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TUBERCULOSIS EXPOSURE AND CHILDHOOD GROWTH FALTERING: A SYSTEMATIC REVIEW OF STUNTING AND UNDERNUTRITION OUTCOMES IN CHILDREN

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ABSTRACT
Tuberculosis (TB) and childhood undernutrition remain major public health challenges, particularly in high-burden countries. Although undernutrition is widely recognized as a risk factor for TB, less attention has been given to the potential impact of TB exposure, infection, and disease on child growth. Children living in households affected by TB may experience biological, nutritional, and socioeconomic vulnerabilities that contribute to growth faltering and stunting. This systematic review aimed to synthesize available evidence on the association between TB exposure and impaired growth and nutritional outcomes among children. A systematic review will be conducted following the PRISMA 2020 guidelines. Relevant studies published from database inception up to November 2026 will be identified through electronic databases including PubMed, Scopus, Web of Science, and ProQuest. Eligible studies will include children and adolescents aged 0–18 years with household TB exposure, TB infection, or active TB disease. The primary outcome will be stunting, defined primarily as height-for-age Z-score below −2 standard deviations. Secondary outcomes will include wasting, underweight, growth faltering, height-for-age, weight-for-age, weight-for-height or BMI-for-age, and other anthropometric indicators. Study quality will be assessed using appropriate critical appraisal tools, and findings will be synthesized narratively and, where sufficient homogeneous data are available, quantitatively. Existing evidence suggests that children exposed to or affected by TB may have a greater burden of stunting, underweight, wasting, and impaired linear growth. Household TB exposure may contribute to childhood nutritional vulnerability through recurrent infection, chronic inflammation, reduced dietary intake, household food insecurity, and increased socioeconomic burden. However, the magnitude and consistency of these associations remain incompletely synthesized. This systematic review is expected to clarify the relationship between TB exposure and childhood growth impairment and identify important gaps in current evidence. The findings may support the integration of nutritional assessment and growth monitoring into pediatric TB contact investigation and TB prevention programs, particularly in high-burden settings.

Keywords: children; growth faltering; household contact; stunting; systematic review; tuberculosis; undernutrition

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INTRODUCTION

Tuberculosis (TB) and undernutrition are closely interconnected through a complex bidirectional relationship. Undernutrition can impair innate and adaptive immune responses, increasing susceptibility to Mycobacterium tuberculosis infection and progression to active TB disease. A 2024 Cochrane systematic review reported that undernutrition probably increases the short-term risk of developing TB disease approximately two-fold (Franco *et al.*, 2024)), while the World Health Organization recognizes undernutrition as a major modifiable determinant of TB incidence and mortality (World Health Organization, 2024). Conversely, active TB may exacerbate undernutrition through chronic inflammation, anorexia, increased metabolic demands, altered nutrient utilization, and catabolism, resulting in weight loss, muscle wasting, and micronutrient deficiencies. In children, these processes may interfere with expected weight gain and linear growth, potentially contributing to growth faltering and stunting (Tugra Ozer, 2024). Beyond these

biological mechanisms, TB may also affect childhood nutrition through household-level socioeconomic pathways, including income loss, healthcare expenditure, food insecurity, and disruption of caregiving and feeding practices. These vulnerabilities may extend to children living in TB-affected households even when they do not develop active TB disease, supporting the concept of a TB–undernutrition cycle in which biological and socioeconomic mechanisms reinforce one another.

Despite substantial evidence demonstrating undernutrition as a risk factor for TB, comparatively less attention has been directed toward the reverse pathway—whether TB exposure, infection, or active disease contributes to impaired linear growth and childhood stunting. This distinction is important because stunting reflects cumulative impairment of linear growth rather than acute weight loss and may therefore represent a longer-term nutritional consequence of TB-related biological and household vulnerabilities. Children with active TB and those living in TB-affected households may be particularly vulnerable to growth impairment through prolonged inflammation, inadequate dietary intake, food insecurity, and disrupted caregiving. However, evidence specifically examining stunting and height-for-age outcomes in relation to childhood TB exposure or disease remains insufficiently synthesized. Therefore, systematically evaluating this relationship is important to clarify the potential contribution of TB to childhood growth impairment and to inform the integration of growth monitoring and nutritional assessment into pediatric TB care and household contact investigation.

Tuberculosis (TB) and undernutrition are closely interconnected through a complex bidirectional relationship. Undernutrition can impair innate and adaptive immune responses, increasing susceptibility to *Mycobacterium tuberculosis* infection and progression to active TB disease. A 2024 Cochrane systematic review reported that undernutrition probably increases the short-term risk of developing TB disease approximately two-fold (Franco *et al.*, 2024), while the World Health Organization recognizes undernutrition as a major modifiable determinant of TB incidence and mortality (World Health Organization, 2024). Conversely, active TB may exacerbate undernutrition through chronic inflammation, anorexia, increased metabolic demands, altered nutrient utilization, and catabolism, resulting in weight loss, muscle wasting, and micronutrient deficiencies. In children, these processes may interfere with expected weight gain and linear growth, potentially contributing to growth faltering and stunting (Tugra Ozer, 2024). Beyond these biological mechanisms, TB may also affect childhood nutrition through household-level socioeconomic pathways, including income loss, healthcare expenditure, food insecurity, and disruption of caregiving and feeding practices. These vulnerabilities may extend to children living in TB-affected households even when they do not develop active TB disease, supporting the concept of a TB–undernutrition cycle in which biological and socioeconomic mechanisms reinforce one another.

Despite substantial evidence demonstrating undernutrition as a risk factor for TB, comparatively less attention has been directed toward the reverse pathway whether TB exposure, infection, or active disease contributes to impaired linear growth and childhood stunting. This distinction is important because stunting reflects cumulative impairment of linear growth rather than acute weight loss and may therefore represent a longer-term nutritional consequence of TB-related biological and household vulnerabilities. Children with active TB and those living in TB-affected households may be particularly vulnerable to growth impairment through prolonged inflammation, inadequate dietary intake, food insecurity, and disrupted caregiving. However, evidence specifically examining stunting and height-for-age outcomes in relation to childhood TB exposure or disease remains insufficiently synthesized. Therefore, systematically evaluating this relationship is important to clarify the potential contribution of TB to childhood growth impairment and to inform the integration of growth monitoring and nutritional assessment into pediatric TB care and household contact investigation.

Household contact is a critical context for tuberculosis (TB) transmission in children, as close and prolonged exposure to a person with pulmonary TB substantially increases the risk of Mycobacterium tuberculosis infection and subsequent disease, particularly among children under five years of age (Martinez, 2016); (World Health Organization, 2024). However, household TB exposure may represent not only an infectious risk but also a broader socioeconomic and nutritional vulnerability, as TB-related healthcare costs, income loss, and work disruption can reduce household resources for food, dietary diversity, and childcare (Organization, 2021). Illness of a parent or primary caregiver may further disrupt feeding practices, healthcare-seeking, and growth monitoring, potentially compromising child nutrition and development. Evidence from household-contact studies, including the RATIONS trial, further demonstrates the close interaction between nutritional status and TB outcomes, with nutritional supplementation associated with a reduced incidence of TB disease among household contacts (Bhargava *et al.*, 2023). Thus, household TB exposure may affect children through interconnected biological and socioeconomic pathways, involving increased infectious exposure, TB infection or disease, food insecurity, inadequate dietary intake, and disrupted caregiving, which may contribute to nutritional vulnerability and impaired linear growth. If these conditions persist during critical periods of childhood development, they may contribute to growth faltering and potentially stunting. Nevertheless, while the relationships between household TB exposure and TB infection and between undernutrition and TB progression are well established, the pathway linking household TB exposure specifically to childhood growth faltering and stunting remains insufficiently synthesized, representing an important research gap for systematic investigation.

The relationship between tuberculosis (TB) and malnutrition has predominantly been examined from the perspective of undernutrition as a risk factor for TB. Systematic evidence indicates that undernutrition may approximately double the risk of developing TB disease, primarily through impaired immune responses that increase susceptibility to progression from TB infection to active disease (World Health Organization, 2024). Similarly, studies of children living in households with individuals affected by TB have largely focused on the risk of TB infection and disease progression. Children with household TB exposure have been reported to have a 3.79-fold higher likelihood of infection than unexposed children, with a greater risk among younger age groups (Martinez, 2016). Furthermore, a meta-analysis involving 137,647 TB-exposed children demonstrated a substantial risk of developing TB disease following close exposure, particularly among children aged <5 years (Martinez et al., 2020). However, compared with these well-established research directions, evidence regarding the effects of TB exposure, infection, or disease on childhood growth and nutritional status remains fragmented and has not been comprehensively synthesized. Although several studies have reported associations between TB and stunting in children, much of the existing literature continues to conceptualize malnutrition as a condition preceding TB (Haerana et al., 2021). Therefore, a shift in perspective is warranted from malnutrition as a risk factor for TB toward TB exposure as a potential determinant of childhood growth impairment, particularly to elucidate the potential development of growth faltering, stunting, and undernutrition among children exposed to or affected by TB.

Previous research on tuberculosis (TB) and childhood nutrition has predominantly focused on undernutrition as a risk factor for TB and household TB exposure as a determinant of TB infection and disease, with systematic evidence showing that undernutrition approximately doubles the risk of developing TB (Franco et al., 2024) and that household-exposed children have a substantially higher risk of TB infection and disease, particularly those younger than five years (Martinez et al., 2017; Martinez et al., 2020). In contrast, considerably less attention has been directed toward the reverse nutritional pathway, namely whether TB exposure, infection, or active disease adversely affects childhood growth and nutritional status. Although individual studies have reported stunting and other forms of undernutrition among TB-exposed children, this evidence remains fragmented,

largely observational, and has often conceptualized malnutrition as an antecedent rather than a potential consequence of TB exposure (Haerana et al., 2021). Thus, an important knowledge gap remains regarding whether children exposed to or affected by TB experience a greater burden of stunting, growth faltering, wasting, underweight, or other anthropometric impairments, and whether these outcomes vary according to exposure type, age, disease status, or socioeconomic context. The novelty of this systematic review therefore lies in shifting the perspective from malnutrition as a risk factor for TB-to-TB exposure as a potential determinant of childhood growth impairment, while recognizing the potentially bidirectional nature of the TB–undernutrition relationship. Accordingly, this systematic review aims to synthesize the available evidence on whether TB exposure, infection, or disease adversely affects childhood growth and nutritional status, with particular emphasis on stunting and impaired linear growth, thereby informing the potential integration of nutritional assessment and longitudinal growth monitoring into paediatric TB contact investigation, prevention, and follow-up programs.

METHOD

A systematic literature search was conducted using PubMed/MEDLINE and Scopus to identify relevant studies on pulmonary tuberculosis, nutrition, and growth among children. The search was performed using the predefined keywords "tuberculosis pulmonary AND children AND nutrition AND growth." The same core search terms were applied across both databases, with adjustments to the search format where required by each database. All retrieved records were exported and combined, and duplicate records were removed before screening. Titles and abstracts were subsequently screened, followed by full-text assessment according to the predefined eligibility criteria. The study identification, screening, eligibility assessment, and inclusion process was documented using the PRISMA 2020 flow diagram.

Search Strategy

The research question and search strategy for this systematic review were developed using the PECO (Population, Exposure, Comparison, and Outcome) framework, which is appropriate for evaluating exposure–outcome relationships in observational studies. The Population (P) comprised children and adolescents aged 0–18 years. The Exposure (E) included household or close contact with a person with tuberculosis (TB), Mycobacterium tuberculosis infection, or active TB disease. The Comparison (C) consisted of children without the corresponding TB exposure, infection, or disease, where a comparison group was available. The primary Outcome (O) was stunting or impaired linear growth, particularly when defined using height-for-age or length-for-age indicators, including a height-for-age Z-score (HAZ) or length-for-age Z-score (LAZ) below −2 standard deviations. Secondary outcomes included other indicators of nutritional and growth status, such as growth faltering, underweight, wasting, HAZ/LAZ, weight-for-age Z-score (WAZ), weight-for-height Z-score (WHZ), body mass index-for-age Z-score (BAZ), mid-upper arm circumference (MUAC), and other relevant anthropometric measures.

Tabel 1.
PECO Component

PECO component	Definition in this systematic review
P – Population	Children and adolescents aged 0–18 years
E – Exposure	Household/close TB exposure, *M. tuberculosis* infection, or active TB disease
C – Comparison	Children without the corresponding TB exposure, infection, or disease, where available
O – Primary outcome	Stunting/impaired linear growth, particularly HAZ or LAZ < −2 SD
O – Secondary outcomes	Growth faltering, underweight, wasting, HAZ/LAZ, WAZ, WHZ, BAZ, MUAC, and other anthropometric growth indicators

Inclusion Criteria

Studies were considered eligible for inclusion if they involved children and adolescents aged 0–18 years and examined exposure to tuberculosis (TB), including household or close TB exposure,

Mycobacterium tuberculosis infection, or active TB disease. Eligible studies were required to report at least one nutritional or growth-related outcome, including stunting, impaired linear growth, growth faltering, underweight, wasting, or relevant anthropometric indicators such as height-for-age, length-for-age, weight-for-age, weight-for-height, body mass index-for-age, or mid-upper arm circumference. The review primarily included observational studies, comprising cohort, case-control, and cross-sectional designs. Intervention studies were also considered eligible when they provided relevant baseline or comparative data that allowed assessment of the relationship between TB exposure, infection, or disease and childhood nutritional or growth outcomes. Only full-text scientific publications published from database inception up to November 2026 containing sufficient data for eligibility assessment and evidence synthesis were included. Studies published in English, as well as publications in other languages that could be reliably translated and adequately assessed, were considered eligible for inclusion.

Exclusion Criteria

Studies were excluded if they involved adult populations only or included mixed populations of children and adults without providing separately extractable pediatric data. Studies were also excluded if they did not report any relevant anthropometric, nutritional, or growth-related outcomes, or if they lacked clear information regarding TB exposure, *Mycobacterium tuberculosis* infection, or active TB disease. Publications with study designs that did not provide sufficient analytical evidence for the review question were excluded, including case reports, very small case series, editorials, commentaries, and narrative reviews**.** Systematic reviews and meta-analyses were not included as primary studies in the evidence synthesis, although their reference lists could be examined to identify potentially eligible original studies. Conference abstracts were excluded when they did not provide sufficient methodological information or outcome data for eligibility assessment and data extraction.

Study Selection

All records identified through the PubMed/MEDLINE and Scopus database searches were collected and combined before the screening process. Duplicate records retrieved from both databases were identified and removed prior to eligibility screening. The remaining records underwent title and abstract screening to exclude studies that were clearly unrelated to the review question or did not meet the predefined inclusion criteria. Articles considered potentially relevant were subsequently retrieved for full-text assessment, during which eligibility was evaluated according to the predefined population, TB exposure, nutritional or growth outcomes, and study design criteria. Full-text articles that did not meet the eligibility criteria were excluded, and the specific reasons for exclusion were documented. Studies fulfilling all eligibility criteria were included in the final systematic review and proceeded to data extraction and evidence synthesis. The entire study selection process, including the numbers of records identified, duplicates removed, records screened, records excluded, full-text reports assessed, full-text reports excluded with reasons, and studies finally included, was documented using a PRISMA 2020 flow diagram

Data Extraction

Data were systematically extracted from all studies that met the eligibility criteria using a standardized data extraction form. The extracted information included author(s), year of publication, country and study setting, study design, sample size, participants' age range, and population characteristics. Information related to tuberculosis included the type of TB exposure or status, categorized as household or close TB exposure, Mycobacterium tuberculosis infection, or active TB disease, together with the methods or criteria used to define TB exposure, infection, or disease. Nutritional and growth-related data included stunting, height-for-age Z-score (HAZ), length-for-age Z-score (LAZ), linear growth or growth faltering, underweight, weight-for-age Z-score (WAZ), wasting, weight-for-height Z-score (WHZ), body mass index-for-age Z-score (BAZ), mid-upper arm circumference (MUAC), and other relevant anthropometric indicators. Where

available, information on the definition and cut-off values used for nutritional outcomes, timing of anthropometric assessment, comparison groups, prevalence or mean anthropometric values, and measures of association such as odds ratios (ORs), risk ratios (RRs), hazard ratios (HRs), regression coefficients, and corresponding 95% confidence intervals (CIs) was extracted. Information on potential confounding variables and adjusted estimates was also recorded when reported. These data were subsequently organized to compare growth and nutritional outcomes according to TB exposure, infection, or disease status and to support the qualitative and, where appropriate, quantitative synthesis of the evidence.

Quality / Risk of Bias

The methodological quality and risk of bias of the studies included in the final evidence synthesis were assessed using the Joanna Briggs Institute (JBI) Critical Appraisal Tools. The appropriate JBI checklist was selected according to the methodological design of each study. Each appraisal item was rated as "Yes," "No," "Unclear," or "Not applicable." The assessment focused on study-specific methodological domains, including participant selection, measurement of exposure and outcomes, identification and management of potential confounding factors where applicable, completeness of follow-up, and appropriateness of statistical analysis. Risk-of-bias assessment was conducted at the individual-study level, and the results were not combined into a single numerical quality score across studies because the number and nature of JBI appraisal items differ according to study design. Studies retained solely as supporting or contextual references were not included in the primary risk-of-bias assessment.

Table 2.
Methodological Design, Eligibility, and Appraisal Status of Studies Assessed During Full-Text Review

No.	Study	Study Design	JBI Checklist	Yes	No	Unclear	Overall Appraisal
1	Malik *et al.*, 2020	Narrative review / review article	Not applicable	-	-	-	Not appraised; narrative review
2	Visser *et al.*, 2018	Overview of systematic reviews (Cochrane Review)	JBI Systematic Reviews and Research Syntheses*	-	-	-	Not included in primary-study RoB assessment
3	Gou *et al.*, 2018	Systematic review & meta-analysis	JBI Systematic Reviews and Research Syntheses*	-	-	-	Not included in primary-study RoB assessment
4	Harausz *et al.*, 2018	Systematic review + individual patient data (IPD) meta-analysis	JBI Systematic Reviews and Research Syntheses*	-	-	-	Not included in primary-study RoB assessment
5	Marangu and Zar, 2019	Narrative/review article of published literature (2013–2019)	Not applicable	-	-	-	Not appraised; narrative review
6	Duke *et al.*, 2017	Clinical review / Global Child Health article	Not applicable	-	-	-	Not appraised; clinical review
7	Sudfeld *et al.*, 2017	Study protocol – randomized, double-blind, placebo-controlled trial	Not applicable to outcome-level RoB assessment	-	-	-	Not appraised; study protocol without outcome data
8	Yakoob *et al.*, 2016	Cochrane systematic review of RCTs / Intervention Review	JBI Systematic Reviews and Research Syntheses*	-	-	-	-
9	Damanik *et al.*, 2026	Scoping review (PRISMA-ScR)	Not included in primary-study JBI appraisal				Supporting review; not appraised in primary-study RoB assessment

No.	Study	Study Design	JBI Checklist	Yes	No	Unclear	Overall Appraisal
10	Arolkar, Damle and Gala, 2022	Narrative/clinical review	Not applicable	-	-	-	Not appraised; narrative/clinical review
11	Reddy *et al.*, 2021	Community-based observational household-contact study / secondary analysis of RePORT cohort	JBI Analytical Cross-Sectional†	-	-	-	Not included in final core evidence based on eligibility assessment
12	Tezol *et al.*, 2019	Retrospective case series	JBI Case Series	8	0	2	Acceptable; some concerns

Notes: JBI = Joanna Briggs Institute; RoB = risk of bias; RCT = randomized controlled trial; IPD = individual patient data; "–" = not assessed/not applicable.Y = Yes; N = No; U = Unclear; NA = Not applicable

Table 2 presents the methodological appraisal of the studies assessed during the eligibility process. Risk of bias was evaluated using the Joanna Briggs Institute (JBI) Critical Appraisal Tool appropriate to each study design. Reviews, study protocols, and other publications retained only as supporting or contextual evidence were not included in the primary-study risk-of-bias assessment and therefore were not assigned numerical JBI ratings. Tezol et al. (2019) was appraised using the JBI Critical Appraisal Checklist for Case Series and met eight of ten criteria, with two items rated as unclear and none as no. The main concerns related to unclear consecutive and complete inclusion of eligible cases. Overall, the study was considered methodologically acceptable, with some concerns regarding participant selection**.**

Statistical Analysis

The characteristics and findings of the included studies were initially summarized using descriptive and narrative synthesis. Study characteristics, including population, type of tuberculosis (TB) exposure, study design, nutritional and growth outcomes, and measures of association, were presented in summary tables. The primary outcome of interest was stunting or impaired linear growth, particularly when assessed using height-for-age or length-for-age indicators, while secondary outcomes included underweight, wasting, growth faltering, and other relevant anthropometric measures.

Where two or more studies were sufficiently comparable in terms of population, TB exposure, outcome definition, and study design, quantitative synthesis through meta-analysis was considered. For dichotomous outcomes, such as stunting, wasting, and underweight, effect estimates were expressed as odds ratios (ORs), risk ratios (RRs), or other reported measures of association with 95% confidence intervals (CIs)**.** Where appropriate and methodologically justified, comparable effect estimates were pooled. For continuous outcomes, such as height-for-age Z-score (HAZ), weight-for-age Z-score (WAZ), or weight-for-height Z-score (WHZ), mean differences (MDs) or standardized mean differences (SMDs) with 95% CIs were considered, depending on the consistency of outcome measurement across studies.

Given the anticipated clinical and methodological heterogeneity across studies, a random-effects model was considered appropriate when meta-analysis was feasible. Statistical heterogeneity was assessed using the I^2 statistic and, where applicable, Cochran's Q test. Potential sources of heterogeneity were explored according to factors such as type of TB exposure (household TB exposure, TB infection, or active TB disease), age group, nutritional outcome, study design, and study setting**,** where sufficient data were available. When quantitative pooling was not appropriate because of substantial heterogeneity, incompatible outcome definitions, or insufficient comparable studies, findings were synthesized narratively according to TB exposure and growth or nutritional outcome.

RESULT

Study Selection

All records identified through the PubMed/MEDLINE and Scopus database searches were collected and combined before the screening process. The identified records were screened sequentially according to the predefined eligibility criteria. The initial screening was conducted based on titles and their relevance to the review topic, followed by a more detailed assessment of titles and abstracts. Potentially eligible articles were subsequently retrieved and assessed in full text. During the full-text assessment, eligibility was determined based on the predefined criteria, including the pediatric population, TB exposure, infection or disease, relevant nutritional or growth outcomes, and study design. Studies that did not meet the eligibility criteria were excluded, and the reasons for exclusion at the full-text stage were documented. Studies meeting all eligibility criteria were included in the final systematic review. The study identification, screening, eligibility assessment, and inclusion process was documented using the PRISMA 2020 flow diagram.

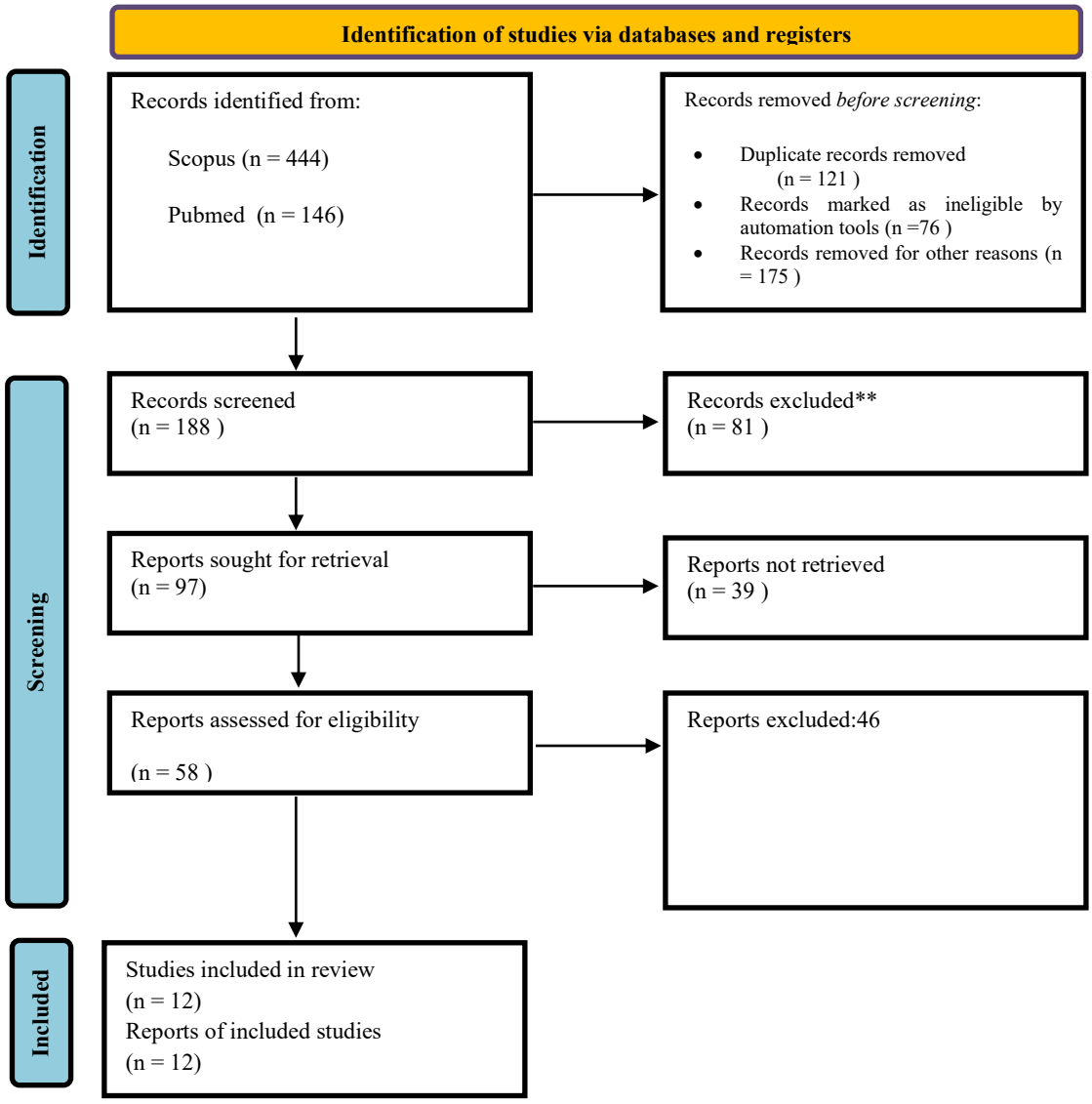


Figure 1. Preferred Reporting Items for Systematic Reviews and Meta-Analyses

Table 3.
Eligibility Assessment and Relevance of Full-Text Articles to the TB–Stunting Relationship

No.	Author, Year, Title	Study Design / Article Type	Population / Focus	Nutritional / Stunting Indicators	Main Relevant Findings	Relevance to TB–Stunting	Final Decision
1	Malik et al., 2020. *Non-nutritional anemia:*	Narrative review	Anemia and its various causes, including TB	Nutritional status discussed; no direct	TB was discussed in relation to anemia and	Indirect	Supporting / Excluded From Core Evidence

No.	Author, Year, Title	Study Design / Article Type	Population / Focus	Nutritional / Stunting Indicators	Main Relevant Findings	Relevance to TB–Stunting	Final Decision
	Malaria, thalassemia, G6PD deficiency and tuberculosis in Indonesia			measurement of HAZ or stunting	nutritional conditions, but no direct analysis of the TB–stunting relationship was reported		
2	Visser et al., 2018. *Community-based supplementary feeding for food insecure, vulnerable and malnourished populations – an overview of systematic reviews*	Overview of systematic reviews	Food-insecure, vulnerable, and malnourished populations, including individuals with TB	Weight, height, and other growth indicators	Supplementary feeding in people with TB was associated with improvements in body weight, whereas evidence for height or HAZ improvement was inconsistent	Indirect	Supporting / Excluded From Core Evidence
3	Gou et al., 2018. *The association between vitamin D status and tuberculosis in children: A meta-analysis*	Systematic review and meta-analysis	Children with TB/LTBI and controls	Vitamin D status; no direct anthropometric stunting indicator	Vitamin D deficiency was associated with pediatric TB, with increased odds of TB among children with vitamin D deficiency	Indirect; stunting not assessed	Supporting / Excluded From Core Evidence
4	Harausz et al., 2018. *Treatment and outcomes in children with multidrug-resistant tuberculosis: A systematic review and individual patient data meta-analysis*	Systematic review and individual patient data (IPD) meta-analysis	975 children aged <15 years with MDR-TB	Severe underweight (WAZ <−3), clinical malnutrition, and nutritional edema	Malnutrition was strongly associated with a lower probability of treatment success (aOR 0.30; 95% CI 0.20–0.60)	Strong for TB–malnutrition, but not specific to stunting	Supporting / Excluded From Core Tb–Stunting Evidence
5	Marangu & Zar, 2019. *Childhood pneumonia in low-and-middle-income countries: An update*	Narrative review	Childhood pneumonia in LMICs	Malnutrition as a risk factor for pneumonia	TB was discussed as a potential cause or comorbidity of pneumonia, while malnutrition was described as increasing pneumonia risk and severity	Very indirect	Excluded
6	Duke et al., 2017. *Holistic care of complicated tuberculosis in healthcare settings with limited resources*	Clinical review	Children with complicated TB	Wasting and stunting explicitly discussed	Children with TB meningitis, chronic pulmonary TB, and disseminated TB were described as	High conceptual relevance, but no primary analytical evidence	Supporting / Excluded From Core Evidence

No.	Author, Year, Title	Study Design / Article Type	Population / Focus	Nutritional / Stunting Indicators	Main Relevant Findings	Relevance to TB–Stunting	Final Decision
					frequently experiencing weight loss, severe wasting, and stunting		
7	Sudfeld et al., 2017. *Effect of maternal vitamin D3 supplementation on maternal health, birth outcomes, and infant growth among HIV-infected Tanzanian pregnant women: Study protocol for a randomized controlled trial*	Randomized controlled trial protocol	HIV-positive pregnant women and their infants	LAZ; stunting defined as LAZ <−2 SD	Stunting was planned as an outcome at 12 months; however, TB was not the exposure of interest and no trial outcome data were available	Indirect	Excluded
8	Yakoob et al., 2016. *Vitamin D supplementation for preventing infections in children under five years of age*	Cochrane systematic review	Children aged <5 years	Vitamin D status and infection outcomes	TB was specified as a potential outcome, but none of the included trials reported TB outcomes	No direct TB–stunting evidence	Excluded
9	Damanik et al., 2026. *Food Intake and Nutrition in Children Undergoing Treatment for Pulmonary Tuberculosis: A Scoping Review*	Scoping review	Children undergoing treatment for pulmonary TB	Stunting, HAZ, linear growth, wasting, and underweight	The review indicated that children with TB may have lower HAZ and that nutritional and linear-growth deficits may persist during or after treatment	Very high relevance, but secondary evidence	Supporting / Excluded From Primary-Study Synthesis
10	Arolkar et al., 2022. *Role of Nutrition in Pediatric Patients with Respiratory Failure*	Narrative / clinical review	Children with respiratory failure	General malnutrition, wasting, and stunting	TB was mentioned as one potential cause of respiratory failure, but the specific relationship between TB and stunting was not evaluated	Low	Excluded
11	Reddy et al., 2021. *Severe undernutrition in children affects tuberculin skin test performance in Southern India*	Community-based observational study / secondary analysis	Household contacts of pulmonary TB cases, including 342 children aged 5–17	BMI-based undernutrition; HAZ/stunting not assessed	Severe undernutrition was associated with lower TST positivity (approximately aOR 0.30),	Indirect; reverse direction of association	Excluded

No.	Author, Year, Title	Study Design / Article Type	Population / Focus	Nutritional / Stunting Indicators	Main Relevant Findings	Relevance to TB–Stunting	Final Decision
			years		indicating an undernutrition → TB-test relationship rather than TB → growth impairment		
12	Tezol et al., 2019. *Clinical and Laboratory Evaluation of Pediatric Tuberculosis Cases in Light of Nutritional Indicators*	Retrospective case series / observational study	77 children with definite, probable, or latent TB	HFA, WFH, BMI, and chronic malnutrition	Mean HFA at diagnosis was 94.35 ± 7.50%; nutritional impairment persisted following treatment, and the change in HFA was not statistically significant (p = 0.246)	Direct and highly relevant to chronic linear-growth impairment	Included – Core Evidence

The available evidence suggests a close and potentially bidirectional relationship between childhood tuberculosis and malnutrition. While malnutrition may increase susceptibility to tuberculosis and contribute to poorer treatment outcomes, tuberculosis may further compromise nutritional status through chronic inflammation, reduced appetite, increased metabolic demands, and inadequate nutrient intake. However, most existing studies have focused on weight-based indicators such as underweight, wasting, weight-for-height, and body mass index rather than height-for-age. Evidence specifically addressing stunting or impaired linear growth among children with tuberculosis remains limited. The available primary evidence indicates that height-for-age deficits may be present at tuberculosis diagnosis and may persist despite completion of anti-tuberculosis treatment. Nevertheless, differences in anthropometric definitions, small samples, retrospective designs, and the lack of appropriate non-TB comparison groups limit causal inference. Therefore, current evidence supports an association between pediatric tuberculosis and impaired nutritional and linear growth status, but remains insufficient to establish the magnitude or direction of a causal relationship between tuberculosis and childhood stunting.

DISCUSSION

The findings of this systematic review suggest that tuberculosis exposure, infection, and active TB disease may be associated with impaired nutritional status and growth among children**,** although the strength and consistency of the evidence vary across exposure categories and anthropometric outcomes. Overall, children exposed to or affected by TB tended to demonstrate a greater nutritional vulnerability, including stunting or impaired linear growth, underweight, wasting, and growth faltering**.** However, the evidence for stunting and height-for-age impairment was less extensive than that for weight-based indicators of undernutrition, as many studies primarily assessed underweight, wasting, body mass index, or weight-for-height rather than linear growth (Nataprawira *et al.*, 2024). The available evidence also suggests that growth impairment may persist during or following TB disease and treatment, indicating that clinical recovery from TB may not necessarily be accompanied by complete nutritional or linear growth recovery (Vasiliu *et al.*, 2024). Nevertheless, the observed associations should be interpreted cautiously because the included studies differed in TB exposure definitions, anthropometric indicators, age groups, study designs, and availability of non-TB comparison groups (Masita *et al.*, 2025)**.** Importantly, household TB exposure, TB infection, and active TB disease represent clinically distinct exposure states and should not be interpreted as having equivalent effects on child growth. Therefore, the current evidence supports a potential association between TB exposure or disease and impaired childhood growth**,** particularly nutritional vulnerability and linear growth impairment, but remains

insufficient to establish a definitive causal relationship between TB exposure and childhood stunting (Mexitalia *et al.*, 2017). Therefore, the current evidence supports a potential association between TB exposure or disease and impaired childhood growth, particularly nutritional vulnerability and linear growth impairment, but remains insufficient to establish a definitive causal relationship between TB exposure and childhood stunting (Haerana *et al.*, 2021).

Several biological mechanisms may explain the observed relationship between tuberculosis and impaired growth in children. Active TB disease induces a persistent inflammatory response that can alter energy and nutrient metabolism and increase metabolic demands at a time when children require adequate energy and nutrients to support both immune function and normal physical growth (Amelia, Anggriani and Ahmad, 2026). Chronic inflammation may also contribute to anorexia and reduced dietary intake, while increased metabolic requirements and catabolic processes promote the depletion of fat and lean body mass (Massironi *et al.*, 2023). Consequently, the combination of reduced nutrient intake, increased energy expenditure, and altered nutrient utilization may create a negative nutritional balance, contributing initially to weight loss, wasting, and growth faltering (Batra *et al.*, 2026). When these nutritional deficits and inflammatory processes persist during critical periods of childhood development, they may interfere with skeletal and linear growth and potentially contribute to height-for-age deficits and stunting. This mechanism is particularly relevant because stunting reflects cumulative impairment of linear growth rather than an acute reduction in body weight; therefore, prolonged or recurrent TB-related nutritional disturbances may have consequences that extend beyond the period of active illness (Batra *et al.*, 2026). These mechanisms provide a biologically plausible explanation for the impaired nutritional and growth outcomes observed among children with TB; however, the available evidence does not establish that TB independently causes stunting, as child growth is also influenced by dietary intake, recurrent infections, socioeconomic conditions, and other potential confounding factors (Nezenega, Perimal-lewis and Maeder, 2020).

Beyond the biological effects of TB infection and disease, household TB exposure may influence child growth through socioeconomic and nutritional pathways. When a household member develops active TB, the disease may impose a substantial burden on the family through loss of income, reduced work capacity, healthcare-related expenditures, and disruption of usual caregiving responsibilities (Kilima *et al.*, 2024). These pressures may reduce the resources available for food and other essential household needs, thereby increasing the risk of household food insecurity. In food-insecure households, children may experience reduced dietary quantity and diversity, resulting in inadequate intake of energy, protein, and micronutrients required for normal growth. If these nutritional constraints are prolonged, particularly during critical periods of childhood growth, they may contribute to growth faltering and impaired linear growth and, ultimately, increase the risk of stunting (Nataprawira *et al.*, 2024).

Importantly, this pathway suggests that the potential relationship between TB and impaired childhood growth extends beyond the direct biological consequences of infection or active disease. Two interconnected pathways may therefore contribute to growth impairment: a biological pathway and a household socioeconomic pathway. In children with active TB, chronic inflammation, increased metabolic demands, reduced appetite, and altered nutrient utilization may directly compromise nutritional status and growth. In contrast, children who are only household contacts may experience nutritional vulnerability even in the absence of confirmed TB infection, because TB-related illness within the family can affect household income, food security, dietary adequacy, and caregiving capacity (Nagaraj *et al.*, 2026). These pathways may also coexist when a child both lives in a TB-affected household and develops TB infection or disease, potentially creating a compounded nutritional burden. This broader framework is important because it positions household TB exposure not merely as a risk factor for TB transmission, but as a potential marker of a wider household environment that may adversely influence child growth. Nevertheless, the socioeconomic pathway should be interpreted cautiously, as household poverty, pre-existing food

insecurity, and other social determinants may both increase TB vulnerability and independently contribute to childhood stunting (Batra *et al.*, 2026).

The relationship between tuberculosis and childhood undernutrition should be understood as bidirectional rather than unidirectional. On one side of this relationship, undernutrition may increase children's susceptibility to *Mycobacterium tuberculosis* infection and progression to active TB disease by impairing immune function (Juliasih *et al.*, 2024). Inadequate intake of energy, protein, and essential micronutrients may compromise host defense mechanisms, thereby reducing the child's capacity to mount an effective immune response against TB. Consequently, children with poor nutritional status may represent a particularly vulnerable population for TB infection and disease progression. This pathway has traditionally dominated the literature, with undernutrition commonly conceptualized as a risk factor or predisposing condition for childhood TB (Parwitha *et al.*, 2025).

Conversely, the findings synthesized in this review highlight the importance of considering the reverse pathway, in which TB exposure, infection, or active disease may contribute to subsequent nutritional deterioration and impaired growth. In children with active TB, persistent inflammation, increased metabolic demands, reduced appetite, and altered nutrient utilization may result in negative energy balance and nutritional depletion (Mangela *et al.*, 2026). When these processes are prolonged, they may manifest as weight loss, wasting, or growth faltering and may eventually interfere with linear growth. At the household level, TB may further compromise child nutrition through loss of household income, healthcare expenditure, food insecurity, reduced dietary diversity, and disruption of caregiving. Thus, TB-related growth impairment may arise not only from the direct biological consequences of disease but also from the broader socioeconomic consequences of living in a TB-affected household (Parwitha *et al.*, 2025).

Taken together, these pathways support a conceptual model of a vicious cycle between TB and undernutrition. Undernutrition may increase susceptibility to TB, while TB disease and TB-related household vulnerability may further worsen nutritional status and child growth (Cooke *et al.*, 2023). Repeated or sustained interaction between these processes may be particularly detrimental during critical periods of childhood development, when adequate nutrition is essential for linear growth. Importantly, stunting may represent a cumulative manifestation of prolonged growth impairment rather than an immediate consequence of TB exposure itself**.** This distinction is particularly relevant because household TB exposure does not necessarily indicate that a child is infected or has active disease; therefore, the biological and socioeconomic pathways should be considered separately while recognizing that they may coexist in the same child (Hassani *et al.*, 2025).

From this perspective, the contribution of the present review is to extend the conventional undernutrition to TB framework toward a bidirectional TB to undernutrition and growth impairment framework. Rather than viewing nutritional deficits solely as antecedent risk factors for childhood TB, the findings emphasize the need to consider TB exposure, infection, and disease as potential contributors to subsequent nutritional vulnerability and impaired growth (Vasiliu *et al.*, 2024). This bidirectional perspective may have important implications for TB control programs, suggesting that the management of children in TB-affected households should consider not only infection detection and disease prevention but also longitudinal monitoring of nutritional status and linear growth (Grigoryan *et al.*, 2022).

The findings of this review highlight the need to consider growth and nutritional assessment as part of a comprehensive approach to children affected by household TB (Turyasiima *et al.*, 2026). In addition to evaluating TB exposure, infection, and disease, assessment of height or length, weight, and age-appropriate anthropometric indicators such as HAZ, WAZ, and WHZ may help identify children experiencing stunting, underweight, wasting, or growth faltering (Nataprawira *et al.*, 2024). At the community and public health levels, greater integration between TB contact

investigation, growth monitoring, and nutritional screening may provide opportunities for earlier identification of vulnerable children, particularly in settings where TB and childhood undernutrition coexist (Massironi *et al.*, 2023). This review also extends the conventional perspective of undernutrition as a risk factor for TB by considering the reverse pathway from TB exposure, infection, or disease to impaired growth. Nevertheless, the findings should be interpreted cautiously because the available evidence is predominantly observational, with heterogeneity in study designs, definitions of TB exposure, anthropometric indicators, age groups, and study populations. Limited longitudinal evidence, few appropriate comparison groups, socioeconomic confounding, and potential publication bias further restrict causal interpretation (Tsibiyane *et al.*, 2026).

Future research should therefore prioritize prospective longitudinal cohorts of children with household TB exposure compared with non-exposed children, with anthropometric assessment at baseline and repeated follow-up, for example at 6, 12, and 24 months. Particular attention should be given to HAZ/LAZ trajectories, growth faltering, and incident stunting**,** while distinguishing children who remain TB-exposed but uninfected from those who develop TB infection or active disease. Future studies should also account for baseline nutritional status, dietary intake, food insecurity, socioeconomic conditions, recurrent infections, and TB preventive or therapeutic interventions. Such longitudinal evidence would help establish temporality and clarify whether impaired growth reflects the biological consequences of TB infection or disease, the socioeconomic consequences of living in a TB-affected household, or an interaction between both pathways, thereby providing stronger evidence for integrated TB and childhood nutrition interventions.

CONCLUSION

This systematic review suggests that TB exposure, infection, and active TB disease are associated with nutritional vulnerability and impaired growth in children, including stunting or impaired linear growth, wasting, underweight, and growth faltering, although the strength of evidence varies across exposure types and growth outcomes. The findings support a bidirectional relationship between TB and childhood undernutrition, in which undernutrition may increase susceptibility to TB, while TB may further compromise child growth through interacting biological and household socioeconomic pathways.

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