\alpha + \mu + \tau_H} + \frac{\theta \alpha}{\mu + \delta_H + \tau_H + \theta \alpha},$$

so that

$$\boxed{\Upsilon_{\alpha}^{R_0} = -\frac{\alpha}{\alpha + \mu + \tau_H} + \frac{\theta \alpha}{\mu + \delta_H + \tau_H + \theta \alpha}.$$

Furthermore,

$$\gamma_{\tau_H}^{R_0} = \frac{\partial R_{0H}}{\partial \tau_H} \cdot \frac{\tau_H}{R_{0H}} = -\frac{\tau_H}{\alpha + \mu + \tau_H} - \frac{\tau_H \theta \alpha}{(\mu + \delta_H + \tau_H)(\mu + \delta_H + \tau_H + \theta \alpha)},$$

hence

$$\gamma_{\tau_H}^{R_0} = -\frac{\tau_H}{\alpha + \mu + \tau_H} - \frac{\tau_H \theta \alpha}{(\mu + \delta_H + \tau_H)(\mu + \delta_H + \tau_H + \theta \alpha)}.$$

Also,

$$\gamma_{\delta_H}^{R_0} = \frac{\partial R_{0H}}{\partial \delta_H} \cdot \frac{\delta_H}{R_{0H}} = -\frac{\delta_H \theta \alpha}{(\mu + \delta_H + \tau_H)(\mu + \delta_H + \tau_H + \theta \alpha)},$$

so that

$$\gamma_{\delta_H}^{R_0} = -\frac{\delta_H \theta \alpha}{(\mu + \delta_H + \tau_H)(\mu + \delta_H + \tau_H + \theta \alpha)}.$$

Finally,

$$\gamma_{\mu}^{R_0} = \frac{\partial R_{0H}}{\partial \mu} \cdot \frac{\mu}{R_{0H}} = -\frac{\mu}{\alpha + \mu + \tau_H} - \frac{\mu \theta \alpha}{(\mu + \delta_H + \tau_H)(\mu + \delta_H + \tau_H + \theta \alpha)},$$

hence

$$\gamma_{\mu}^{R_0} = -\frac{\mu}{\alpha + \mu + \tau_H} - \frac{\mu \theta \alpha}{(\mu + \delta_H + \tau_H)(\mu + \delta_H + \tau_H + \theta \alpha)}.$$

In this situation, the parameters associated with the TB pathway do not determine the dominant threshold value of the full model. Therefore, their local contribution to the sensitivity of R_0 is negligible as long as R_{0H} remains strictly greater than R_{0T} .

Case 2: If $R_{0T} > R_{0H}$, then

$$R_0 = R_{0T}.$$

In this case, the sensitivity indices of the full coinfection model coincide with those of the TB submodel. Hence,

$$\gamma_{\beta_T}^{R_0} = \frac{\partial R_{0T}}{\partial \beta_T} \cdot \frac{\beta_T}{R_{0T}} = 1,$$

so that

$$\boxed{Y_{\beta_T}^{R_0} = 1.}$$

Also,

$$Y_{\eta}^{R_0} = \frac{\partial R_{0T}}{\partial \eta} \cdot \frac{\eta}{R_{0T}} = \frac{\eta \rho}{\tau_{DR} + \mu + \delta_{DR} + \eta \rho},$$

hence

$$\boxed{Y_{\eta}^{R_0} = \frac{\eta \rho}{\tau_{DR} + \mu + \delta_{DR} + \eta \rho}.}$$

Similarly,

$$Y_{\rho}^{R_0} = \frac{\partial R_{0T}}{\partial \rho} \cdot \frac{\rho}{R_{0T}} = -\frac{\rho}{\tau_T + \rho + \mu + \delta_T} + \frac{\eta \rho}{\tau_{DR} + \mu + \delta_{DR} + \eta \rho},$$

so that

$$\boxed{Y_{\rho}^{R_0} = -\frac{\rho}{\tau_T + \rho + \mu + \delta_T} + \frac{\eta \rho}{\tau_{DR} + \mu + \delta_{DR} + \eta \rho}.}$$

Furthermore,

$$Y_{\tau_T}^{R_0} = \frac{\partial R_{0T}}{\partial \tau_T} \cdot \frac{\tau_T}{R_{0T}} = -\frac{\tau_T}{\tau_T + \rho + \mu + \delta_T},$$

thus

$$\boxed{Y_{\tau_T}^{R_0} = -\frac{\tau_T}{\tau_T + \rho + \mu + \delta_T}.}$$

Also,

$$Y_{\tau_{DR}}^{R_0} = \frac{\partial R_{0T}}{\partial \tau_{DR}} \cdot \frac{\tau_{DR}}{R_{0T}} = -\frac{\tau_{DR} \eta \rho}{(\tau_{DR} + \mu + \delta_{DR})(\tau_{DR} + \mu + \delta_{DR} + \eta \rho)},$$

hence

$$\boxed{Y_{\tau_{DR}}^{R_0} = -\frac{\tau_{DR} \eta \rho}{(\tau_{DR} + \mu + \delta_{DR})(\tau_{DR} + \mu + \delta_{DR} + \eta \rho)}.}$$

Similarly,

$$\Upsilon_{\omega}^{R_0} = \frac{\partial R_{0T}}{\partial \omega} \cdot \frac{\omega}{R_{0T}} = -\frac{\omega}{\omega + \mu},$$

so that

$$\boxed{\Upsilon_{\omega}^{R_0} = -\frac{\omega}{\omega + \mu}.$$

Also,

$$\Upsilon_{\delta_T}^{R_0} = \frac{\partial R_{0T}}{\partial \delta_T} \cdot \frac{\delta_T}{R_{0T}} = -\frac{\delta_T}{\tau_T + \rho + \mu + \delta_T},$$

thus

$$\boxed{\Upsilon_{\delta_T}^{R_0} = -\frac{\delta_T}{\tau_T + \rho + \mu + \delta_T}.$$

Again,

$$\Upsilon_{\delta_{DR}}^{R_0} = \frac{\partial R_{0T}}{\partial \delta_{DR}} \cdot \frac{\delta_{DR}}{R_{0T}} = -\frac{\delta_{DR}\eta\rho}{(\tau_{DR} + \mu + \delta_{DR})(\tau_{DR} + \mu + \delta_{DR} + \eta\rho)},$$

hence

$$\boxed{\Upsilon_{\delta_{DR}}^{R_0} = -\frac{\delta_{DR}\eta\rho}{(\tau_{DR} + \mu + \delta_{DR})(\tau_{DR} + \mu + \delta_{DR} + \eta\rho)}.$$

Finally,

$$\Upsilon_{\mu}^{R_0} = \frac{\partial R_{0T}}{\partial \mu} \cdot \frac{\mu}{R_{0T}} = 1 - \frac{\mu}{\omega + \mu} - \frac{\mu}{\tau_T + \rho + \mu + \delta_T} - \frac{\mu\eta\rho}{(\tau_{DR} + \mu + \delta_{DR})(\tau_{DR} + \mu + \delta_{DR} + \eta\rho)},$$

thus

$$\boxed{\Upsilon_{\mu}^{R_0} = 1 - \frac{\mu}{\omega + \mu} - \frac{\mu}{\tau_T + \rho + \mu + \delta_T} - \frac{\mu\eta\rho}{(\tau_{DR} + \mu + \delta_{DR})(\tau_{DR} + \mu + \delta_{DR} + \eta\rho)}.$$

In this case, the parameters associated with the HIV pathway do not determine the dominant threshold value of the full model, and their local effect on the sensitivity of R_0 is negligible as long as R_{0T} remains strictly greater than R_{0H} .

Remark. If

$$R_{0H} = R_{0T},$$

then the reproduction number of the full system is non-differentiable at that point because it is defined as the maximum of two threshold quantities. In such a case, the sensitivity analysis should be interpreted by comparing the separate sensitivities of R_{0H} and R_{0T} .

CONCLUSION

The sensitivity analysis of the full coinfection model shows that the dominant epidemic pathway determines the sensitivity of the overall reproduction number. When HIV transmission dominates, the full model is most sensitive to the HIV transmission rate β_H , the infectiousness modification parameter θ , and the HIV treatment rate τ_H . On the other hand, when TB transmission dominates, the full model is most sensitive to the TB transmission rate β_T , the resistance-related modification parameter η , the progression rate to drug resistance ρ , and the treatment parameters τ_T and τ_{DR} . Therefore, effective control of the coinfection system depends on identifying the dominant transmission pathway and targeting the parameters that most strongly influence that component of the reproduction number.

Numerical Simulation

To complement the analytical results obtained in this study, numerical simulations are carried out to illustrate the dynamical behaviour of the model under different epidemiological conditions. The simulation is structured in three stages in order to provide a clear understanding of each component of the model.

First, the HIV-only submodel is simulated to examine the transmission dynamics of HIV, including the effects of disease progression and treatment. Next, the TB-only submodel is considered to study the spread of tuberculosis, with particular emphasis on vaccination, treatment, and the emergence of drug resistance. Finally, the full HIV–TB–DRTB coinfection model is simulated to capture the interaction between the two diseases and to assess the combined impact of intervention strategies on disease control.

For the purpose of simulation, parameter values are selected from existing literature where available, while parameters without direct epidemiological estimates are assigned biologically reasonable values. This approach is widely adopted in epidemiological modelling, particularly for complex coinfection systems where some parameters are difficult to estimate directly from available data (Hethcote, 2000; Brauer, 2017).

Parameter	Value	Source
Π	1000	(Momoh, 2019)
μ	0.0143	(Momoh, 2019)
ϕ	0.30	(Kirimi, 2024)
ψ	0.05	(Nandi, 2024)
β_H	0.45	(Ayele, 2024)
β_T	12.0	(Momoh, 2019)
β_R	8.0	(Mulugeta, 2025)
α	0.10	(Anderson, 2016)
τ_H	0.50	(WHO, 2023)
τ_T	2.00	(WHO, 2022)
τ_R	1.00	(Mulugeta, 2025)
τ_C	1.20	(Ayele, 2024)
τ_{HR}	0.80	(Momoh, 2019)
ω	0.08	(Moya, 2021)
ω_C	0.10	(Ayele, 2024)
ω_T	0.05	(Moya, 2021)
δ_H	0.03	(UNAIDS, 2023)
δ_T	0.10	(WHO, 2022)
δ_R	0.15	(WHO, 2022)
δ_C	0.12	(Ayele, 2024)
δ_{HR}	0.18	(Mulugeta, 2025)
J	0.80	(Momoh, 2019)
K	1.20	(Ayele, 2024)
η	0.90	(Momoh, 2019)
θ	1.50	(Momoh, 2019)
τ	1.10	(Ayele, 2024)
P	1.30	(Ayele, 2024)

Reproduction Numbers Used for Simulation

Before carrying out the numerical simulations, the threshold quantities of the three models are evaluated using the baseline parameter values. For the HIV submodel, the basic reproduction number is

$$R_{0H} = \frac{\beta_H}{\alpha + \mu + \tau_H} \left(1 + \frac{\theta\alpha}{\mu + \delta_H + \tau_H} \right) = 0.9344.$$

For the TB submodel, the basic reproduction number is

$$R_{0T} = \frac{\beta_T\mu}{(\phi + \mu)(\tau_T + \omega + \mu + \delta_T)} \left(1 + \frac{\eta\omega}{\tau_R + \mu + \delta_R} \right) = 0.2642.$$

For the full coinfection model, the threshold quantity is taken as

$$R_0 = \max\{R_{0H}, R_{0T}\} = 0.9344.$$

Since $R_0 = R_{0H} > R_{0T}$ under the baseline parameter set, the HIV pathway is the dominant transmission component of the full model. Consequently, in addition to varying the HIV-related parameters with strong positive or negative sensitivity influence, selected TB-related parameters are also varied in order to show how changes in the TB pathway affect the coinfection burden.

Simulation Results

Figure A shows the effect of varying the HIV transmission rate β_H on the HIV-only submodel.

A: Effect of varying β_H on the HIV-only submodel

The figure shows that increasing β_H raises the number of HIV-infected individuals and sustains transmission for a longer period of time. This agrees with the sensitivity result, which identified β_H as a strongly positive driver of R_{0H} .

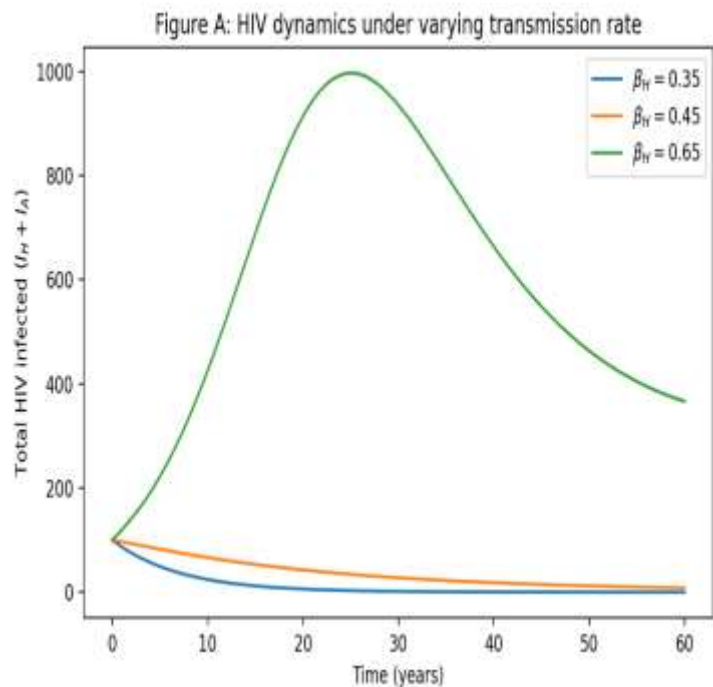


Figure B presents the effect of varying the HIV treatment rate τ_H on the HIV-only submodel.

B: Effect of varying τ_H on the HIV-only submodel

The figure indicates that higher treatment rates reduce the HIV burden more rapidly. This supports the sensitivity analysis, where τ_H had a negative effect on R_{0H} and therefore acts as a control parameter.

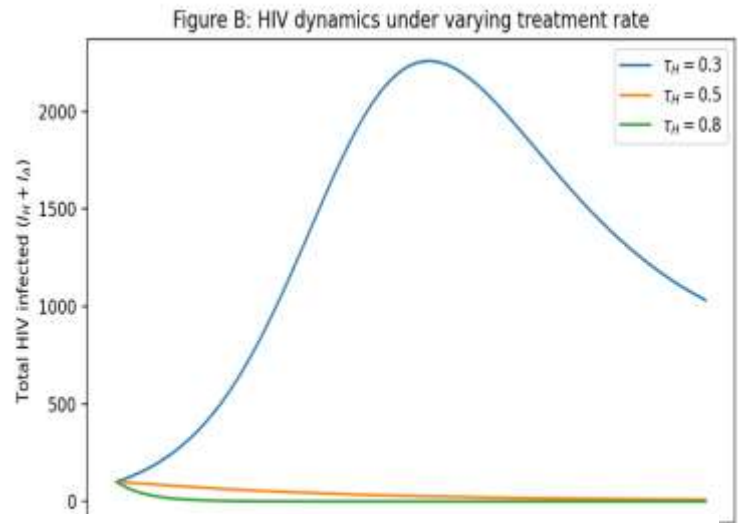


Figure C shows the effect of varying the TB transmission rate β_T on the TB-only submodel.

C: Effect of varying β_T on the TB-only submodel

It is observed that increasing β_T produces a larger TB infectious population and slows down the decline of infection. This confirms that the transmission rate is one of the most influential positive parameters in the TB pathway.

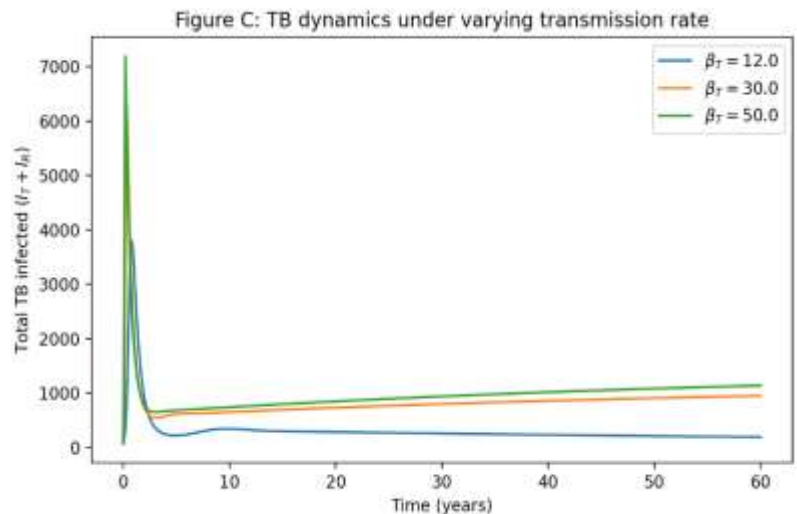


Figure D shows the effect of varying the TB treatment rate τ_T on the TB-only submodel.

D: Effect of varying τ_T on the TB-only submodel

The result shows that increasing τ_T lowers the number of infectious TB individuals and enhances disease control. This is consistent with the negative sensitivity index obtained for the treatment parameter.

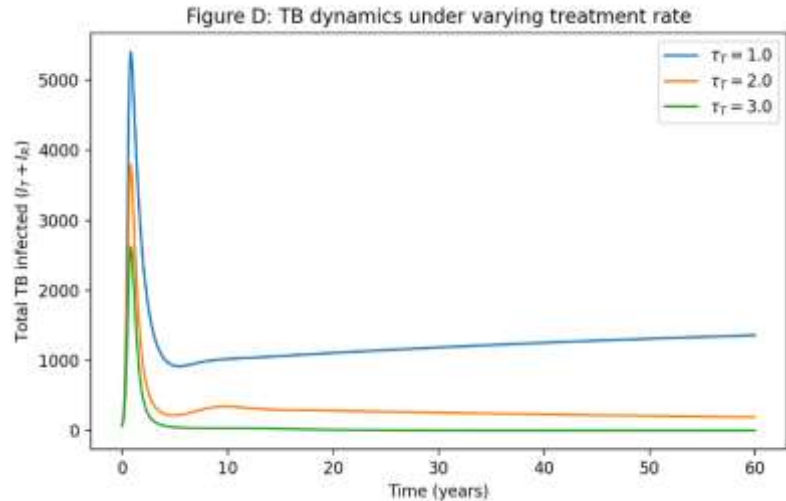


Figure E illustrates the effect of varying β_H on the coinfecting classes of the full model.

E: Effect of varying β_H on the coinfection burden

The graph shows that a higher HIV transmission rate increases the burden of coinfection. This means that stronger HIV transmission not only increases HIV prevalence but also intensifies the interaction between HIV and TB in the full system.

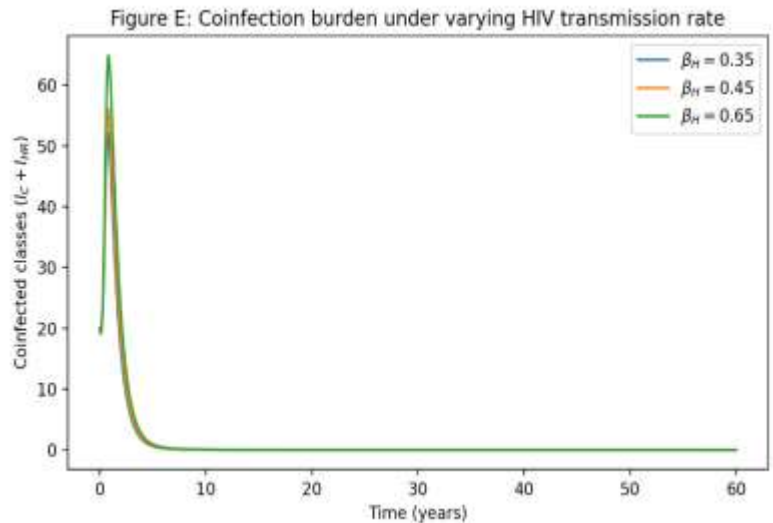


Figure F presents the effect of varying τ_H on the coinfecting classes.

F: Effect of varying τ_H on the coinfection burden

The figure reveals that improved HIV treatment reduces the size of the coinfecting population. This suggests that HIV treatment remains a critical intervention even in the presence of TB and drug-resistant TB.

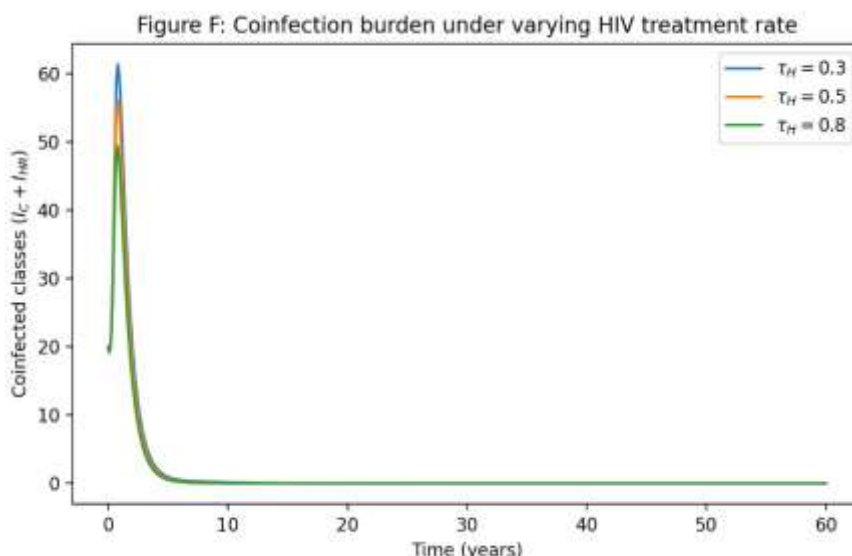


Figure G shows the effect of varying β_T on the coinfecting classes of the full model.

G: Effect of varying β_T on the coinfection burden

The result indicates that increasing TB transmission produces a higher coinfection burden. This reflects the important role of TB exposure in driving HIV–TB interaction within the full model.

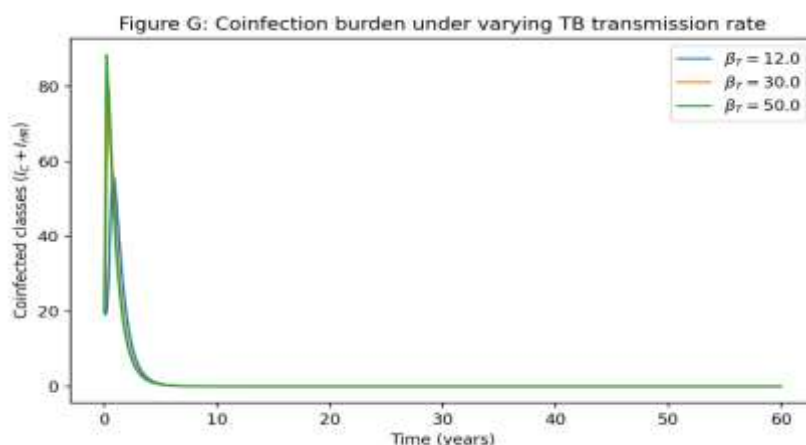
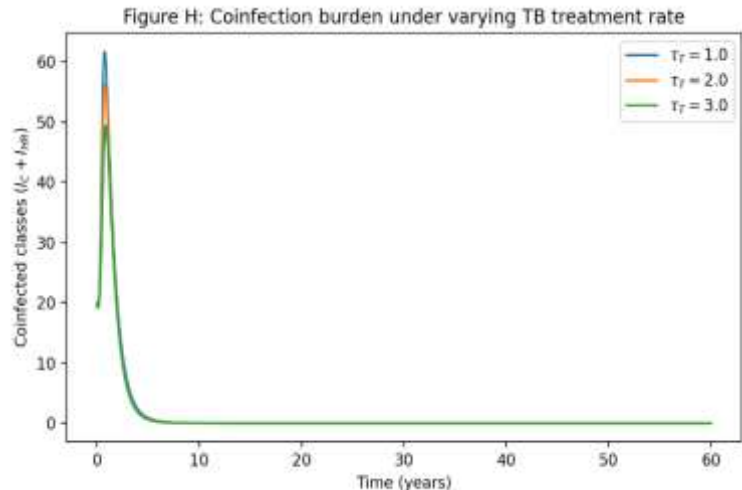


Figure H shows the effect of varying τ_T on the coinfecting classes.

H: Effect of varying τ_T on the coinfection burden

The graph shows that increasing TB treatment reduces the coinfecting population over time. This means that strengthening TB treatment not only lowers TB prevalence, but also weakens the persistence of the joint HIV–TB disease burden in the full model.



DISCUSSION OF RESULTS

The results obtained from the analytical and numerical investigations of the HIV, TB, and HIV–TB–DRTB coinfection models provide important insights into the transmission dynamics of the diseases and the impact of key epidemiological parameters on disease progression and control.

From the computation of the basic reproduction numbers, it was observed that the HIV submodel has a higher reproduction number compared to the TB submodel under the chosen baseline parameter values. This indicates that, in the absence of control measures, HIV transmission has a greater potential to persist in the population than tuberculosis. Consequently, the overall reproduction number of the full model is largely driven by the HIV transmission pathway.

The sensitivity analysis revealed that the transmission rates β_H and β_T are the most influential parameters in their respective submodels, with sensitivity indices equal to one. This implies that any proportional increase in these parameters leads to a corresponding increase in the reproduction numbers. Similarly, the modification parameters θ and η , which account for increased infectiousness due to disease progression and drug resistance, were found to have significant positive effects on disease transmission.

On the other hand, treatment-related parameters such as τ_H , τ_T , and τ_{DR} exhibited negative sensitivity indices, indicating that increasing treatment rates reduces the basic reproduction numbers. This highlights the critical role of treatment in controlling both HIV and tuberculosis. In addition, vaccination-related parameters and disease-induced mortality rates were also found to contribute to the reduction of transmission.

The numerical simulations further supported these analytical findings. An increase in the HIV transmission rate β_H led to a rapid rise in the number of infected individuals, demonstrating the strong influence of contact rates on disease spread. Conversely, increasing the HIV treatment rate τ_H resulted in a significant decline in the infected population, confirming the effectiveness of treatment interventions.

Similarly, in the TB submodel, increasing the transmission rate β_T led to higher levels of infection, while increasing the treatment rate τ_T reduced the number of infectious individuals. The simulations also showed that drug resistance plays an important role in sustaining TB transmission, particularly when treatment is inadequate.

For the full coinfection model, the simulations revealed that the interaction between HIV and TB leads to a more complex disease dynamic. In particular, HIV infection increases susceptibility to TB, thereby amplifying the overall burden of disease. The results showed that controlling only one disease is insufficient; rather, integrated intervention strategies targeting both HIV and TB are required for effective disease control.

Overall, the simulation results are consistent with the theoretical analysis and clearly demonstrate the importance of transmission reduction and treatment improvement in managing the spread of HIV, TB, and drug-resistant TB.

CONCLUSION

In this study, a deterministic compartmental model describing the transmission dynamics of HIV, tuberculosis, and drug-resistant tuberculosis was formulated and analyzed. The model was structured to capture the interaction between the diseases, including the effects of treatment, vaccination, progression, and modification parameters.

The analysis began with the derivation of key properties of the model, including positivity and boundedness, which ensured that the solutions remain biologically meaningful. The disease-free and endemic equilibrium states were obtained for the submodels, and the basic reproduction numbers for the HIV and TB submodels were derived using the next generation matrix method.

Stability analysis showed that the disease-free equilibrium is locally and globally asymptotically stable whenever the reproduction number is less than unity, indicating that the diseases can be eliminated under appropriate conditions. Sensitivity analysis further identified the most influential parameters affecting disease transmission, highlighting the dominant role of transmission rates and treatment parameters.

Numerical simulations were conducted to illustrate the behavior of the system under different parameter variations. The results confirmed that increased transmission rates lead to higher infection levels, while improved treatment and intervention strategies significantly reduce disease prevalence.

The study therefore provides a comprehensive mathematical framework for understanding the dynamics of HIV and TB coinfection and offers valuable insights into the factors that drive disease persistence and control.

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