

Post-pulmonary tuberculosis lung function: a systematic review and meta-analysis

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Summary

Background Although post-tuberculosis lung disease poses a substantial threat to individuals who have recovered from pulmonary tuberculosis, data showing objective functional impairment in such people are scarce. We did a systematic review and meta-analysis to estimate respiratory impairment after pulmonary tuberculosis disease and examine differences in ventilatory defects.

Methods We systematically searched Embase, MEDLINE, and CINAHL from Jan 1, 2000, to Dec 13, 2024. We included any study design with data on lung function tests in individuals with a previous diagnosis of pulmonary tuberculosis versus healthy controls. Outcomes extracted from eligible studies included forced expiratory volume in 1 s (FEV₁), forced vital capacity (FVC), FEV₁ as a percentage of the predicted value (FEV₁%), FVC as a percentage of the predicted value (FVC%), and FEV₁/FVC ratio. Pre-bronchodilator values were preferentially selected. Random effects mean difference models were used when possible and standardised mean difference where it was necessary to standardise to a single scale (ie, FEV₁%, FVC%, and FEV₁/FVC ratio). Between-study heterogeneity was estimated with *I*². This study was prospectively registered with PROSPERO (CRD42021248838).

Findings Of the 5594 publications found, data from 19 studies were included for meta-analyses, reporting on 75 960 individuals of whom 7447 had past pulmonary tuberculosis. All studies reporting absolute values, using various levels of adjustment or standardisation, showed that previous pulmonary tuberculosis had a negative effect across all spirometric values: FEV₁ −0.41 L (95% CI −0.51 to −0.32, *P*=90.4%), FVC −0.25 L (−0.33 to −0.17, *P*=80.6%), and FEV₁/FVC ratio −0.37 (−0.54 to −0.19, *P*=92.0%). In those studies, using reference values to derive FEV₁% and FVC%, prior pulmonary tuberculosis had a pooled standardised mean difference of −0.44 (−0.60 to −0.28, *P*=95.6%) and −0.33 (−0.54 to −0.13, *P*=91.3%), respectively, compared with controls.

Interpretation People who recover from pulmonary tuberculosis have significantly decreased lung function compared with controls, with FEV₁ more affected than FVC, giving a mixed obstructive and restrictive picture with predominantly airflow obstruction.

Funding Breathing Matters.

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Introduction

In 2023 an estimated 10.8 million people were affected by tuberculosis disease, resulting in 1.25 million deaths.¹ Although treatment of drug-susceptible pulmonary tuberculosis is highly effective at 88%,¹ microbiological cure is unlikely to represent the end of the illness.² People who recover can be affected by residual pulmonary fibrosis, cavitation, and structural distortion ultimately leading to pulmonary remodelling, which affects respiratory capacity and function.³ This situation is further complicated by the rising burden of multidrug-resistant and extensively drug-resistant tuberculosis, resulting in more destructive lung disease which exacerbates complications.⁴ Consequently, respiratory diseases remain the leading cause of excess deaths in people who recover from tuberculosis, among whom mortality is three to six times greater than among their peers in the same location.^{5,6} With an estimated 79 million lives saved through successful

tuberculosis treatment since 2000¹ and a rising life expectancy, this tuberculosis-related lung condition poses a major threat to public health.

Post-tuberculosis sequelae with residual lung damage occurs in about 18–80% of patients³ and pulmonary dysfunction increases risk of death from respiratory causes.⁶ A distinct feature of pulmonary tuberculosis is its striking heterogeneity in severity and clinical outcomes between patients. This heterogeneity remains largely unexplained and contributes to the difficulties in accurate estimation of disease burden. The term post-tuberculosis lung disease is increasingly recognised within the tuberculosis research community, highlighting the need for more attention and interventions targeting these long-term sequelae.⁷

Post-tuberculosis lung disease can cause airflow obstruction, restrictive ventilatory defects, and impairment in gas exchange. Recent data, including large

Lancet Glob Health 2025;
13: e1020–29

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Research in context

Evidence before this study

An estimated 79 million people have recovered from tuberculosis since 2000, and a growing number of cross-sectional and longitudinal studies have described chronic lung disease and increased mortality among these individuals. However, as data on continuing care beyond tuberculosis treatment are incomplete, the extent of the respiratory impairment among individuals who have recovered from pulmonary tuberculosis is poorly characterised. Before undertaking this systematic review and meta-analysis, we searched MEDLINE, Embase, Scopus, and the Cochrane Library from database inception to March 1, 2024, with the use of search terms including “tuberculosis”, “pulmonary function”, “lung impairment”, “chronic lung disease”, “airflow obstruction”, and “post-tuberculosis sequelae”. We screened for systematic reviews, meta-analyses, and primary studies describing long-term pulmonary outcomes after tuberculosis treatment. Although several primary studies reported post-tuberculosis lung function, we identified no comprehensive systematic reviews or meta-analyses that pooled lung function measures across diverse populations, stratified by tuberculosis status and adjusted for confounders. To our knowledge, this study is the first to provide a global synthesis of spirometric outcomes in individuals who have recovered from pulmonary tuberculosis.

Added value of this study

This is a comprehensive systematic review and meta-analysis exploring the extent of lung function impairment in

individuals who recovered from pulmonary tuberculosis against healthy populations. A total of 75 960 participants, across five WHO regions, were included for quantitative analysis, of which 7447 had a history of pulmonary tuberculosis disease. We found that, overall, previous pulmonary tuberculosis had a negative pooled effect on all measured spirometric parameters. The condition affects forced expiratory volume in 1 s more substantially than forced vital capacity, in both absolute and percentage of predicted values, thus showing a mixed obstructive and restrictive picture, with a predominant airflow obstruction deficit in people who recovered from pulmonary tuberculosis. Our research provides a new insight into documented respiratory impairment in pulmonary tuberculosis survivors that has been overlooked in previous population studies, which have mainly focused on chronic obstructive pulmonary disease.

Implications of all the available evidence

Our study has substantial implications for clinical practice and policy. Post-tuberculosis lung disease is an under-recognised global challenge, with no evidence-based recommendations for investigation and management available. However, this systematic review provides compelling evidence that post-tuberculosis lung disease requires long-term respiratory care, which should be an explicit component of the WHO End Tuberculosis strategy.

population-based studies such as PREPOCOL (Columbia),⁸ PLATINO (South America),⁹ and BOLD (19 global sites),¹⁰ indicate that tuberculosis contributes substantially to the growing burden of chronic obstructive pulmonary disease. In some low-income and lower-middle-income countries, tuberculosis-associated obstructive pulmonary disease has emerged as a distinct clinical entity, disproportionately affecting younger populations, unlike smoking-associated chronic obstructive pulmonary disease (COPD), which develops later in life.^{11,12}

However, due to the insufficient number of high-quality controlled trials in this population, no evidence-based recommendations for the investigation and management of post-tuberculosis lung disease are currently available. The WHO End Tuberculosis strategy still focuses on reducing tuberculosis incidence and mortality and makes no mention of post-tuberculosis lung disease.¹³ Furthermore, WHO-recommended tuberculosis registries do not capture data beyond cure.¹⁴ Only over the last 4 years, a few consensus-based sets of clinical standards for post-tuberculosis lung disease have been published, which are mainly directed towards pulmonary rehabilitation strategies.^{15,16}

There has been little comprehensive examination of the evidence base on changes in lung function tests after

tuberculosis disease. We conducted a systematic review and meta-analysis to estimate respiratory impairment after pulmonary tuberculosis disease and examine differences in ventilatory defects.

Methods

Search strategy and selection criteria

We did a systematic review and meta-analysis, registered with PROSPERO (CRD42021248838), following the University of York Centre for Reviews and Dissemination guidelines¹⁷ and PRISMA reporting standards.¹⁸ We searched the medical databases MEDLINE, Embase, and CINAHL from Jan 1, 2000, to Dec 13, 2024, using a Boolean search strategy to combine keywords and subject headings for tuberculosis with those for lung function tests (appendix p 1). Our search strategy started from the year 2000, to coincide with the active implementation of the Global Initiative for Chronic Obstructive Lung Disease, and therefore better capture ventilatory deficits across the studies.¹⁹ No language restrictions were imposed as we did not want to introduce bias by excluding non-English publications. We also completed bibliographic screening and citation searching (with the use of the Web of Science citation search tool) of the included papers, did bibliographic screening of any

See Online for appendix

existing reviews identified as being of relevance, and consulted independent experts in the field (DG, CWMO, and WC).

All records retrieved during the searches were imported into EndNote and duplicates were deleted. Title and abstract as well as full-text screening was done by pairs of independent reviewers (SR, RE, EKD, MG, JZ, and JCP) with the use of Rayyan (v1.5.3)²⁰ and disagreements were resolved through discussion and input of a third reviewer when necessary. Google Translate was used for screening of non-English titles and abstracts, with the plan for professional translation services for full texts when required. Reason for exclusion was noted at the full-text screening stage.

Studies were included if they met all the following criteria: original research papers; included clinical, microbiological, self-reported, or treatment history of pulmonary tuberculosis; had a healthy control population; reported lung function tests as an outcome for participants; and reported or provided enough data to separate outcomes between individuals with previous pulmonary tuberculosis and controls. Studies were excluded if the control had a substantial selection bias, specifically to groups with respiratory disease, or if studies only contained data on secondary outcomes or reported duplicate data. When a population had been reported in several publications, we extracted results from the study with the most extensive exclusion criteria for the healthy control group, particularly if these factors might influence lung function outcomes (eg, acute illness and chronic respiratory conditions). Full selection criteria are outlined in the appendix (p 2).

The primary outcomes extracted were measurable effects on lung function, as identified through at least one lung function test but not limited to: forced vital capacity (FVC), forced expiratory volume in 1 s (FEV₁), FEV₁ as a percentage of that predicted (FEV₁%), FVC as a percentage of that predicted (FVC%), FEV₁/FVC ratio, flow volume loops, peak expiratory flow, transfer and diffusion factors, and residual volume. Effect measures for the relationship between pulmonary tuberculosis and lung function were extracted when reported in the included studies (eg, relative risk and odds ratios). Secondary outcomes extracted from included studies were any significant radiological findings or data on markers of inflammation.

Data analysis

Quality appraisal of all included studies was done with the use of the appropriate Joanna Briggs critical appraisal tool for cohort, case-control, or cross-sectional studies.²¹ All papers were given a quality score, with a score of 75% or more representing high quality, more than 50% and less than 75% representing medium quality, and 50% or less low quality. Data extraction and appraisal was carried out in duplicate by two independent reviewers, with disagreements resolved through discussion and input of

a third reviewer where necessary. Studies were not excluded based on quality, but information on quality was considered in the synthesis.

We used STATA (version 18)²² to calculate effect estimates and associated 95% confidence intervals (CIs). For the outcomes FEV₁ and FVC, random-effects models were used to calculate mean difference. Random effects standardised mean difference (SMD) models were used for the outcomes FEV₁%, FVC%, and FEV₁/FVC ratio. A combined natural log odds ratio was obtained by back transformation. When there were several results reported for a single outcome of interest, we extracted results based on the following preferences: pre-bronchodilator values, data after one episode of treated pulmonary tuberculosis rather than multiple episodes, estimates adjusted for the greatest number of potentially confounding baseline covariates, and data for the longest follow-up period.

Further regression and sensitivity analyses were done in STATA with the use of the metan command.²² To assess the robustness of our findings, we repeated all analyses with the use of fixed-effects models and carried out leave-one-out analysis and subgroup analyses according to study characteristics chosen post hoc. Statistical heterogeneity was assessed with the use of the *I*² statistic and *p* values reported. An *I*² value of 50–75% suggested moderate heterogeneity and 75% or more suggested high heterogeneity.²³ We assessed publication bias by visual examination of funnel plots

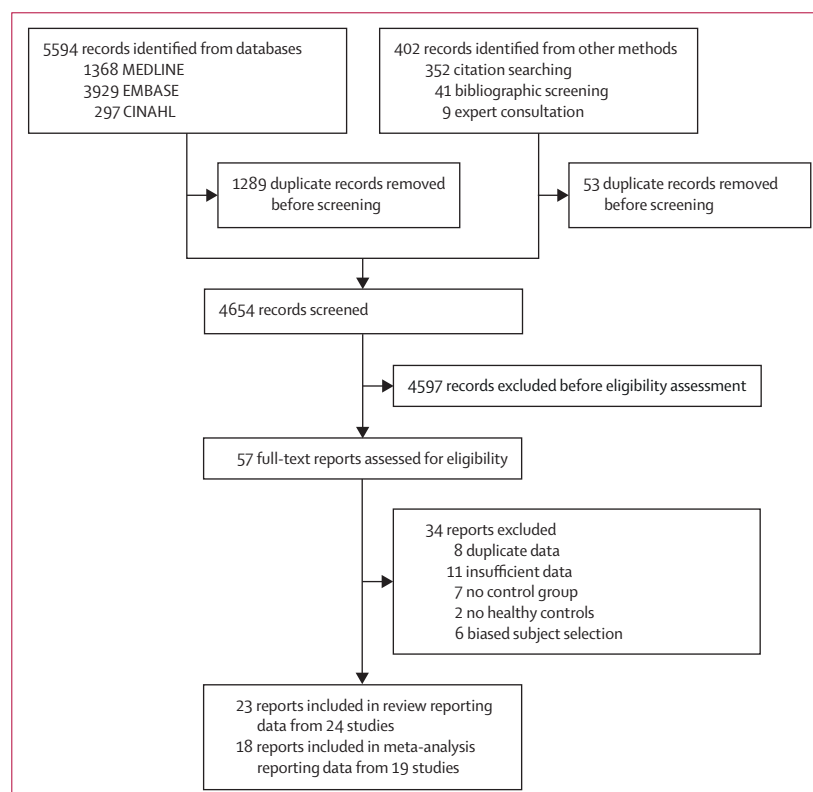


Figure 1: Study selection

	Location	Study type	Past PTB n (men/women)	Control n (men/women)	Outcome measure	Levels of adjustment or standardisation	Study quality
Hnizdo et al (2020) ²⁵	South Africa	Cohort	2137 (2137/0)	23 712 (23 712/0)	FEV ₁ , FVC, and FEV ₁ %	Population of men only, all adjusted for age and height. Predicted values from minors without PTB or pneumoconiosis	73%
Menezes et al (2007) ⁹	Brazil, Uruguay, Mexico, Chile, and Venezuela	Cross-sectional	132 (44/88)	5439 (2148/3291)	FEV ₁ , FVC, FEV ₁ /FVC; FEV ₁ %, FVC%; and OR of AFO*	Predicted values from PLATINO reference curves. OR adjusted for sociodemographic, smoking, indoor and occupational pollution, and history of hospitalisation	88%
Lam et al (2010) ³⁶	China	Cross-sectional	1954 (722/1232)	6112 (1411/4701)	FEV ₁ % and OR for AFO†	Predicted values from Chinese reference population. OR adjusted for age, sex education, smoking, biomass, and dust exposure	100%
Fullerton et al (2011) ²⁷	Malawi	Cross-sectional	19	278	FEV ₁	Adjusted for age, sex, height, cooking material, household location, sleeping location, and economic status	50%
Lee et al (2011) ³⁸	South Korea	Cross-sectional	294 (184/110)	3393 (1510/1883)	FEV ₁ , FVC, FEV ₁ /FVC; FEV ₁ %, FVC%; and OR for AFO*	Predicted values from KNHANES survey population; OR adjusted for CXR lesion, smoking, sex, and age	75%
Gomez (2012) ^{39‡}	Argentina	Case-control	25 (12/13)	27 (9/18)	FEV ₁ %	Not adjusted	40%
Ralph et al (2013) ^{30‡}	Indonesia	Prospective and case-control	200 (131/69)	40 (31/9)	FEV ₁	No difference in sex, age, ethnicity, and height between groups. Controls age, sex, height, and ethnicity matched	60%
Dhooria et al (2014) ³¹	India	Prospective and case-control	50 (33/17)	50 (31/19)	FEV ₁ and FVC	Controls age and sex matched	90%
Cole et al (2016) ³²	South Africa	Cross-sectional	15	12	FEV ₁ /FVC and FEV ₁ %	All adjusted for age, sex, current smoking, body mass index, and HIV status	63%
Osman et al (2016) ³³	Sudan	Case-control	136 (99/37)	136 (99/37)	FEV ₁ , FVC, and FEV ₁ /FVC; FEV ₁ % and FVC%; and OR for AFO*	Controls age and sex matched. Predicted values from European Respiratory Society 93' or adjusted for age and treatment delay	80%
Byrne et al (2017) ³⁴	Peru	Cohort—two groups (drug-sensitive and MDR)	Drug susceptible 144 (83/61); MDR 33 (19/14)	161 (49/112)	FEV ₁ and FVC; and OR for AFO*	All values adjusted for: age, sex, height, smoking, environmental exposure to tobacco smoke, indoor air pollution, occupational dust, born outside Lima, and presence of atopy	64%
Fiogbe et al (2019) ³⁵	Benin	Cross-sectional	189 (128/61)	70 (48/22)	FEV ₁ and FVC	Adjusted for age, sex and tobacco and biomass exposure. Controls matched by sex, age, and size	100%
Nightingale et al (2019) ³⁶	Malawi	Cross sectional	47	1434	FEV ₁ , FVC, and FEV ₁ /FVC	Adjusted for age, sex, weight, and height	88%
El Sayed El Shourbagy et al (2019) ^{37*‡}	Egypt	Case-control	20 (10/10)	20 (10/10)	FEV ₁ , FVC; FEV ₁ %; and FEV ₁ /FVC	Controls were age and sex matched	70%
Kim et al (2019) ³⁸	South Korea	Cross-sectional	1482 (847/635)	14 034 (6013/8021)	FEV ₁ , FVC, and FEV ₁ /FVC; and FEV ₁ % and FVC%	Not adjusted. Predicted values from KNHANES survey population ³⁹	100%
Kamenar et al (2022) ⁴⁰	Argentina, Uruguay, Bangladesh, Uganda, Peru, Congo, India, and Chile	Cross-sectional	332	12 064	FEV ₁ and FVC; and FEV ₁ /FVC	All adjusted for sex, smoking, age, education, and biomass fuel use	100%
Nkereuwem et al (2022) ⁴¹	The Gambia	Cross-sectional	52	89	FEV ₁ , FVC, and FEV ₁ /FVC	Data were standardised using the Global Lung Initiative 2012 African American reference ranges. The African American reference ranges have been validated among African children	88%
Fink et al (2022) ^{42‡}	Nigeria	Cohort	49	147	FEV ₁ , FVC, and FEV ₁ /FVC	Values used not adjusted	75%

(Table continues on next page)

	Location	Study type	Past PTB n (men/women)	Control n (men/women)	Outcome measure	Levels of adjustment or standardisation	Study quality
(Continued from previous page)							
Shanmugasundaram et al (2022) ^{43‡}	India	Case-control	10 (5/5)	10 (5/5)	FEV ₁ %, FVC%; FEV ₁ /FVC; and impulse oscillometry parameters	No difference in age, height, or sex between groups. Predicted values based on reference values from Global Lung Initiative South-East Asian population	70%
Shui et al (2023) ⁴⁴	China	Case-control	51 (18/33)	51 (24/27)	FEV ₁ %, FVC%, FEV ₁ /FVC; and transfer factor, lung volumes, and cardiopulmonary exercise test parameters	BMI and gender-matched to non-smoking healthy controls. Percentage of the predicted values based on patient gender, age, weight, and height	50%
Xing et al (2023) ⁴⁵	Tibet and Xiangjiang Uygur autonomous region	Cross-sectional	610 (328/282)	8070 (3945/4125)	FEV ₁ , FEV ₁ %, FVC, FVC%, and FEV ₁ /FVC; small airway dysfunction assessment; and OR for AFO*	Predicted values based on the reference values from the lung function equations for a northeast Asian population. Adjusted OR: adjustments for age, sex, region, education plus history of asthma, exposure to household air pollution and occupation, and smoking status	75%
Martinez et al (2023) ^{46‡}	South Africa	Prospective cohort	96	972	Tidal breathing measures, respiratory impedance, and lung clearance index	Adjusted infant length, sex, maternal HIV status, maternal smoking during pregnancy, age at visit, and previous lung function measurements	91%
van der Zalm et al (2024) ⁴⁷	South Africa	Prospective cohort	50 (19/31)	50 (28/22)	FEV ₁ and FVC; FEV ₁ /FVC; and transfer factor and lung volumes	Adjusted for age, sex, height, and ethnicity (other) ⁴⁸ with the use of Global Lung Initiative reference	90%
AFO=airflow obstruction. CXR=chest x-ray. FEV ₁ =forced expiratory volume in 1 s. FVC=forced vital capacity. FEV ₁ %=FEV ₁ as a percentage of the predicted value. FVC%=FVC as a percentage of the predicted value. MDR=multidrug resistant. OR=odds ratio. PTB=pulmonary tuberculosis. *Defined by FEV ₁ /FVC<0.7. †Defined by FEV ₁ /FVC<lower limit of normal. ‡Not included in meta-analysis.							
Table: Summary of included study characteristics							

and with the use of Egger tests where the analysis included at least the minimum number of studies needed to distinguish chance from real asymmetry.^{22,24}

Role of the funding source

The funder of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

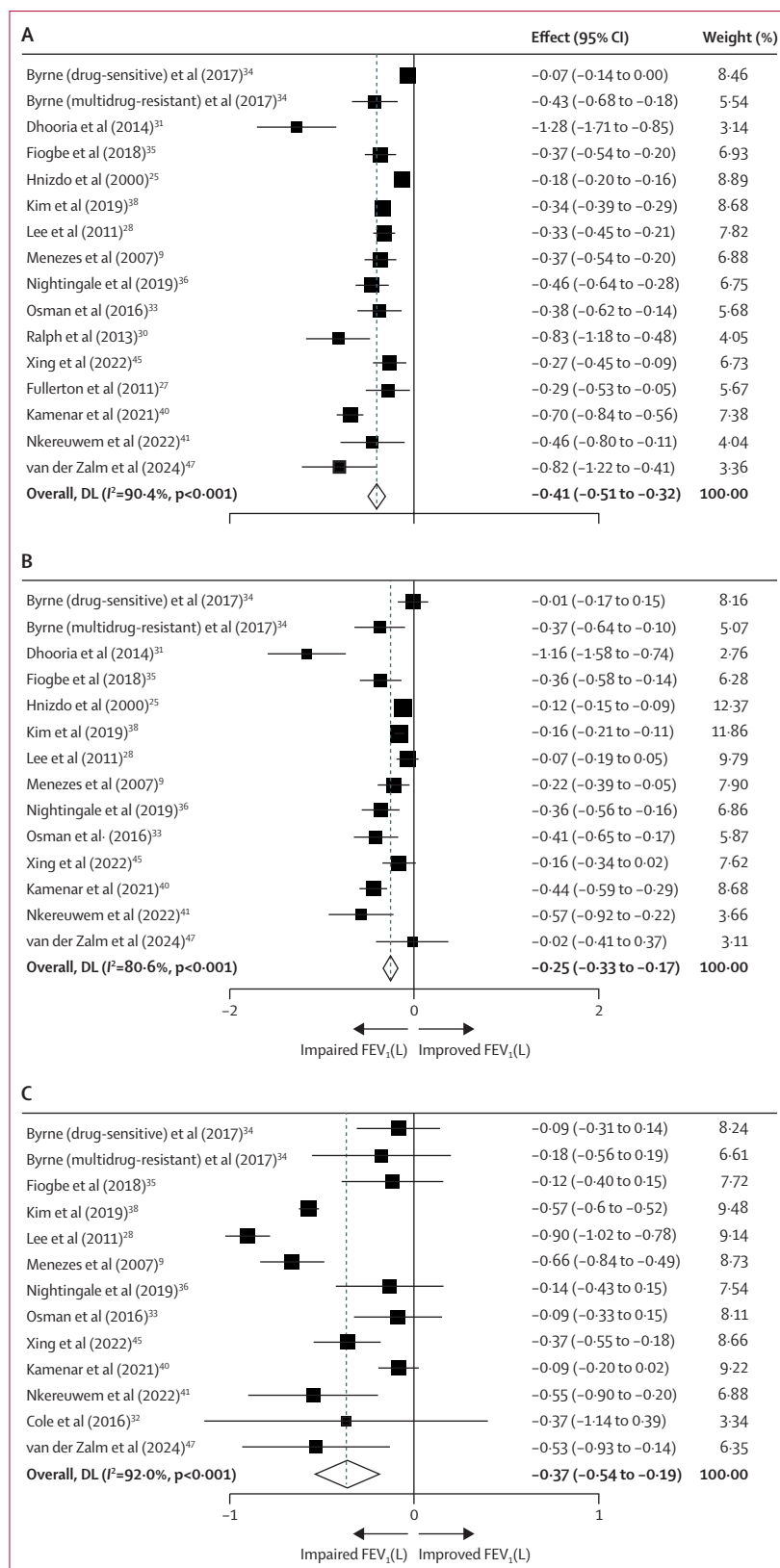
Results

Database searches yielded 5594 publications, with 4305 publications carried forward for title and abstract screening after removal of duplicates (figure 1). A further 349 records were identified through bibliography screening and recommendations from experts. The full texts of 57 publications were screened, of which 34 records were excluded. Altogether, 23 publications (reporting data from 24 studies) met the inclusion criteria, with 19 studies included for quantitative synthesis (figure 1). No translations were required. Studies excluded consisted of eight due to duplicates, 11 with insufficient data, seven without control groups, two with no healthy controls, and six with biased selection.

A total of 75 960 participants were included for quantitative analysis, of whom 7447 had a history of pulmonary tuberculosis. The characteristics of these studies are presented in the table. The publications

included in the analyses comprised 12 cross-sectional,^{9,26–28,32,35,36,38–41,45} three cohort,^{25,47,48} and four case-controls studies^{30,32,42,47} containing data from 2000 to 2024. Studies were conducted in five WHO regions (appendix pp 4–7), across 22 countries: South Africa (four studies),^{25,32,46,47} Benin (one study),³⁵ Sudan (one study),³³ Malawi (two studies),^{27,46} Uganda (one study),⁴⁰ India (three studies),^{31,40,43} South Korea (two studies),^{28,38} China (two studies),^{26,44} Tibet (one study),⁴⁵ Indonesia (one study),³⁰ Peru (two studies),^{34,40} Brazil (one study),⁹ Uruguay (two studies),^{9,40} Mexico (one study),⁹ Chile (two studies),^{9,40} Venezuela (one study),⁹ The Gambia (one study),⁴¹ Bangladesh (one study),⁴⁰ and Congo (one study),⁴⁰ Argentina (two studies),^{29,40} Egypt (one study),³⁷ and Nigeria (one study;⁴² table).

The average age range of individuals who had recovered from pulmonary tuberculosis was 5–62 years, with many studies skewed towards a younger population (<50 years). One study included children younger than 12 years (appendix pp 4–7).⁴¹ Altogether 47% of participants were women and many were from upper-middle-income to low-income countries (appendix pp 4–7). Several studies defined a previous diagnosis of pulmonary tuberculosis with confirmed medical histories,^{9,25,29–33,35,37,47–49} whereas the remaining studies used either self-reporting^{27,36,38,40} or radiology (appendix pp 4–7).^{26,28,38,45}



Three studies used post-bronchodilator values,^{33,39,43} whereas one³¹ did not specify whether pre-bronchodilator or post-bronchodilator values were used, but all were included in analyses. One study³⁴ presented two populations with previous pulmonary tuberculosis—drug-sensitive and multidrug-resistant disease. One study included a single participant who was HIV-positive in the healthy control group.⁴⁷ Given the negligible effect on the overall control group, the study was retained in the meta-analysis. Most studies used some level of adjustment or standardisation to process absolute values and reference populations to derive percentage of predicted values (table). The quality of the included studies ranged from fair to high, with three studies noted as low quality (table, appendix pp 8–9).^{27,29,44} Publications were not excluded based on study quality.

All studies consistently observed a negative effect of previous pulmonary tuberculosis on lung function compared with healthy controls across all spirometry measures. Two studies reported on a reduction of transfer factor, lung volumes, and exercise capacity in participants with previous tuberculosis.^{44,47} Xing and colleagues⁴⁵ specifically showed small airways dysfunction was associated with previous pulmonary tuberculosis. Four studies excluded from meta-analysis showed significantly lower FEV₁^{37,43} and FEV₁%^{29,37} in individuals with previous pulmonary tuberculosis versus controls. Two studies^{29,37} reported no difference in bronchial responsivity, measured by methacholine challenge testing between groups, although increased bronchial responsivity was found in patients with active tuberculosis.³⁷

A total of 16 studies were included for FEV₁ analysis and 14 for FVC, with varying levels of in-study adjustments or standardisation (table). Individuals with previous pulmonary tuberculosis had significantly lower pooled effect estimates for both FEV₁ (-0.41 L, 95% CI -0.51 to -0.32, $I^2=90.8\%$) and FVC (-0.25 L, 95% CI -0.33 to -0.17, $I^2=80.6\%$) than controls (figure 2). Pooled SMD for FEV₁/FVC ratio was -0.37 (95% CI -0.53 to -0.19, $I^2=92.0\%$; figure 2).

Nine studies included data on FEV₁% and six on FVC% (table). Participants with previous pulmonary tuberculosis had significantly lower pooled estimates than controls: FEV₁% was -0.44 (95% CI -0.60 to -0.28, $I^2=95.6\%$) and FVC% -0.33 (-0.54 to -0.13, $I^2=91.3\%$) (figure 3). Given that four studies reported adjusted odds ratios (OR) for airflow obstruction after pulmonary tuberculosis, post-hoc analysis was done to give a combined natural log OR of 0.81 (95% CI 0.41–2.2; figure 3). Back transformation gave a log OR of 2.25 (1.51–1.95). One study explicitly

Figure 2: Effect of history of pulmonary tuberculosis on (A) FEV₁, (B) FVC, and (C) FEV₁/FVC ratio

DL=DerSimonian and Laird method. FEV₁=forced expiratory volume in 1 s. FVC=forced vital capacity. PTB=pulmonary tuberculosis. DS=drug sensitive. MDR=multidrug resistant. Weights are from random-effects model.

showed irreversible airflow obstruction.²⁶ Regression analysis between each spirometric parameter and weighted mean age showed no significant correlation (appendix p 10).

Sensitivity analysis with the use of both fixed-effect and random-effect models (appendix pp 11–12) mostly showed higher pooled effect sizes with the latter, although both models reached statistical significance for all parameters. Leave-one-out analysis confirmed all studies remained within the 95% CIs, with no clear outliers (appendix p 13).

Heterogeneity was moderate to high across all analyses (I^2 72.1–95.6%; figures 2, 3). Subgroup analyses based on geographical region, adjustments, and study type conducted (appendix pp 14–15) did not significantly alter between-study heterogeneity. In instances when heterogeneity appeared to decrease, the number of included studies was substantially reduced, limiting the ability to draw definitive conclusions.

Funnel plot analysis and Egger's test for publication bias showed significant asymmetry with FEV₁ ($p=0.002$) and FVC ($p=0.01$; appendix p 16). FEV₁/FVC ratio did not show evidence of publication bias ($p=0.2$; appendix p 16). We did not have sufficient power to do statistical tests for bias for FEV₁% and FVC% because a minimum of ten studies is necessary;²⁴ however, visual inspection of FEV₁% and FVC% funnel plots did not suggest small-study bias (appendix p 16).

Secondary outcomes collected showed that three studies found an association between chest x-ray lesions and lung function impairment.^{25,27,34} A west African study showed that previous tuberculosis was strongly associated with increased risk of chronic lung disease, outweighing typical risk factors in the region, such as smoking.⁴² One study showed no difference in inflammatory markers (ie, erythrocyte sedimentation rate, sputum and blood eosinophils, or C-reactive protein) between groups.³⁷ However, in another study,⁴³ elevated concentrations of matrix metalloproteinase-1 were observed in individuals with post-tuberculosis sequelae.

Discussion

This systematic review and meta-analysis shows that a history of pulmonary tuberculosis significantly reduces objectively measured lung function compared with healthy controls. Overall, individuals with previous pulmonary tuberculosis had lower measured spirometric parameters than healthy controls—FEV₁ (mean difference -0.41 L), FVC (mean difference -0.25 L), and FEV₁/FVC ratio (SMD -0.37)—implicating a mainly obstructive ventilatory deficit in people who recovered. These findings are compelling when put into clinical context, as a decrease in FEV₁ of 100 mL is considered clinically significant and a strong, independent predictor of cardiovascular and respiratory disease outcomes.⁵⁰ These studies cross various WHO regions with varying tuberculosis incidence and income levels but consistently observe some form of pulmonary impairment following tuberculosis (appendix pp 4–7).

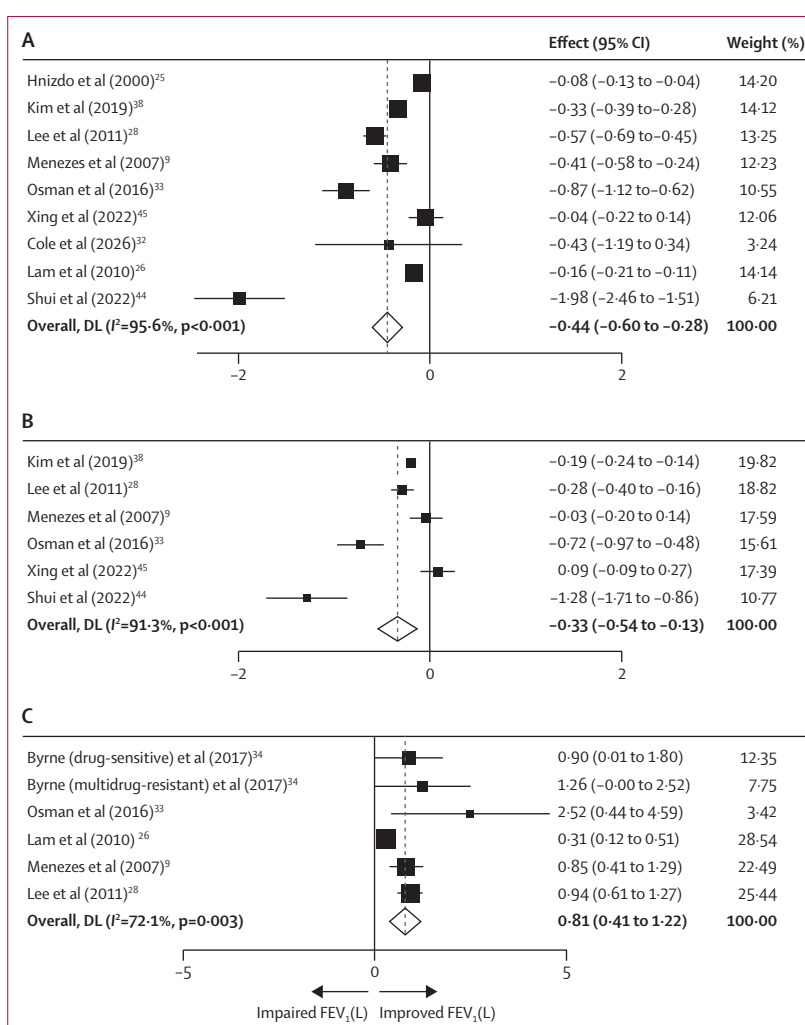


Figure 3: Effect of history of pulmonary tuberculosis on (A) FEV₁%, (B) FVC%, and (C) log odds ratio of airflow obstruction

DL=DerSimonian and Laird method. FEV₁=forced expiratory volume in 1 s. FEV₁%=FEV₁ as a percentage of the predicted value. FVC=forced vital capacity. FVC%=FVC as a percentage of the predicted value. PTB=pulmonary tuberculosis. Airflow obstruction defined by an FEV₁/FVC <0.7 or FEV₁/FVC <lower limit of normal.

Our data are consistent with those of other population studies showing an increased risk of air flow obstruction in individuals who recovered from tuberculosis.^{10,51} A meta-analysis from Byrne and colleagues⁴⁹ found a pooled OR of 3.05 (95% CI 2.42–3.85) between past tuberculosis and COPD, with the strongest associations in countries with high tuberculosis incidence and in young people. Checkley's group further supports this finding in their study,⁴⁰ which exclusively looked at low-income and middle-income countries (LMICs). The authors report an observed OR of 3.78 (2.87–4.98) between previous tuberculosis and airflow obstruction. Although we reported a modest adjusted pooled OR of 2.25, our primary meta-analysis dissects this relationship further with more detailed spirometric data.

We found that absolute FEV₁ decreased more than did FVC, by 0.41 L and 0.26 L, respectively. Prospective studies, although scarce, suggest that the pattern of respiratory impairment varies through and beyond treatment completion.^{35,50,51} In pulmonary tuberculosis at diagnosis, Plit and colleagues⁵² found that FVC improved to a greater degree than FEV₁ over the course of treatment. Nightingale and colleagues,⁵⁴ having the longest follow-up period of 3 years, reported a third of individuals who recovered from tuberculosis experienced accelerated FEV₁ decline. This observation implies that successful treatment might prevent restrictive sequelae to a greater degree than obstructive loss.

Although our data showed a lower FVC and FVC% decline, the effect size remains clinically and statistically significant (mean difference -0.25 L and SMD -0.33, respectively, $p=0.0001$ for both), suggesting that, although the net effect of pulmonary tuberculosis appears to be obstructive, a restrictive component is also present. Similarly, in a South African population study,⁵⁵ a combined obstructive and restrictive defect was the most common functional outcome as a sequela of pulmonary tuberculosis in a South African population. Amaral and colleagues⁵¹ found that LMICs had the strongest association with both spirometric obstruction and restriction. It is apparent that current estimates of residual spirometric abnormalities after pulmonary tuberculosis vary widely according to the population.

The association between tuberculosis and lung function impairment is influenced by confounding risk factors such as biomass exposure, HIV, diabetes, and smoking.⁵⁶ Smoking remains the highest risk factor for COPD globally, as well as a powerful risk factor for developing pulmonary tuberculosis.⁵⁶ This bidirectional relationship might have influenced our findings; however, the data remained consistent even when adjusted for smoking (OR 2.25 for tuberculosis and airflow obstruction). Population studies in LMICs also indicate that pulmonary tuberculosis is a stronger risk factor for COPD than smoking.^{8,12}

These findings support the hypothesis that previous pulmonary tuberculosis is an independent risk factor for obstructive airways disease. In cumulative lung damage caused by smoking-related COPD, the extent of alveolar destruction and airway obstruction is slowly progressive, which makes this condition uncommon in younger people. On the other hand, pulmonary tuberculosis is primarily a disease of young adults, and the associated lung damage occurs during the acute disease process. This characteristic explains why pulmonary tuberculosis' relative contribution to COPD is higher in the younger population, especially in tuberculosis endemic areas. This pattern has led to the term tuberculosis-associated obstructive lung disease.^{11,12}

The pathophysiology of the functional tuberculosis sequelae is speculative but is likely to be heterogeneous given the spectrum of clinical and radiological outcomes.

Structural pulmonary changes resulting from aberrant tissue repair (eg, bronchovascular distortion, fibrosis, and pleural thickening) might explain airflow restriction.³ However, the mechanisms that drive airflow obstruction are poorly understood.⁵³ This area of research is an important line of research as any pulmonary dysfunction can increase the risk of death from respiratory causes³ and contribute to the excess deaths observed in people who recover from tuberculosis.^{5,6}

The limitations of this study are in part related to the use of published data, with the validity of the results dependent on the conduct and reporting of the studies included. Second, a causal relationship between pulmonary tuberculosis and lung function impairment cannot be fully determined as the current evidence is primarily from cross-sectional and case-control studies. Pulmonary tuberculosis exposure was defined in some studies clinically, which could be another potential limitation if these individuals were not correctly diagnosed. However, all included studies were completed in regions of high tuberculosis incidence, so exposure risk is high (appendix pp 4–7). Furthermore, globally only 63% of pulmonary tuberculosis cases are diagnosed with microbiological confirmation,¹ particularly in areas of high exposure where clinically diagnosed tuberculosis might be more common than in areas of low exposure.

Comparison of spirometric values across studies is challenging without adjusting for key variables. Although using percentage predicted values accounts for some variability, biases related to age, height, and ethnicity can remain, particularly at extremes. However, age-related bias might be mitigated by the predominantly younger study populations (<50 years; table). Although ethnic adjustments for lung function were not explicit, our findings align with those of other large single race reviews.⁵⁷ Most studies also included some level of adjustment or standardisation for absolute spirometric values (table). Furthermore, OR analysis for airflow obstruction used the most adjusted values and still support our findings. Overall, despite these limitations, the observed effect size remains consistent and significant across all spirometric measures.

Although significant heterogeneity was observed with I^2 (72.1–95.6%; figures 2, 3), this figure does not account for the other sources of statistical variability. Consequently, we compared I^2 values with the use of both random effect and fixed-effect models (appendix pp 11–12). The latter method yielded lower effect estimates, suggesting true between-study variability.²⁴ To address clinical and methodological heterogeneity, we conducted post-hoc subgroup analyses based on geographical region, adjustments, and study type (appendix pp 14–16). Although the direction of effect size remained consistent across subgroups, other reductions in heterogeneity were constrained by a substantial drop in study numbers (appendix pp 14–16). To further validate our findings, we performed leave-one-out analysis, confirming that all studies remained within the 95% CI, which

indicates the robustness and reliability of our data, with no significant influence from a single study (appendix p 13). Residual heterogeneity might be partly attributed to study differences such as case selection, study quality, timing of lung function after treatment, and tuberculosis diagnostic criteria. However, tuberculosis itself is a highly complex disease with a spectrum of variable outcomes at both a population and individual level.⁵⁸

A substantial research gap in this review is caused by the absence of data on plethysmography and gas transfer assays, which might be due to resource limitations. As lung pathologies often coexist (eg, emphysema and fibrosis) this can potentially lead to falsely low or preserved FVC readings.^{59,60} In this instance, simple spirometry alone does not fully reveal residual respiratory impairments after pulmonary tuberculosis. Furthermore, potential effect modifiers, such as smoking history, HIV status, and other comorbidities, should also be consistently reviewed to understand the relationship between exposure and outcome.

The strengths of this review come with our adherence to PRISMA standards and the use of global multi-database searches, along with the input of expert groups, ensuring as much of the available literature as possible was captured. The decision to pool data was carefully considered based on comparable lung function outcomes (ie, FEV₁, FVC) in comparison to healthy controls. Despite some variability across study populations, the core research question remained consistent between studies. This meta-analysis enhances understanding of respiratory deficits that are clinically and policy-relevant, providing granularity beyond broader spirometric definitions such as the Global Initiative for Obstructive Lung Disease criteria.¹⁹ The narrative synthesis further contextualizes these findings, offering insights into the factors driving the observed results.

In summary, these data present strong evidence that individuals affected by pulmonary tuberculosis have significant lung function loss with a mixed restrictive and obstructive picture with predominantly airflow obstruction compared with healthy populations. Previous population studies have observed a mainly obstructive pattern potentially due to their narrower focus on COPD. Our study has substantial implications for clinical practice and policy as currently post-tuberculosis lung disease remains an under-recognised global challenge, affecting tens of millions of people who have undergone pulmonary tuberculosis treatment worldwide. International guidelines need to include recommendations for medium and long-term follow-up of patients with pulmonary tuberculosis. This requirement will impose a large burden on both health and social care, which deserve greater integration into the WHO End Tuberculosis strategy. Finally, understanding the mechanisms that underpin this process might enable the development of host-directed therapies to limit or prevent the development of the chronic sequelae of pulmonary tuberculosis.

Contributors

JSF, JCP, and SEH developed the concept and study design. SR, SEH, EKD, RE, MG, and JZ carried out screening, data extraction, and critical appraisal with input from all other authors. WC, JSF, DG, CWMO, and JCP provided expert input on included papers. SR, SEH, and LPG conducted meta-analyses with oversight from WC. SR and SEH wrote the first draft of the manuscript, with input from JCP and JSF. All authors contributed to critical review of data and manuscript review.

Declaration of interests

We declare no competing interests.

Data sharing

Materials on characteristics of each study are available in the appendices. The extracted data used within our analysis are available on request to the authors.

Acknowledgments

SR is supported by Breathing Matters, UCL Respiratory, and The Mason Medical Research Fellowship. SEH acknowledges funding from the UK Medical Research Council (MR/N013638/1) and the National Institute for Health and Care Research (NIHR206676). DG is partially supported by Ricerca Corrente funding from the Italian Ministry of Health, Linea 4. CWMO is supported by the National Medical Research Council, Singapore (grant numbers CSAINV21nov-0003 and CIRG21jun-0004). EKD, RE, MG, JZ, and JCP acknowledge funding from the National Institute of Health and Care Research (NIHR) University College London Hospitals Biomedical Research Centre, which receives funding from NIHR.

References

- 1 WHO. Global tuberculosis report 2024. <https://www.who.int/teams/global-programme-on-tuberculosis-and-lung-health/tb-reports/global-tuberculosis-report-2024> (accessed April 25, 2025).
- 2 Allwood B, van der Zalm M, Makanda G, Mortimer K, and the Steering Committee of the First International Post-Tuberculosis Symposium. The long shadow post-tuberculosis. *Lancet Infect Dis* 2019; **19**: 1170–71.
- 3 Ravimohan S, Kornfeld H, Weissman D, Bisson GP. Tuberculosis and lung damage: from epidemiology to pathophysiology. *Eur Respir Rev* 2018; **27**: 170077.
- 4 Singla R, Mallick M, Mrigpuri P, Singla N, Gupta A. Sequelae of pulmonary multidrug-resistant tuberculosis at the completion of treatment. *Lung India* 2018; **35**: 4–8.
- 5 Romanowski K, Baumann B, Basham CA, Ahmad Khan F, Fox GJ, Johnston JC. Long-term all-cause mortality in people treated for tuberculosis: a systematic review and meta-analysis. *Lancet Infect Dis* 2019; **19**: 1129–37.
- 6 Ranzani OT, Rodrigues LC, Bombarda S, Minto CM, Waldman EA, Carvalho CRR. Long-term survival and cause-specific mortality of patients newly diagnosed with tuberculosis in São Paulo state, Brazil, 2010–15: a population-based, longitudinal study. *Lancet Infect Dis* 2020; **20**: 123–32.
- 7 Allwood BW, Byrne A, Meghji J, Rachow A, van der Zalm MM, Schoch OD. Post-tuberculosis lung disease: clinical review of an under-recognised global challenge. *Respiration* 2021; **100**: 751–63.
- 8 Caballero A, Torres-Duque CA, Jaramillo C, et al. Prevalence of COPD in five Colombian cities situated at low, medium, and high altitude (PREPOCOL study). *Chest* 2008; **133**: 343–49.
- 9 Menezes AMB, Hallal PC, Perez-Padilla R, et al. Tuberculosis and airflow obstruction: evidence from the PLATINO study in Latin America. *Eur Respir J* 2007; **30**: 1180–85.
- 10 Buist AS, McBurnie MA, Vollmer WM, et al. International variation in the prevalence of COPD (the BOLD Study): a population-based prevalence study. *Lancet* 2007; **370**: 741–50.
- 11 Aggarwal D, Gupta A, Janmeja AK, Bhardwaj M. Evaluation of tuberculosis-associated chronic obstructive pulmonary disease at a tertiary care hospital: a case-control study. *Lung India* 2017; **34**: 415–19.
- 12 Zoller T, Mfinanga EH, Zumba TB, et al. Chronic airflow obstruction in Tanzania—a cross-sectional study. *BMC Pulm Med* 2018; **18**: 11.
- 13 WHO. The End TB Strategy. 2015. <https://www.who.int/publications-detail-redirect/WHO-HTM-TB-2015.19> (accessed April 25, 2025).

- 14 WHO. Understanding and using tuberculosis data. 2014. <https://www.who.int/publications/i/item/9789241548786> (accessed April 25, 2025).
- 15 Migliori GB, Marx FM, Ambrosino N, et al. Clinical standards for the assessment, management and rehabilitation of post-TB lung disease. *Int J Tuberc Lung Dis* 2021; **25**: 797–813.
- 16 Nightingale R, Carlin F, Meghji J, et al. Post-TB health and wellbeing. *Int J Tuberc Lung Dis* 2023; **27**: 248–83.
- 17 Centre for Reviews and Dissemination. Systematic reviews: CRD's guidance for undertaking reviews in health care. 3rd ed. University of York, 2009.
- 18 Page MJ, McKenzie JE, Bossuyt PM, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. *BMJ* 2021; **372**: n71.
- 19 Rodriguez-Roisin R, Rabe KF, Vestbo J, Vogelmeier C, Agustí A, and the all previous and current members of the Science Committee and the Board of Directors of GOLD. Global Initiative for Chronic Obstructive Lung Disease (GOLD) 20th Anniversary: a brief history of time. *Eur Respir J* 2017; **50**: 1700671.
- 20 Ouzzani M, Hammady H, Fedorowicz Z, Elmagarmid A. Rayyan-a web and mobile app for systematic reviews. *Syst Rev* 2016; **5**: 210.
- 21 Joanna Briggs Institute. Critical appraisal tools. 2020. <https://jbi.global/critical-appraisal-tools> (accessed April 25, 2025).
- 22 Stata Corp. Stata statistical software: release 17. 2021. <https://www.stata.com> (accessed April 24, 2023).
- 23 Higgins JPT, Thompson SG, Deeks JJ, Altman DG. Measuring inconsistency in meta-analyses. *BMJ* 2003; **327**: 557–60.
- 24 Higgins JPT, Thomas J. Cochrane handbook for systematic reviews of interventions version 6.3. Cochrane, 2022.
- 25 Hnizdo E, Singh T, Churchyard, G. Chronic pulmonary function impairment caused by initial and recurrent pulmonary tuberculosis following treatment. *Thorax* 2020; **55**: 32–38.
- 26 Lam KBH, Jiang CQ, Jordan RE, et al. Prior TB, smoking, and airflow obstruction: a cross-sectional analysis of the Guangzhou Biobank Cohort Study. *Chest* 2010; **137**: 593–600.
- 27 Fullerton DG, Suseno A, Semple S, et al. Wood smoke exposure, poverty and impaired lung function in Malawian adults. *Int J Tuberc Lung Dis* 2011; **15**: 391–98.
- 28 Lee SW, Kim YS, Kim DS, Oh YM, Lee SD. The risk of obstructive lung disease by previous pulmonary tuberculosis in a country with intermediate burden of tuberculosis. *J Korean Med Sci* 2011; **26**: 268–73.
- 29 Gomez Tejada R. Bronchial responsiveness (BR) evaluation in TB patients. Comparison to asthma patients and normal subjects. *Am J Respir Crit Care Med* 2012; **185**: A3319.
- 30 Ralph AP, Kenangalem E, Waramori G, et al. High morbidity during treatment and residual pulmonary disability in pulmonary tuberculosis: under-recognised phenomena. *PLoS One* 2013; **8**: e80302.
- 31 Dhooria S, Kumar P, Saikia B, et al. Prevalence of Aspergillus sensitisation in pulmonary tuberculosis-related fibrocavitary disease. *Int J Tuberc Lung Dis* 2014; **18**: 850–55.
- 32 Cole G, Miller D, Ebrahim T, Dreyden T, Simpson R, Manie S. Pulmonary impairment after tuberculosis in a South African population. *S Afr J Physiother* 2016; **72**: 307.
- 33 Osman RK, Mortimer K, Bjune G, El Sony AI. Chronic respiratory disease in adults treated for tuberculosis in Khartoum, Sudan. *Public Health Action* 2016; **6**: 199–204.
- 34 Byrne AL, Marais BJ, Mitnick CD, et al. Chronic airflow obstruction after successful treatment of multidrug-resistant tuberculosis. *ERJ Open Res* 2017; **3**: 00026–2017.
- 35 Fiogbe AA, Agodokpessi G, Tessier JF, et al. Prevalence of lung function impairment in cured pulmonary tuberculosis patients in Cotonou, Benin. *Int J Tuberc Lung Dis* 2019; **23**: 195–202.
- 36 Nightingale R, Lesosky M, Flitz G, et al. Noncommunicable respiratory disease and air pollution exposure in Malawi (CAPS). *Am J Respir Crit Care Med* 2019; **199**: 613–21.
- 37 El Sayed El Shourbagy NM, Abumossalam AM, Arram EO, et al. Study of airway hyperresponsiveness in patients with pulmonary tuberculosis. *Egypt J Chest Dis Tuberc* 2019; **68**: 505–13.
- 38 Kim MS, Choi CJ, Kwon KM, Kim KS, Choi WS, Oh YJ. Association of lung function with serum 25-hydroxyvitamin D level according to the presence of past pulmonary tuberculosis in Korean adults. *Korean J Fam Med* 2019; **40**: 93–99.
- 39 Oh K, Kim Y, Kweon S, et al. Korea National Health and Nutrition Examination Survey, 20th anniversary: accomplishments and future directions. *Epidemiol Health* 2021; **43**: e2021025.
- 40 Kamenar K, Hossen S, Gupta AN, et al. Previous tuberculosis disease as a risk factor for chronic obstructive pulmonary disease: a cross-sectional analysis of multicountry, population-based studies. *Thorax* 2022; **77**: 1088–97.
- 41 Nkereuwem E, Agbla S, Sallahdeen A, et al. Reduced lung function and health-related quality of life after treatment for pulmonary tuberculosis in Gambian children: a cross-sectional comparative study. *Thorax* 2023; **78**: 281–87.
- 42 Fink DL, Oladele DA, Slack AJ, et al. A multi-centre observational study of HIV, tuberculosis and risk of chronic lung disease in urban West Africa. *AIDS* 2022; **36**: 1987–95.
- 43 Shanmugasundaram K, Talwar A, Madan K, Bade G. Pulmonary Functions and Inflammatory Biomarkers in Post-Pulmonary Tuberculosis Sequelae. *Tuberc Respir Dis* 2022; **85**: 175–84.
- 44 Shui L, Wang H, Yang B, Li G, Chen Y. Characteristics of cardiopulmonary exercise test in tracheobronchial tuberculosis patients with central airway stenosis. *Heart Lung* 2023; **59**: 73–81.
- 45 Xing Z, Sun T, Janssens JP, et al. Airflow obstruction and small airway dysfunction following pulmonary tuberculosis: a cross-sectional survey. *Thorax* 2023; **78**: 274–80.
- 46 Martinez L, Gray DM, Botha M, et al. The long-term impact of early-life tuberculosis disease on child health: a prospective birth cohort study. *Am J Respir Crit Care Med* 2023; **207**: 1080–88.
- 47 van der Zalm MM, Jongen VW, Swanepoel R, et al. Impaired lung function in adolescents with pulmonary tuberculosis during treatment and following treatment completion. *EClinicalMedicine* 2024; **67**: 102406.
- 48 Smith S-J, Gray DM, MacGinty RP, et al. Choosing the better global lung initiative 2012 equation in South African population groups. *Am J Respir Crit Care Med* 2020; **202**: 1724–27.
- 49 Byrne AL, Marais BJ, Mitnick CD, Lecca L, Marks GB. Tuberculosis and chronic respiratory disease: a systematic review. *Int J Infect Dis* 2015; **32**: 138–46.
- 50 Cazzola M, MacNee W, Martinez FJ, et al. Outcomes for COPD pharmacological trials: from lung function to biomarkers. *Eur Respir J* 2008; **31**: 416–69.
- 51 Amaral AFS, Coton S, Kato B, et al. Tuberculosis associates with both airflow obstruction and low lung function: BOLD results. *Eur Respir J* 2015; **46**: 1104–12.
- 52 Plit ML, Anderson R, Van Rensburg CE, et al. Influence of antimicrobial chemotherapy on spirometric parameters and pro-inflammatory indices in severe pulmonary tuberculosis. *Eur Respir J* 1998; **12**: 351–56.
- 53 Allwood BW, Maasdorp E, Kim GJ, et al. Transition from restrictive to obstructive lung function impairment during treatment and follow-up of active tuberculosis. *Int J Chron Obstruct Pulmon Dis* 2020; **15**: 1039–47.
- 54 Nightingale R, Chinoko B, Lesosky M, et al. Respiratory symptoms and lung function in patients treated for pulmonary tuberculosis in Malawi: a prospective cohort study. *Thorax* 2022; **77**: 1131–39.
- 55 Ehrlich RI, Adams S, Baatjies R, Jeebhay MF. Chronic airflow obstruction and respiratory symptoms following tuberculosis: a review of South African studies. *Int J Tuberc Lung Dis* 2011; **15**: 886–91.
- 56 Alvarado González A. Tuberculosis obstructive pulmonary diseases (TOPD). *Clin Res Trials* 2021; **7**: 2–7.
- 57 Jung J-W, Choi JC, Shin JW, Kim JY, Choi BW, Park IW. Pulmonary impairment in tuberculosis survivors: The Korean National Health and Nutrition Examination Survey 2008–2012. *PLoS One* 2015; **10**: e0141230.
- 58 Cadena AM, Fortune SM, Flynn JL. Heterogeneity in tuberculosis. *Nat Rev Immunol* 2017; **17**: 691–702.
- 59 Heckman EJ, O'Connor GT. Pulmonary function tests for diagnosing lung disease. *JAMA* 2015; **313**: 2278–79.
- 60 Doherty MJ, Pearson MG, O'Grady EA, Pellegrini V, Calverley PM. Cryptogenic fibrosing alveolitis with preserved lung volumes. *Thorax* 1997; **52**: 998–1002.