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Original Research Article

Asthma and Tuberculosis: Implications for Onset, Clinical Progression, and Therapeutic Management

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Abstract: ***Introduction*:** Asthma and tuberculosis (TB) are two major lung diseases whose coexistence could alter the risk of onset, clinical course, and therapeutic management of TB. This review aimed to synthesize the available data on the impact of asthma on these various aspects. ***Methods***: A narrative literature review was conducted in PubMed, Semantic Scholar, Google Scholar, and the Cochrane Library. Publications in English or French published between 2010 and 2025 were considered. Original articles, systematic reviews, meta-analyses, and clinical guidelines addressing the asthma–tuberculosis relationship were included. ***Results***: The data suggested that asthma may increase susceptibility to TB, primarily through the prolonged use of inhaled or systemic corticosteroids, with a dose-dependent effect. A Th1/Th2 immune imbalance, certain genetic factors, and common environmental and socioeconomic determinants may also play a role. The coexistence of the two diseases could lead to atypical clinical presentations and therapeutic challenges, particularly due to interactions between rifampin, corticosteroids, isoniazid, and theophylline. However, some data suggested a possible protective effect of eosinophilic inflammation. ***Conclusion***: Asthma may promote the onset, reactivation, and progression of TB, but the evidence remains mixed and is primarily observational. Longitudinal and interventional studies are needed to clarify this association and optimize the management of patients with this comorbidity.

Keywords: Asthma, Tuberculosis, Corticosteroids, Immunity, Th1/Th2, Comorbidity, Drug interactions, Mycobacterium Tuberculosis.

INTRODUCTION

Asthma, a disease characterized by inflammation of the airways in response to harmless environmental compounds, and tuberculosis, caused by *Mycobacterium tuberculosis*, are two major lung diseases that share complex inflammatory mechanisms and significant clinical implications (Gillissen & Paparoupa, 2014). Tuberculosis (TB) remains one of the leading causes of death from infectious diseases worldwide (Floyd *et al.*, 2023; World Health Organization, 2025). At the same time, asthma is a common chronic respiratory disease that affects hundreds of millions of people of all ages worldwide (Enilari & Sinha, 2019; Song *et al.*, 2022). The coexistence of asthma and tuberculosis represents a growing clinical challenge, particularly in countries with a high burden of TB (Fekih *et al.*, 2010; Nguyen-Ho *et al.*, 2021). Although distinct in their etiology, growing evidence suggests a phenotypic overlap and a complex interaction between these two diseases, particularly with regard to the pathophysiology of inflammation and immune responses (Ricciardolo *et al.*, 2023; Spiropoulos *et al.*, 2015). Although numerous studies have explored the interactions between chronic respiratory diseases and tuberculosis, specific data on the impact of asthma on tuberculosis are scarce and sometimes contradictory (Gyawali *et al.*, 2023; Hamada *et al.*, 2021). A comprehensive narrative review is therefore needed to synthesize current knowledge and guide clinical practice and research. Accordingly, this review aims to synthesize the available data on the impact of asthma on the risk, clinical presentation, and therapeutic management of tuberculosis.

METHODOLOGY

Literature Search Strategy

The literature search was conducted in PubMed, Semantic Scholar, Google Scholar, and the Cochrane Library. Key terms included: *asthma, tuberculosis (TB), chronic respiratory disease, corticosteroids, immune response,* and *treatment outcomes*, combined using Boolean operators (AND, OR), for example: *"Asthma AND tuberculosis AND corticosteroids*." Only publications from 2010 to 2025 were considered. Only articles written in English or French were included in the analysis to ensure a thorough understanding of the scientific content.

Study Selection Criteria

Studies eligible for this review included original articles, systematic reviews, meta-analyses, and clinical guidelines addressing the relationship between asthma and tuberculosis, regardless of participants' age or geographic location. Case reports, commentaries, or editorials without original data, as well as articles not available in full text, were excluded.

Epidemiology of Asthma and Tuberculosis

The epidemiology of asthma and tuberculosis (TB) reveals two major global health crises whose interactions are complex and often bidirectional. In 2024, an estimated 10.7 million people contracted the disease, and 1.23 million died from it (World Health Organization, 2025). TB remains the leading cause of death from a single infectious agent worldwide, ahead of HIV/AIDS (World Health Organization, 2025). Eight countries account for two-thirds of the global burden, with India being the hardest-hit country, accounting for 25% of cases (World Health Organization, 2025).

Asthma affects approximately 300 million people worldwide, causing nearly 250,000 deaths per year. Although the mortality rate is lower than that of TB, its economic impact is massive, sometimes exceeding the combined costs of TB and HIV in certain countries (Gyawali *et al*., 2023).

The prevalence of comorbidity between asthma and TB is relatively poorly documented, but several studies indicate a significant association. In particular, the study conducted by Bogomolov *et al.,* (2020) showed that asthma was diagnosed in 5.4% of patients with active tuberculosis, with annual variations ranging from 2.8% to 7.3% (Bogomolov & Зайков, 2020). Similarly, Adchitre *et al.,* (2020) reported a 4% incidence of pulmonary tuberculosis among patients with chronic asthma (Adchitre & Adchitre, 2020). These figures highlight the need for a more in-depth examination of the risk factors and underlying mechanisms that could explain this comorbidity, as well as its potential impact on diagnostic and therapeutic approaches (Hamada *et al*., 2021).

Impact of Asthma on the Development of Tuberculosis

The influence of asthma on the development of tuberculosis (TB) is complex and results from the interaction between the pathophysiology of the disease, the immunosuppressive effects of treatments, and certain shared genetic factors. Asthma patients exhibit changes in immune response, bronchial architecture, and lung function that may influence susceptibility to tuberculosis, its clinical presentation, and its course (Fang *et al*., 2020). Long-term use of inhaled or systemic corticosteroids in asthma may alter the risk of tuberculosis infection and treatment outcomes (Castellana *et al*., 2019; Lee *et al*., 2013).

Available studies indicate that the use of corticosteroids for the treatment of asthma is a major factor increasing susceptibility to tuberculosis by weakening local and systemic immune defenses. A study conducted in Quebec by Brassard *et al.,* (2010) showed that inhaled corticosteroids (ICS) increase the risk of TB, particularly in patients not receiving oral corticosteroids, with the risk doubling at the highest doses (Brassard *et al*., 2010). Similarly, Lee *et al*., (2013), in Seoul (South Korea), demonstrated that the risk of TB increases in a dose-dependent manner among IDU, with a particular vulnerability associated with prolonged, high-dose treatments (Lee *et al*., 2013). In Taiwan, Chung *et al*., (2014) reported that the use of CSI in patients with chronic respiratory diseases, including asthma, increases the risk of pulmonary TB by a factor of 2.04, with this risk rising to 4.31 when patients use both inhaled and oral corticosteroids simultaneously (Chung *et al*., 2014). In India, Tiwari *et al*., (2018) observed a TB incidence of 5.26% among asthmatics using corticosteroid metered-dose inhalers, compared with no infections in the control group (Tiwari, 2018). Finally, Anees *et al*., (2022) had emphasized that systemic corticosteroid therapy carries a significantly higher risk than the inhaled route, with 19% of patients on oral steroids developing TB compared to only 3.2% of those using inhalers alone (Anees *et al*., 2022).

The risk of tuberculosis associated with inhaled corticosteroids may persist for several years after treatment is discontinued (Lee *et al*., 2018). In 2018, Lee *et al*., observed high vulnerability for up to three years after the end of treatment (Lee *et al*., 2018). Furthermore, Jian *et al.,* reported that asthmatics who developed TB after using ICS had an increased risk of lung cancer (HR 2.52), likely due to persistent chronic inflammation (Jian *et al*., 2016).

Certain genetic variations appear to influence the risk of both asthma and tuberculosis. A study conducted in Russia showed that the IL10 gene (rs1800872) increases susceptibility to both atopic asthma and pulmonary tuberculosis (Yu *et al*., 2016).

Conversely, Yii *et al*., in Singapore, observed that patients with asthma had a 45% reduced risk of developing TB, suggesting that the eosinophilic inflammation characteristic of asthma may exert a protective effect against *Mycobacterium tuberculosis* (Yii *et al*., 2018). This paradox raises questions about the specificity of the immune response and the impact of different asthma phenotypes on the pathogenesis of tuberculosis (Yii *et al*., 2018).

Overall, asthma primarily acts as a risk factor for tuberculosis through its immunosuppressive treatments. However, the natural inflammation associated with asthma could, in certain contexts, provide partial resistance to infection.

Pathophysiological Mechanisms

Asthma can influence the risk of tuberculosis (TB) through several pathophysiological mechanisms, involving both treatments and immune imbalances.

1. Corticosteroid-Induced Immunosuppression

The main documented mechanism is the weakening of the immune system by anti-asthma treatments, namely corticosteroids (Lee *et al*., 2013). Prolonged corticosteroid therapy can lead to transient sequestration of T lymphocytes, particularly CD4+ cells, causing lymphopenia and impairing the immune response against Mycobacterium tuberculosis reactivation (Fekih *et al*., 2010). The use of CSI via metered-dose inhalers also affects antibody formation and cellular immunity, thereby promoting the reactivation of latent TB (Tiwari, 2018).

2. Th1/Th2 Imbalance

Protection against TB relies on a Th1-type response, mediated by the activation of macrophages by interferon-gamma (Yii *et al*., 2018). However, asthma is characterized by a Th2-type immune response, leading to a shift in the cytokine profile and a potential reduction in the body's ability to contain tuberculosis infection (Hamada *et al*., 2021; Tarancón *et al*., 2021) . In patients with asthma and tuberculosis, an imbalance in the immune system results in a conflict between the Th1 response—which is essential for controlling *Mycobacterium tuberculosis* (Mtb)—and the Th2 response, which is characteristic of asthma (Lee *et al*., 2025). This Th2 response, characteristic of asthma, promotes mast cell activation and the polarization of macrophages toward an alternative M2 phenotype, contributing to airway remodeling (Lee *et al*., 2025). Furthermore, this Th2- dominance antagonizes the Th1 response and reduces the production of interferon-gamma (IFN-γ), which is necessary to activate macrophages and eliminate intracellular bacteria (Lee *et al*., 2025). Histamine released by hyperactivated mast cells further inhibits IFN-γ synthesis by T and NK cells, weakening antimycobacterial defenses (Lee *et al*., 2025). This imbalance increases susceptibility to tuberculosis infection, disrupts granuloma formation and stability, and may accelerate pulmonary fibrosis (Lee *et al*., 2025).

3. Shared Genetic and Molecular Factors

Some people are born with a version of the IL10 gene that alters their immune response, which can both increase the risk of asthma and affect susceptibility to or the severity of tuberculosis. (Yu *et al*., 2016). Studies have shown that high levels of IL-10, associated with an enhanced Th2 response, can inhibit the Th1 responses necessary for the elimination of *Mycobacterium tuberculosis* and promote the reactivation of latent tuberculosis (Chin *et al*., 2023). Consequently, downregulation of IL-10 could enhance anti-tuberculosis immunity in patients with asthma, although this may exacerbate their Th2 inflammation (Fekih *et al*., 2010; López-Cervantes *et al*., 2022).

4. The Paradox of Eosinophilic Inflammation

In certain contexts, the eosinophilic inflammation characteristic of asthma may exert a protective effect against *M. tuberculosis*, as eosinophils are capable of inhibiting bacterial growth through the release of cytotoxic granules (Yii *et al*., 2018). However, this potential protective role is qualified by studies suggesting that eosinophilia may also contribute to chronic inflammation and tissue remodeling, which could indirectly compromise the clearance of pulmonary infections (Gauthier *et al*., 2025). This paradox highlights the complexity of immunological interactions and the need for further investigation of specific asthma phenotypes and their impact on the pathogenesis of tuberculosis (Gyawali *et al*., 2023).

Shared Risk Factors

1. Environmental and Lifestyle Factors

Risk factors common to both asthma and tuberculosis include exposure to air pollution, smoking, and genetic predispositions, which can collectively compromise the integrity of the respiratory epithelial barrier and modulate innate and adaptive immune responses (Fang *et al*., 2020; Wang *et al*., 2020). Smoking is a major risk factor for bronchial obstruction and active tuberculosis, as it impairs pulmonary clearance and weakens alveolar macrophages (Hamada *et al*., 2021). Exposure to indoor pollution from fossil fuels or biomass also increases the risk of TB and asthma by creating an irritating and immunosuppressive environment (World Health Organization, 2025). Furthermore, densely populated environments and precarious socioeconomic conditions increase the likelihood of exposure to infectious agents such as Mycobacterium tuberculosis, while malnutrition can further weaken the immune system, making individuals more vulnerable to both diseases (Bastos *et al*., 2018).

2. Socioeconomic Factors

Adverse socioeconomic conditions, increased physical and mental stress, and inadequate nutrition are also shared risk factors that can exacerbate susceptibility

to both diseases (Garg & Karahyla, 2017). Poverty, overcrowding, and low income contribute to the transmission of TB and exposure to indoor allergens, increasing the prevalence of respiratory diseases (Lee *et al*., 2013; Tiwari, 2018). Furthermore, the lack of access to healthcare and adequate education among these populations exacerbates the difficulty of diagnosing and effectively managing asthma and tuberculosis, thereby contributing to increased morbidity (Gupta *et al*., 2023).

3. Medical and Nutritional Comorbidities

Diabetes is a key risk factor for TB and complicates asthma control by promoting systemic inflammation (Chung *et al.*, 2014; World Health Organization, 2025). Malnutrition weakens cell-mediated immunity, increasing the risk of active TB and impairing the repair of lung tissue damaged by asthma (World Health Organization, 2025). Obesity, for example, is a comorbidity that affects both asthma and tuberculosis, as it impairs the immune response and can increase the risk of disease as well as affect the effectiveness of treatment (Herman *et al*., 2024). The presence of other comorbidities can also affect the clinical intensity or severity of asthma, potentially influencing its interaction with tuberculosis (Cazzola *et al*., 2022).

5. Demographic Factors

Age and sex influence disease prevalence: adult men have a higher risk of TB, while severe asthma is common among older adults, who have reduced immunity and multiple comorbidities (Lee *et al*., 2013).

Impact of Asthma on the Clinical Progression of Tuberculosis

Atypical Clinical Presentation

In patients with co-infection of asthma and tuberculosis, the type 2 inflammation characteristic of asthma may influence the immune response to *Mycobacterium tuberculosis,* potentially leading to clinical manifestations that are less typical of pulmonary tuberculosis, such as a less specific radiological or symptomatic presentation (Yii *et al*., 2018). This immunological influence can lead to a more insidious spread of the infection or delays in diagnosis, due to possible confusion with asthma exacerbations or other respiratory conditions. Furthermore, asthma as a comorbid condition can complicate the interpretation of respiratory symptoms, sometimes masking the early signs of tuberculosis and thereby delaying the initiation of appropriate antituberculosis treatment (Fekih *et al*., 2010). This situation may also be exacerbated by preexisting lung damage, potentially due to long-standing asthma, which could mask the classic radiological manifestations of tuberculosis (Garg & Karahyla, 2017).

Disease Progression and Complications

As mentioned above, available studies suggest that asthma promotes the reactivation of tuberculosis (TB), primarily in connection with the use of corticosteroids (Lee *et al*., 2013). Inhaled corticosteroids (ICS) alter local pulmonary immunity, reducing the activity of macrophages and T cells, and increase the risk of TB in a dose-dependent manner, particularly at doses exceeding 1,000 µg/day of fluticasone equivalent (Brassard *et al*., 2010; Chung *et al*., 2014; Lee *et al*., 2013). The combination of CSIs with systemic corticosteroids increases this risk and raises the likelihood of progression to active TB (Chung *et al*., 2014).

The coexistence of asthma and TB complicates therapeutic management. Rifampin reduces the bioavailability of corticosteroids, leading to a loss of asthma control, while isoniazid can worsen bronchoconstriction and increase the toxicity of theophylline (Fekih *et al*., 2010). Severe asthma exacerbations have thus been reported following the initiation of antituberculosis treatment (Fekih *et al*., 2010).

In the long term, this comorbidity is associated with a poor prognosis, including an increased risk of lung cancer (Jian *et al*., 2016) and higher rates of treatment failure, particularly in drug-resistant forms of TB (Alemu *et al*., 2020). Although a potential protective role for eosinophilic inflammation has been suggested, its benefits appear to be largely outweighed by the deleterious effects of corticosteroid-induced immunosuppression (Yii *et al*., 2018).

Therapeutic Considerations in Patients with Co-Infections

Potential Drug Interactions

The concurrent management of asthma and tuberculosis (TB) is complicated by significant drug interactions, primarily involving antituberculosis drugs (Fekih *et al*., 2010; Gharsalli *et al.*, 2014). Rifampin, a potent hepatic enzyme inducer, significantly reduces the bioavailability and efficacy of oral corticosteroids, compromising asthma control, particularly in corticosteroid-dependent patients (Fekih *et al.*, 2010). A direct pharmacodynamic interaction with glucocorticoid receptors has also been described (Fekih *et al*., 2010). Theophylline is subject to significant fluctuations in concentration: rifampin reduces its efficacy, while isoniazid increases its plasma levels, posing a risk of toxicity and requiring close monitoring (Fekih *et al*., 2010). Isoniazid may also exacerbate bronchoconstriction or induce hypersensitivity reactions (Fekih *et al*., 2010). Rigorous clinical monitoring remains essential, however (Gharsalli *et al*., 2014).

Optimization of Antituberculosis Treatment

Optimizing tuberculosis treatment in patients with asthma relies on rigorous management of drug interactions and the integration of therapeutic strategies aimed at maintaining control of both conditions (Fekih *et al*., 2010). Rifampin, a key component of

antituberculosis regimens, complicates the management of asthma due to its potent enzyme-inducing effect, which significantly reduces the bioavailability of oral corticosteroids (Fekih *et al.*, 2010). To optimize treatment, it is often necessary to increase the prednisolone dosage in corticosteroid-dependent asthmatics to prevent a serious deterioration in respiratory control (Fekih *et al*., 2010). Furthermore, isoniazid can inhibit the metabolism of theophylline, leading to an increased risk of toxicity, which warrants close monitoring of theophylline plasma concentrations (Fekih *et al*., 2010).

Addressing modifiable risk factors, particularly smoking, is a key strategy for improving tuberculosis cure rates (Chiang *et al*., 2011). Finally, the use of inhaled corticosteroids at the minimum effective dose and enhanced clinical monitoring are recommended, particularly in patients with preexisting pulmonary complications (Venkitakrishnan *et al*., 2021).

Asthma Management in Patients with Tuberculosis

The management of asthma in patients with tuberculosis requires an individualized approach, with particular attention to selecting treatments that minimize drug interactions and the risk of exacerbation of either condition (Fekih *et al*., 2010). It is crucial to consider adjusting doses of inhaled and systemic corticosteroids to maintain asthma control while avoiding excessive immunosuppression that could promote the progression of tuberculosis (Tiwari, 2018). The choice of bronchodilators must also be carefully evaluated, taking into account potential interactions and side effects, while prioritizing an educational approach for the patient regarding treatment adherence and the recognition of signs of worsening symptoms (Miget, 2017). Strategies to minimize the impact of drug interactions include adjusting the doses of oral corticosteroids or using alternative therapies when possible (Zaidi *et al*., 2023).

DISCUSSION

Summary of the Evidence

Overall, the literature highlights a complex, multifactorial, and sometimes bidirectional interaction between asthma and tuberculosis. From an epidemiological perspective, these two diseases represent major global health burdens and frequently coexist in similar socioeconomic and environmental contexts (Gyawali *et al*., 2023; World Health Organization, 2025). Although the asthma–tuberculosis comorbidity remains underdocumented, several observational studies suggest a significant association (Adchitre & Adchitre, 2020; Bogomolov & Зайков, 2020).

The majority of the data suggest that asthma plays a facilitating role in the onset and progression of tuberculosis, primarily mediated by the use of inhaled or systemic corticosteroids (Castellana *et al*., 2019; Fang *et al*., 2020; Lee *et al*., 2013). These treatments, although essential for controlling asthma, appear to alter local and systemic pulmonary immunity in a dose- and duration-dependent manner, increasing susceptibility to tuberculosis infection, promoting the reactivation of latent tuberculosis, and complicating the clinical course of the disease (Brassard *et al*., 2010; Lee *et al*., 2013). This risk sometimes persists for several years after treatment is discontinued, highlighting the prolonged impact of induced immunosuppression (Lee *et al*., 2018).

From a pathophysiological perspective, the immune dysregulation associated with asthma appears to play a central role in this interaction. The imbalance between Th1 and Th2 responses, characteristic of asthma, could compromise the protective Th1 response necessary for controlling *Mycobacterium tuberculosis* (Hamada *et al*., 2021; Lee *et al*., 2025; Tarancón *et al*., 2021; Yii *et al*., 2018) . This alteration is exacerbated by the action of corticosteroids and by shared genetic factors, particularly polymorphisms in the IL10 gene, which may simultaneously modulate susceptibility to asthma and tuberculosis depending on the population context (Fekih *et al*., 2010; Lee *et al*., 2013; Tiwari, 2018; Yu *et al*., 2016).

However, the literature also highlights conflicting findings. One study suggests a potential protective effect of certain asthma phenotypes, particularly those characterized by eosinophilic inflammation, which may exert antimycobacterial activity (Yii *et al*., 2018). These conflicting results reported in the literature could be explained by several factors. Genetic variations may have distinct functional effects depending on the populations studied, due to ethnic, environmental, and immunological differences. Furthermore, asthma represents a heterogeneous set of clinical and immunological phenotypes, whose impact on the response to *Mycobacterium tuberculosis* may vary. While certain forms of atopic asthma, associated with increased IL-10 production, may impair the protective Th1 response, other phenotypes characterized by eosinophilic inflammation may exert a potentially protective immunomodulatory effect. Finally, methodological differences between studies—particularly regarding case definitions and control for confounding factors—may also contribute to these conflicting observations.

Finally, the available data suggest that the coexistence of asthma and tuberculosis significantly complicates clinical management, due to sometimes atypical clinical presentations, an increased risk of complications, and major drug interactions between antituberculosis treatments and asthma therapies (Fekih *et al*., 2010; Gharsalli *et al*., 2014). Environmental and socioeconomic factors, as well as shared metabolic comorbidities , appear to be cross-cutting determinants that reinforce this interaction (Fekih *et al*., 2010; Gharsalli *et al*., 2014).

Overall, the literature suggests that asthma most often acts as a risk factor for tuberculosis, although certain immunological mechanisms may, in specific contexts, modulate this risk in a protective manner. This heterogeneity underscores the need for integrative approaches that take into account immunological, genetic, therapeutic, and contextual factors to better understand the interactions between these two conditions.

Limitations of the Current Literature

The current literature on the association between asthma and tuberculosis (TB) has several significant limitations. Most of the data are based on observational studies, which are subject to confounding factors and reverse causality, with no clear distinction between the intrinsic effect of asthma and that of corticosteroid treatments; furthermore, few randomized clinical trials have been conducted to evaluate specific management strategies. Data from regions with a high TB burden, as well as pediatric studies, remain limited or virtually nonexistent. Furthermore, the immunological and genetic mechanisms have not been sufficiently explored, contributing to sometimes contradictory results. Finally, the long-term effects of modern asthma biologics on TB risk remain largely unknown.

Implications for Clinical Practice and Future Research

Future research should therefore focus on elucidating these underlying mechanisms, developing treatment guidelines based on more robust evidence, and assessing the impact of new asthma therapies on susceptibility to tuberculosis (Garg & Karahyla, 2017). It is imperative to conduct robust prospective studies, including diverse populations and standardized diagnostic criteria for asthma and tuberculosis, in order to better understand this complex relationship (Yii *et al*., 2018). Particular attention must be paid to the impact of socioeconomic factors on access to care and the diagnosis of both conditions, as these determinants may mask or exacerbate the observed associations (Yii *et al*., 2018).

CONCLUSION

This narrative review highlights the complex interactions between asthma and tuberculosis. The available data suggest that asthma may be associated with an increased risk of tuberculosis, may promote the progression of latent tuberculosis infection to active tuberculosis, may contribute to the reactivation of *Mycobacterium tuberculosis*, and may influence disease severity and the occurrence of complications. These associations appear to result from multiple mechanisms, including the use of immunosuppressive therapies, immune dysregulation characterized by an imbalance between Th1 and Th2 responses, and shared risk factors.

However, the current state of knowledge remains limited, heterogeneous, and primarily based on observational studies, which restricts the scope of conclusions, particularly in settings with a high tuberculosis burden. The lack of longitudinal and interventional studies constitutes a major limitation to establishing robust causal relationships.

Despite these limitations, a better understanding of the underlying immunological mechanisms and potential drug interactions between asthma and tuberculosis could contribute to improved clinical management and guide the development of appropriate preventive and therapeutic strategies. Future research based on multidisciplinary approaches and well-designed longitudinal studies is needed to clarify these interactions and strengthen the scientific evidence base.

DISCLOSURES

Jenni AI, Grammarly, and ChatGPT were used to assist with the drafting and stylistic refinement of the manuscript. The scientific analysis, data interpretation, and conclusions are the sole responsibility of the authors.

Ethical Considerations

As this study is a narrative review based exclusively on the analysis of previously published data, with no human participants or personally identifiable information involved, no ethics committee approval was required.

Conflict of Interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Authors' Contributions

C.M.L. contributed to the conceptualization of the study, the development of the methodology, the literature search, the selection and analysis of the literature, and the drafting of the initial version of the manuscript, and provided scientific and methodological supervision of the work. N.M.Z. and F.M.M. contributed to the literature search, the interpretation of data from the literature, the critical review of the scientific content, and the editing of the manuscript. All authors read, reviewed, and approved the final version of the manuscript.

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