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Review Article

Dynamic Epidemiological Models for HIV/AIDS Associated TB Transmission: A Review

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Abstract. An extensive literature review suggests a significant link between HIV/AIDS and other diseases such as Pneumocystis pneumonia (PCP), Candidiasis, Cryptococcal meningitis, Toxoplasmosis, and Tuberculosis (TB). Here, our emphasis is on the HIV/AIDS associated Tuberculosis (TB) transmission dynamics. Since the last decade, due to the advancement in machine languages and neural networking, researchers have developed mathematical models to predict and investigate the dynamics of transmission of HIV/AIDS linked Tuberculosis (TB) globally. It includes the therapeutic aspects, like antiretroviral drug screening and treatment factors, in the study. We aim to cover the diversity of developed models to address the numerous key issues of the infection dynamics and provide the status of current and developed models to the global scientific community. Hence, it would help to build highly accurate future models to track the actual dynamics of the concerned disease. In this paper, we discussed deterministic modeling for the HIV/AIDS mediated Tuberculosis (TB) infection dynamics. Additionally, the threshold behaviour and the extended effect of mediations have been discussed. We conclude with an outline of the utilizations and accomplishments of HIV/AIDS-TB modeling and some proposed future directions.

Keywords. Dynamic epidemiological model, HIV/AIDS-TB transmission, Antiretroviral therapy, Deterministic models, Infection dynamics

Mathematics Subject Classification (2020). Primary: 92D30; Secondary: 34C60

1. Introduction

Infectious diseases have long been a leading cause of death in many nations. Some newly identified infections include Lyme disease (1975), Legionnaires' disease (1976), toxic shock syndrome (1978), hepatitis C (1989), hepatitis E (1990), hantavirus (1993), and SARS-CoV-2 (2019). Some antibiotic-resistant strains of Candidiasis, Pneumocystis pneumonia (PCP), Cryptococcal meningitis, Toxoplasmosis, and Tuberculosis (TB) have also emerged (Hethcote [7]). Among these, Tuberculosis (TB) and HIV/AIDS are particularly widespread, especially in resource-constrained nations. TB, caused by *Mycobacterium tuberculosis*, primarily affects the lungs but can also involve other parts of the body. Tuberculosis is a bacterial disease transmitted through the air via infectious droplet nuclei (<5 microns) of *Mycobacterium tuberculosis*. TB usually affects the lungs (pulmonary) but can also spread to other areas of the body (extrapulmonary) (Azeez *et al*. [2]). HIV is a complex infection characterized by diverse time scales, ranging from hours to months (Cassels *et al*. [5], and Rivadeneira *et al*. [15]). The World Health Organization (WHO) has identified 30 high-burden countries (HBCs) for TB and HIV/AIDS-associated TB in the Global TB Report 2023 [1].

In the public health field, mathematical modeling is increasingly recognized as a valuable research tool for infectious disease control (Bhunu *et al*. [4], Roeger *et al*. [14]). Models assessing the co-epidemic of HIV/AIDS and TB can provide important insights into the interaction between these diseases, predict outcomes, and inform intervention strategies (Shastri *et al*. [18]). Mathematical models are widely applied to evaluate the effectiveness of control measures for tuberculosis, HIV, influenza, West Nile virus, and the emerging Zika virus. They are also used to assess the cost-effectiveness of vaccination campaigns and to prevent hospital-acquired infections (Li [10]). Such models play a crucial role in understanding the dynamics of HIV infection in relation to TB (Kirschne [8], Shah *et al*. [17], Silva and Torres [19]). A variety of mathematical models have been developed to describe the immune system and its interaction with HIV/AIDS, as well as the impact of drug therapy (Xiao *et al*. [20]). Mathematical models can also help policymakers allocate resources for the prevention and control of infectious disease epidemics (Long *et al*. [11]). Modeling is particularly important for predicting nonlinear dynamics that arise from the combined effects of medications (Gilbert *et al*. [6]).

Patients with latent *Mycobacterium tuberculosis* infection who are also infected with HIV are at a higher risk of developing active tuberculosis (Shah and Gupta [16]). The AIDS-causing virus targets the human immune system and reduces its efficiency; therefore, these patients are more prone to developing TB (Long *et al*. [11]). ART reduces viral load and lowers the probability of HIV transmission by about 50% (Lusiana *et al*. [12]). While ART reduces the risk of TB in co-infected individuals by up to 80% and significantly increases life expectancy, their risk of developing TB remains several times higher than that of HIV-negative individuals, which may contribute to continued TB transmission (Muthuri and Malonza [13]). Evidence from different countries shows that early administration of antiretroviral drugs in co-infected patients, even during the intensive phase of TB therapy, can reduce the mortality rate associated with HIV–TB co-infection (Adeyemo *et al*. [1], Bacaër *et al*. [3], Hethcote *et al*. [7], Kubjane *et al*. [9], Zhang *et al*. [21]).

[1]World Health Organization, *Global Tuberculosis Report 2023*, World Health Organization, (2023), retrieved from URL: https://www.who.int/teams/global-tuberculosis-programme/tb-reports/global-tuberculosis-report-2023.

Various mathematical and computer simulation models have been developed to describe TB–HIV co-infection in regions such as sub-Saharan Africa, the USA, Russia, India, and Brazilian prisons. Some models have attempted to provide a global perspective by considering all five WHO regions, while others have addressed the issue more broadly without focusing on a single setting.

Many models use systems of *ordinary differential equations* (ODEs), while others are based on discrete-time difference equations (Muthuri and Malonza [13]). This review focuses on deterministic models developed to study the transmission dynamics of HIV/AIDS-TB co-infection, highlighting advances in modeling techniques and their implications for public health.

Our goal is to examine previously developed mathematical models of HIV/AIDS and TB epidemics in combination. By reflecting on their contributions and limitations, we aim to show how these models can inform the design of effective interventions and support better strategies for controlling the dual epidemic.

2. Review of HIV/AIDS-TB Co-Infection Models

Here, Table 1 represent the summary of reviewed HIV/AIDS-TB co-infection models highlighting their objectives, methodologies, main findings, and limitations. It provides a comparative brief overview of selected HIV/AIDS-TB co-infection models.

3. Methods: Description of Selected HIV/AIDS-TB Co-Infection Models

This section provides a detailed description of selected HIV/AIDS–TB co-infection models.

3.1 Comparative Analysis

A detailed comparison of different modeling approaches reveals the strengths and limitations of each method (Li [10]):

(1) *Deterministic Models*: Often used for their simplicity and ability to provide clear insights into the disease dynamics. However, they may not capture the randomness inherent in disease transmission.

(2) *Stochastic Models*: Offer a more nuanced understanding by incorporating random variations, but are computationally intensive.

(3) *Agent-Based Models*: Provide detailed simulations of individual interactions but require significant computational resources and detailed data.

3.2 Early Models and Their Evolution

We describe deterministic models for HIV/AIDS–TB transmission dynamics, particularly those developed to understand antiretroviral drug responses for HIV infection and TB treatment (Xiao *et al*. [20]). We also explain how these models have been extended to account for drug efficacy in both HIV/AIDS and TB.

Table 1. Description of developed models of HIV/AIDS-TB co-infections

S. No.	Authors' name	Year	Title	Objective	Method	Key findings	Limitations/ Research gap
1	Kirschner	1999	Dynamics of co-infection with M. tuberculosis and HIV-1	To study the dynamics of co-infection of TB and HIV-1	Theoretical modeling using differential equations	Identified critical parameters influencing co-infection dynamics	Limited to theoretical model without empirical validation
2	Muthuri and Malonza	2018	Mathematical Modeling of TB-HIV Co Infection, Case Study of Tigania West Sub County, Kenya	To model TB-HIV co-infection in a specific Kenya sub-county	Mathematical modeling and case study approach	Provided insights specific to the local population dynamics	Case study limited to one region; may not be generalizable
3	Bacaër *et al*.	2008	Mathematical Biology Modeling the joint epidemics of TB and HIV in a South African township	To model joint epidemics of TB and HIV in a South African township	Mathematical biology models	Highlighted the interaction effects between TB and HIV epidemics	Focused on a single township; results may not be widely applicable
4	Lih-Ing *et al*.	2009	Modeling TB and HIV Co-infections	To develop a model for TB and HIV co-infections	Mathematical biosciences engineering	Presented a detailed model for co-infections	Lacked empirical data validation
5	Bhunu *et al*.	2009	Modeling HIV / AIDS and Tuberculosis Coinfection	To model the co-infection dynamics of HIV/AIDS and TB	Use of differential equations and dynamic systems	Identified key factors in co-infection spread	Theoretical model without field data
6	Silva *et al*.	2014	Modeling TB-HIV Syndemic and Treatment	To model the syndemic interaction and treatment of TB and HIV	Mathematical modeling	Examined the impact of treatment strategies on co-infection dynamics	Limited to specific treatment scenarios; lacks broader applicability
7	Shah *et al*.	2020	Dynamics of HIV-TB Co-infection Model	To study the dynamics of HIV-TB co-infection	Use of dynamical systems and stability analysis	Provided insights into the stability and behaviour of co-infection models	Primarily theoretical; requires empirical validation

For instance, Kirschner [8] developed one of the earliest dynamic models to study HIV–TB co-infection, focusing on the interaction between T cells and macrophages with HIV and *M. tuberculosis*. This model demonstrated the significant impact of co-infection on immune system dynamics.

The mathematical model of Bacaër *et al*. [3] examines the impact of various control measures such as condom promotion, increased TB testing, anti-TB therapy, and antiretroviral therapy on the HIV-TB co-epidemic using data from a community near Cape Town, South Africa, where the HIV incidence rate exceeds 20% and the TB notification rate is close to 2,000 per 100,000 per year.

Roeger *et al*. [14] presented a simplified deterministic model of the joint epidemic of TB and HIV in a pseudo-competitive environment at the population level. Their findings suggest that greater investment in reducing HIV prevalence could be an effective approach to limiting or controlling the effects of tuberculosis.

We use the following terms to describe the compartments of the model: S represents susceptible individuals; L_{TB}, denotes latent TB; I_{TB}, denotes infectious TB; and T_{TB}, represents TB-treated individuals.

The model for HIV/TB interaction consists of eight ordinary differential equations. In this context, however, we describe only the four equations that specifically address the transmission dynamics of HIV and TB. H_1 represents HIV-infectious only; H_2 represents HIV-infectious with latent TB; H_3 represents HIV-infectious with TB; and A represents AIDS (Roeger *et al*. [14]),

$$\dot{H}_1 = \lambda\sigma(S + T_{TB})(H^*/R) - \beta c H_1 \frac{I_{TB} + H_3}{N} - (\alpha_1 + \mu)H_1 + r^* H_2, \tag{3.1}$$

$$\dot{H}_2 = \lambda\sigma L_{TB}\frac{H^*}{R} + \beta c H_1 \frac{I_{TB} + H_3}{N} - (\alpha_2 + \mu + k^* + r^*)H_2, \tag{3.2}$$

$$\dot{H}_3 = k^* H_2 - (\alpha_3 + \mu + d^*)H_3, \tag{3.3}$$

$$\dot{A} = \alpha_1 H_1 + \alpha_2 H_2 + \alpha_3 H_3 - (\mu + f)A, \tag{3.4}$$

where N denote the total population, R denote the total active population ($= N - I_{TB} - H_3 - A = S + L_{TB} + T_{TB} + H_1 + H_2$) and other parameters are as follows.

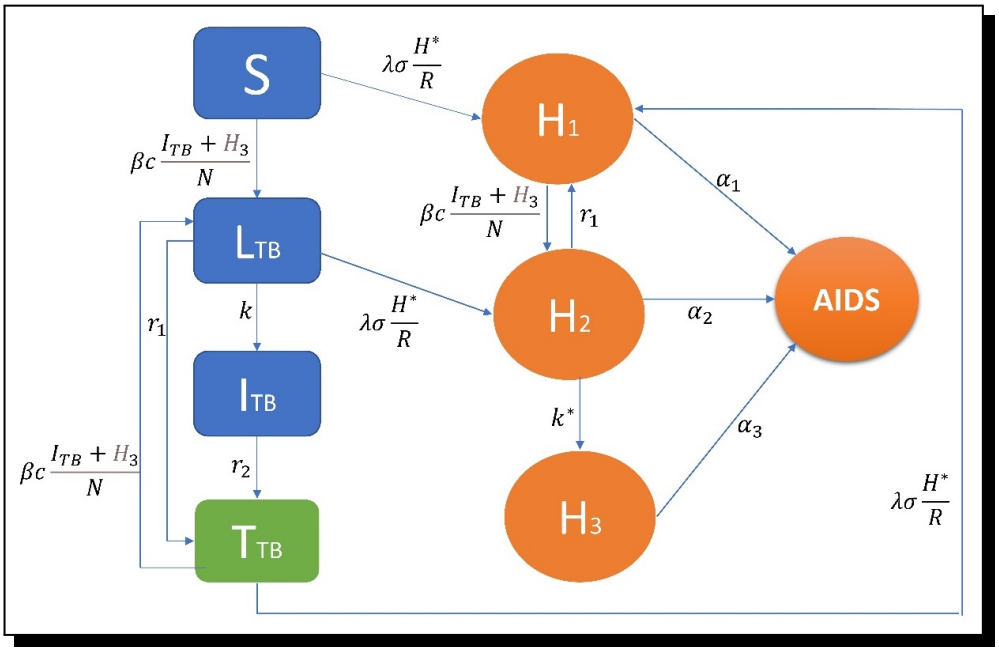

Figure 1. Transmission diagram for the HIV-TB model

One critical parameter to include in the model is k^*, which represents the rate of TB progression in individuals co-infected with HIV and latent TB. Studies have shown that people with both HIV and latent TB are 30 to 50 times more likely to develop active TB than HIV-negative individuals with latent TB. If $\alpha_3 \gg \alpha_1$ (meaning the progression of HIV to AIDS is substantially faster in people also infected with TB than in those without TB), the effect of k

becomes negligible or non-existent. Increasing efforts to reduce the risk of HIV infection can therefore be an effective way to prevent or limit the impact of tuberculosis (Roeger *et al*. [14]).

Table 2. Parametric definitions

Notations	Description
H^*	Individual with HIV who has not developed AIDS ($= H_1 + H_2 + H_3$)
β	Probability of TB infection per contact with a person with active TB
λ	Probability of HIV infection per contact with a person with HIV
c	The per-capita contact rate for TB
σ	The per-capita contact rate for HIV
μ	The per-capita natural death rate
k^*	The per-capita rate of TB progression for individuals infected with HIV
d	The per-capita rate of death due to TB
d^*	The per-capita rate of death due to HIV
f	The per-capita rate of death due to AIDS
r^*	The per-capita rate of latent TB treatment for individuals with HIV
α_i	The per-capita rate of AIDS progression for individuals in H_i ($i = 1,2,3$) class

3.3 Complex and Recent Models

Subsequent studies have built on this work by incorporating more complex interactions and therapeutic interventions.

Bhunu *et al*. [4] developed a comprehensive model with compartments for different stages of TB and HIV infection, integrating the effects of antiretroviral therapy (ART) and TB treatment. Their findings provided insights into transmission dynamics and underscored the importance of early intervention in co-infected patients.

The model includes several compartments: I_{HIV} represents HIV-infectious individuals; A, represents AIDS; E_{AT} represents individuals with AIDS who have been exposed to TB; and I_{TH} represents HIV-infected individuals in the pre-AIDS stage who are showing the TB symptoms. Here, the rate ρ_1 denotes HIV-positive patients with TB who progress to AIDS, while the rate ρ_2 denotes AIDS patients who develop active TB after being exposed to TB. In addition, such patients may also develop TB at rate ρ_3, with different modification parameters.

$$\dot{A}_{AT} = \rho_1 I_{TH} + \rho_2 E_{AT} - (\mu + d + f)A_{AT} + \rho_3 E_{AT}\,. \tag{3.5}$$

In the above equation (3.5), the compartment A_{AT} denotes AIDS patients who are also sick with tuberculosis. This co-infection model has been expanded to include antiretroviral therapy for AIDS patients and antibiotic treatment for tuberculosis patients, assessing the impact of these interventions (Bhunu *et al*. [4]). Active TB is treated with first-line medications (rifampicin, isoniazid, pyrazinamide, and ethambutol) taken daily for two months, followed by four months of rifampicin and isoniazid. In addition, isoniazid is provided to individuals who have been exposed.

$$\dot{A}_{AT} = \rho_1 I_{TH} + \rho_2 E_{AT} - (\mu + d + f + \tau_1 + \tau_2)A_{AT} + \rho_3 E_{AT}\,. \tag{3.6}$$

Here, τ_1 and τ_2 denote the treatment rates for AIDS and TB, respectively, within the co-infected population. When people with active HIV/AIDS begin antiretroviral therapy, their health status improves and they may appear healthy. The number of individuals who recover from TB without being co-infected with HIV initially increases, but this rise does not persist; it soon reaches a peak and then declines to lower levels. This decline may be due to subsequent HIV infection among these individuals (Bhunu *et al*. [4]).

It has been observed that TB patients receiving therapy for HIV/AIDS respond equally well to treatment. Research shows that TB in HIV patients worsens the overall condition and increases the mortality rate. However, evidence also indicates that providing antiretroviral therapy to HIV patients has a significant impact on reducing the spread of TB infection. This study did not include individuals who were co-infected with HIV–TB but had already recovered from TB (Bhunu *et al*. [4]).

Silva and Torres [19] proposed a mathematical model for both TB and AIDS treatment for individuals with either one or both infections. HIV and TB are considered a severe human co-morbid epidemic, where co-morbidity refers to the convergence of two or more diseases that act synergistically to intensify the burden of illness. The model is structured with 10 mutually exclusive compartments. In this framework, individuals who recover naturally from TB move into the R_{TH} compartment, while those infected with HIV, showing AIDS symptoms, and co-infected with TB transition into the A_T compartment. The system of differential equations for the HIV-TB co-infection compartment is given as follows:

$$\dot{R}_{TH}(t) = \tau_2\, I_{TH} + r^* L_{TH} - (\rho_4 + \rho_5 + \mu) R_{TH}\,, \tag{3.7}$$

$$\dot{A}_T(t) = \rho_6 I_{TH} + \rho_5 R_{TH} - (\tau_1 + d_{TA} + \mu) A_T\,. \tag{3.8}$$

where the rate ρ_6 denotes the progress of AIDS-TB infection. r^* represents the treatment rate for the class L_{TH}. Individuals in the class R_{TH} can lose temporary immunity, become reinfected with TB, and progress to active TB at rate ρ_4. Individuals in the R_{TH} class may also move to the A_T class at rate ρ_5. The parameter d_{TA} denotes the death rate of individuals suffering from AIDS and TB in the A_T class (Silva and Torres [19]).

Treatment impact: The death rate is higher for individuals infected with both AIDS and TB compared to untreated active-TB patients. When anti-TB drugs are administered, both latent and active TB individuals with HIV infection can recover and move into the R_{TH} class (Silva and Torres [19]). However, infections among individuals suffering from active TB within the HIV community can contribute to the persistence of the disease at endemic levels (Lusiana *et al*. [12]).

3.4 Impact of interventions

Recent advancements in machine learning and neural networks have further refined these models, enabling more accurate predictions of disease dynamics. Muthuri and Malonza [13] used data from Tigania West Sub-County in Kenya to develop a model that highlights the synergistic relationship between HIV/AIDS and TB, showing how effective treatment of one disease can help reduce the spread of the other. The sexually active human population is divided into nine compartments, including the class T_{TH} which represents individuals receiving treatment for both TB and HIV.

$$\dot{T}_{TH} = \tau_2 I_{TH} - (\tau_4 + \mu + d^*) T_{TH}\,. \tag{3.9}$$

Here, τ_2 is the treatment rate of I_{TH}, d^* is the death rate due to HIV, and τ_4 is the recovery rate of TB in the T_{TH} compartment. In this case, a sensitivity analysis is performed to identify which parameters have the greatest impact on the basic reproduction number (R_0). The analysis shows that the partial derivative of R_0 with respect to the treatment rate τ_3 of infectious TB is less than zero,

$$\frac{\partial R_{0T}}{\partial \tau_3} < 0. \tag{3.10}$$

This directly implies that when individuals undergo TB treatment, the basic reproduction number (R_0) decreases. This shows that with effective medication, TB can be controlled. It is also observed that the partial derivative of R_0 with respect to the treatment rate τ_2 of TB in the HIV/AIDS-TB-infected community is less than zero,

$$\frac{\partial R_{0T}}{\partial \tau_2} < 0. \tag{3.11}$$

This means that when treatment is initiated in the co-infected community, the basic reproduction number (R_0) decreases. This is the most effective way to reduce TB infection among individuals with HIV/AIDS. When the values of the parameters τ_3 and τ_2 increase, the rate of TB treatment also increases. This indicates that treatment is an effective control measure for TB (Muthuri and Malonza [13]).

Another HIV-TB co-infection model was formulated by Shah *et al*. [17], in which the susceptible population is classified as HIV-infected. The model consists of seven distinct compartments, including treatment (M), pre-AIDS (P_A), and pre-AIDS with TB infection (P_{ATB}). The use of antiretroviral therapy (ART) slows the progression from HIV to AIDS, as HIV cannot be cured. The basic reproduction number, $R_0 = 2.262 > 1$, was calculated by the next-generation matrix method, indicating the expected number of new infections in an HIV-infected population caused by individuals with AIDS or TB.

$$\dot{M} = \tau_2\, I_{HTB} + \tau_5 I_H + \tau_6 P_A + \tau_7 P_{ATB} - (\mu + \tau_8) M. \tag{3.12}$$

Individuals infected with HIV and those co-infected with HIV-TB are going for treatment and move to treatment class M at rates τ_5 and τ_2. Individuals from the pre-AIDS class who undergo antiretroviral treatment also join class M at a rate τ_6. Those in the P_{ATB} class are treated for TB at a constant rate τ_7. HIV is not curable, individuals who recover from TB but remain HIV-infected eventually progress to the full-blown AIDS (A) class at a constant rate τ_8 (Shah *et al*. [17]).

In the analysis, the equilibrium points represent scenarios with no co-infection, where individuals in the pre-AIDS–TB class show stability, and these are defined as bifurcation points. Sensitivity analysis of R_0 with respect to parameter τ_5 indicates that the number of people receiving medication can be further increased through public awareness campaigns. During the pre-AIDS stage, half of the population received treatment for tuberculosis. Patients co-infected with TB were treated for 11 months. About 31% of HIV-infected individuals developed AIDS within 26 months. During the pre-AIDS stage, half of the population received treatment for tuberculosis. Since HIV/AIDS is incurable, even after therapy, approximately 17% of cases result to AIDS (Shah *et al*. [17]).

Silva and Torres [19] examined the effectiveness of different TB and HIV interventions, demonstrating the critical role of ART and TB preventive therapy in mitigating the dual epidemics. Anti-tuberculosis drugs are often more affordable and accessible than antiviral medications, TB is usually the primary focus of treatment in most cases of HIV-TB co-infection.

4. Discussion

The reviewed models provide valuable insights into the dynamics of HIV/AIDS-TB co-infection. For example, Kirschner's model [8] demonstrated that co-infected individuals have significantly lower T-cell counts compared to those with HIV alone, highlighting the compounded impact of co-infection on the immune system.

Bhunu *et al*. [4] showed that early ART intervention significantly reduces the transmission of both HIV and TB. Their model indicated that ART not only prolongs the life expectancy of co-infected individuals but also reduces the overall transmission rate of TB. Muthuri and Malonza [13] emphasized the importance of simultaneous treatment for TB and HIV. Their sensitivity analysis revealed that increasing the treatment rate for TB in HIV-infected individuals significantly reduces the basic reproduction number for both diseases.

Other models support and extend these findings. Silva and Torres [19] stressed the importance of treatment coverage in reducing the dual burden of HIV and TB, while Shah *et al*. [17] highlighted parameter sensitivity as a key tool for identifying effective control strategies. Roeger *et al*. [14] applied co-infection models to evaluate different therapeutic interventions, and Bacaër *et al*. [3] demonstrated, in a South African context, how local epidemiological conditions strongly shape transmission outcomes. Similarly, Kubjane *et al*. [9] showed through a long-term analysis that large-scale ART rollout has significantly altered TB incidence trends in South Africa.

4.1 Synthesis of Insights From Reviewed Models

The reviewed studies highlight the complexity of HIV-TB co-infection and show the value of mathematical modeling in capturing its dynamics. Although individual models differ in structure and assumptions, several common findings emerge: early ART initiation slows HIV progression and reduces TB incidence; treating HIV and TB together gives better results than focusing on one disease alone; and demographic, behavioural, and health system factors remain underexplored, even though they are critical for accurate modeling.

To bring these findings together, we present a synthesis framework that combines the main elements identified in the reviewed models (Figure 2). This diagram shows how HIV-related factors such as immune suppression and viral load, TB-related factors including latent and drug-resistant TB, and broader epidemiological drivers like poverty and limited healthcare access interact to shape the burden of co-infection. The interaction between the two diseases creates a synergistic effect, with HIV accelerating TB progression and TB worsening outcomes in people living with HIV.

The diagram integrates key insights from existing mathematical studies, showing how HIV-related factors, TB-related factors, and epidemiological drivers interact to sustain the dual epidemic. This synthesis underscores the need for integrated treatment strategies and

the development of more comprehensive models that account for demographic and healthcare system variability.

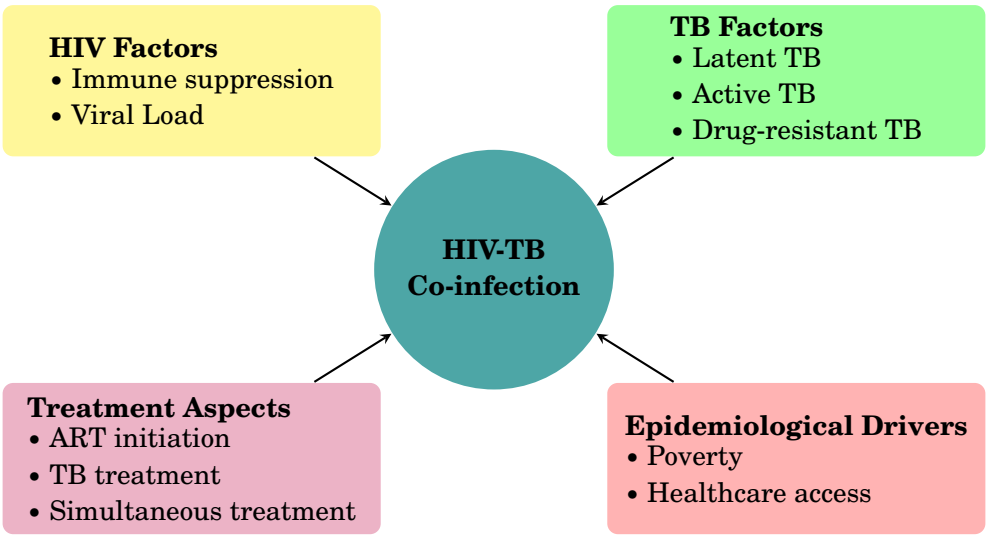

Figure 2. Synthesis of factors influencing HIV-TB co-infection dynamics as highlighted in reviewed models

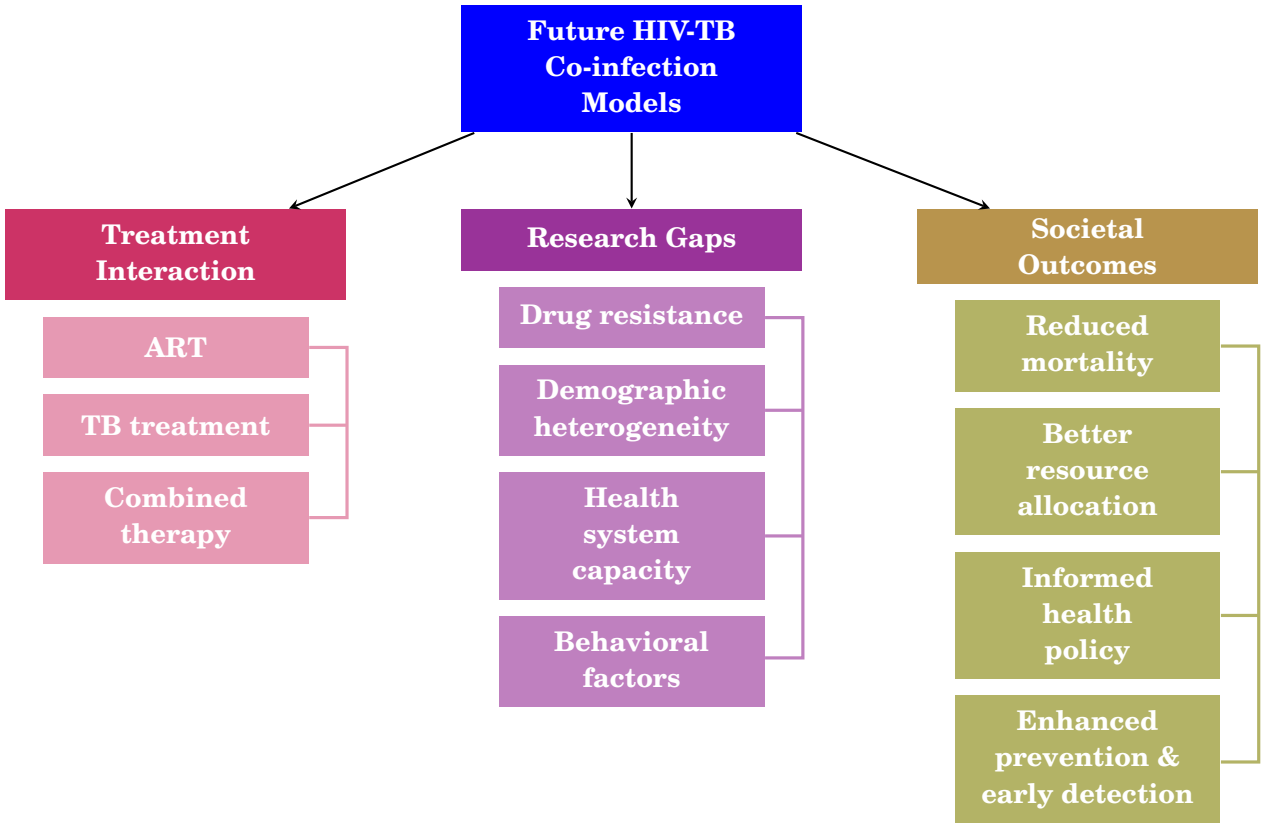

Figure 3. Future HIV-TB co-infection models: A framework for advancing research and societal impact

5. Conclusion

The reviewed models demonstrate that mathematical approaches are essential for understanding the dual epidemic of HIV/AIDS and tuberculosis. They consistently show that early initiation of ART slows HIV progression and reduces the risk of TB, while simultaneous treatment for both infections leads to better outcomes than treating them separately. The models also highlight the role of threshold behaviour in shaping epidemic trends. At the same time, important limitations remain, as factors such as demographic variability, drug resistance, behavioural influences, and health system constraints are often simplified or overlooked.

This diagram illustrates the key directions for improving future model design. On the left, it highlights treatment-related aspects, such as ART, TB therapy, and combined regimens, where further investigation is needed, especially regarding adherence and drug–drug interactions. In the center, major research gaps are presented, including demographic differences, drug resistance, and health system capacity, which should be incorporated into future models. On the right, the possible societal outcomes are shown, such as reduced mortality, better resource allocation, stronger public health policies, and improved prevention strategies. In addition, the integration of machine learning and advanced computational methods with traditional epidemiological modeling is emphasized as a way to increase predictive accuracy and real-world usefulness.

Overall, these insights show that the future of HIV-TB modeling should focus not only on advancing theoretical understanding but also on creating practical tools for policymakers and health systems. Well-designed models can help guide treatment strategies, strengthen prevention programs, and contribute to reducing the global burden of HIV-TB co-infection.

Competing Interests

The authors declare that they have no competing interests.

Authors' Contributions

All the authors contributed significantly in writing this article. The authors read and approved the final manuscript.

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