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Role of interferon-gamma and interleukin-17 in the immune response to pulmonary tuberculosis

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Interferon-gamma and interleukin-17 are important in immune response to pulmonary tuberculosis as they activate the macrophage host defense, promote cell-mediated immunity, and modulate the action of the pulmonary inflammatory processes which are essential in containing *Mycobacterium tuberculosis*.This study was designed to determine the contribution made by interferon-gamma and interleukin-17 to the immune response to pulmonary tuberculosis and measure their possible usefulness as immunological biomarkers with respect to disease activity and host defense. We conducted a case control study (April–December 2025) with 70 new patients with pulmonary tuberculosis and 25 healthy age and sex controls to compare the levels of IFN-7 and IL-17. Clinical, radiological and microbiological parameters were used in the diagnosis and the severity of the disease was determined based on clinical parameters. ELISA was used to measure serum IFN-gamma and IL-17. The normal hematological and inflammatory parameters including ESR and CRP were measured through the normal automated laboratory parameters following the application of stringent exclusion criteria to minimize the confounding factor. There were no significant differences in the baseline characteristics between the patients and controls except that BMI was lower in the patients with tuberculosis. Significantly high levels of IFN-gamma and IL-17 were related to pulmonary tuberculosis, and they escalated with the severity of the disease. The two cytokines were also linked with the duration of the symptoms, ESR, and CRP but not with BMI. The diagnostic sensitivity of IFN-7 and IL-17 was found to be good using ROC analysis, and the combination of the markers had the highest sensitivity, specificity and general accuracy in detecting tuberculosis. High levels of IFN-17 in pulmonary tuberculosis indicate an increase in Th1 and Th17 immune stimulation induced by the continued stimulation of mycobacterial antigen and thereby leading to an increase in effective host defense as well as the severity of inflammation thereby justifying its utility as mechanistically appropriate diagnostic and prognostic biomarkers.

Keywords: pulmonary tuberculosis; interferon-gamma; interleukin-17; immune response; inflammatory biomarkers.

Introduction

One of the most important infectious diseases in the world has been pulmonary tuberculosis (TB), which has been a leading contributor to morbidity and mortality in spite of the methods of diagnosis and the treatment approaches that have been adopted (Ong et al., 2020). *Mycobacterium tuberculosis* causes the disease and it is an intracellular pathogen that has an impressive capacity of avoiding the host immune system and developing chronic infection. The clinical spectrum of pulmonary TB is characterized by mild and localized disease development and severe and disseminated disease, which in turn is the result of complex interactions between the pathogen and the host immune system. The immunological pathways that regulate the course of the disease and the host defense, thus, have to be identified to be able to enhance the accuracy of diagnosis, prognostic analysis, and host-directed therapy development (Patil et al., 2018; Chakaya et al., 2021).

The mechanism of cell-mediated immunity is crucial in controlling the *M. tuberculosis* infection. $CD4^+$ T-lymphocytes are of special importance among immune cells, because they coordinate responses that protect against the threat by means of releasing cytokines which cause the work of the macrophages and modulate the inflammation (Khanna & Yasmeen, 2024). Interferon-gamma (IFN-γ) is widely known as an essential cytokine in antimycobacterial immunity and is a characteristic of the T-helper 1 (Th1) immune response. The effect of IFN-γ on the microbicidal activity of macrophages is the ability to stimulate the maturation of phagolysosomes, the synthesis of nitric oxide, and the presentation of antigens. It is well known that defects in IFN-γ signaling, be they genetic or acquired, are closely linked with high susceptibility to mycobacterial infection, which serves as a reminder of the essential role of IFN-γ in host defense against TB (Al-Qaisi et al., 2025).

Active pulmonary tuberculosis is characterized by the presence of high concentrations of IFN-γ which is an indication of the presence of a continuous immune response to generate control of bacterial multiplication. Nevertheless, too much or too long duration of IFN-g production can also cause immunopathology, tissue damage and the increase the severity of the disease. This dual action makes it important to note that the balanced IFN-γ signaling is important to the clinical outcomes. In addition, differences in the levels of IFN-γ in TB patients have been associated with the disease extent, bacterial load, and treatment response, indicating that it can be used as a biomarker of the disease activity and progression (Lu et al., 2019; Ghanavi et al., 2021; Malik et al., 2025).

Simultaneously with the Th1 reactions, more focus has been given to the contribution of T-helper 17 (Th17) cells and their signature cytokine, interleukin-17 (IL-17) in pulmonary tuberculosis. In its turn, IL-17 is a proinflammatory cytokine that is important in the recruitment of neutrophils and other immune cells to infection sites by inducing chemokines and adhesion molecules (Shen & Chen, 2017) IL-17 in the context of TB plays a role in the early development of granuloma, and also aids in the development of the immune microenvironment necessary to contain *M. tuberculosis*. It has been shown experimentally that IL-17 deficiency has the potential to suppress pro-

tective immunity, especially in the early phase of infection (Bewket et al., 2022).

However, it is also possible that dysregulated IL-17-driven inflammation also worsens the pathology of the lungs. Overproduction of IL-17 has been linked to increased neutrophilic inflammatory changes, tissue damage and cavitary disease in pulmonary TB (Gurczynski & Bethany, 2018). Therefore, just like IFN-G, IL-17 has protective and pathogenic properties, which depend on the extent and the time of its expression. It seems that homeostasis between Th1- and Th17-mediated responses is the key to successful immunity and immune pathology in tuberculosis (Mannion et al., 2023).

It is also indicated that a functional interaction between IFN-γ and IL-17 plays a role in the immune response to *M. tuberculosis*. Although macrophage activation and killing of bacteria require IFN-γ, IL-17 enhances persistent recruiting and expansion of the immune cells and local inflammatory responses (Khanna et al., 2024). The dysregulation of this cytokine network can result in an uncontrolled inflammation, defects in clearance of bacteria or progressive disease. Analyzing the synergistic actions of IFN-γ and IL-17 can thus give more insight into the disease pathogenesis compared to the effect of each cytokine (Diatlova et al., 2023; Khaleel Ibrahim et al., 2025).

Due to the heterogeneity of pulmonary tuberculosis and the shortcomings of traditional diagnostic and prognostic markers, the study of immune biomarkers which would indicate the severity and activity of the disease is drawing increasing interest. The contribution of IFN-γ and IL-17 to the host defense and pathogenesis, exploring the role of these cytokines in pulmonary TB could be an asset in not only elucidating their role in the body but also in clinical applications as immunological biomarkers of disease monitoring and risk assessment.

Material and methods

The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment, and confidentiality of personal data was strictly maintained.

The study was a case–control study that was performed between April 21, 2025, and December 18, 2025. A total of 70 patients with a newly diagnosed pulmonary tuberculosis were registered, and 25 clinically healthy people were utilized as the control group. Controls were age and sex fitted and had no clinical or laboratory history of tuberculosis or other infectious or inflammatory diseases. The diagnosis of pulmonary tuberculosis involved a clinical presentation as well as a combination of chest radiography and microbiological confirmation such as sputum smear microscopy. The treating physicians determined the severity of the disease in terms of mild, moderate, and severe depending on the clinical findings and radiological extent of the lung involvement plus lab findings. To keep the effects of confounding factors such as inflammatory biomarkers to a minimum, the study excluded individuals with extrapulmonary tuberculosis, coexisting inflammatory or autoimmune diseases, chronic liver or renal conditions, malignancy, HIV infection, diabetes mellitus, or those who had received antituberculosis therapy, immunosuppressive drugs, or chemotherapy before inclusion. Venous blood (5 mL) was taken from each participant before and after overnight fasting under aseptic conditions. Blood samples were left to clot and subsequently centrifuged at 3000 rpm and 10 minutes to obtain serum that was aliquoted and stored at –20 °C awaiting analysis. The level of IFN-γ and IL-17 in serum was determined using commercial enzyme-linked immunosorbent assay (ELISA) kits, according to the instructions of the manufacturer. Each sample was subjected to two solutions in order to test the reliability of the assays. An automated hematology analyzer with a white blood cell count, level of hemoglobin and platelet count were used to determine regular hematological parameters. Standard automated laboratory measurements of biochemical parameters were used to assess erythrocyte sedimentation rate (ESR), C-reactive protein (CRP) and liver and renal function tests of choice.

Statistical analysis was performed on SPSS software (version 26.0). The data were given in terms of mean standard deviation (SD) in the case of continuous variables and percentages and frequencies in the case of categorical variables. The independent samples t-test or one-way analysis of variance (ANOVA) was used in making comparisons between groups with normally distributed variables, and nonparametric tests were used where applicable. The correlations between the levels of cytokines and the clinical or laboratory parameters were measured by Pearson's correlation coefficient . The diagnostic performance of IFN-including IL-17 was compared by receiver operating characteristic (ROC) curve analysis. The P-value of less than 0.05 was given significance.

Results

The baseline comparison in Table 1revealed no statistically significant differences between pulmonary tuberculosis patients and healthy controls in terms of age, gender distribution, residence, or smoking status ($P > 0.05$), indicating good demographic matching between the groups. In contrast, body mass index was significantly lower among tuberculosis patients compared with controls (21.3 ± 3.1 vs. 24.2 ± 2.8 kg/m²; $P < 0.001$), reflecting disease-associated weight loss and increased metabolic demand. These findings suggest that nutritional status, rather than demographic factors, may play a relevant role in the clinical profile of pulmonary tuberculosis patients.

Table 1
Sociodemographic and clinical characteristics of pulmonary tuberculosis patients and healthy controls

Variable	Pulmonary TB patients (n = 70)	Controls (n = 25)	P-value
Age (years), mean ± SD	41.6 ± 12.3	39.8 ± 11.1	0.48
Gender (male/female), n (%)	44 (62.9%) / 26 (37.1%)	15 (60%) / 10 (40%)	0.81
Residence (urban/rural), n (%)	38 (54.3%) / 32 (45.7%)	14 (56%) / 11 (44%)	0.88
Smoking status (yes/no), n (%)	29 (41.4%) / 41 (58.6%)	6 (24%) / 19 (76%)	0.12
BMI (kg/m²), mean ± SD	21.3 ± 3.1	24.2 ± 2.8	0.001

Pulmonary tuberculosis patients exhibited significantly higher serum levels of both interferon-gamma and interleukin-17 compared with healthy controls. Mean IFN-γ concentrations were markedly elevated in patients (38.7 ± 9.6 pg/mL) relative to controls (18.4 ± 5.2 pg/mL; $P < 0.001$), indicating enhanced Th1-mediated immune activation. Similarly, IL-17 levels were significantly increased in the tuberculosis group (29.1 ± 7.8 pg/mL) compared with controls (12.6 ± 4.1 pg/mL; $P < 0.001$), reflecting activation of Th17-associated inflammatory pathways involved in host defense and disease-related inflammation.

A clear severity-dependent increase in inflammatory cytokine levels was observed among pulmonary tuberculosis patients. Serum IFN-γ concentrations rose progressively from mild to severe disease (31.4 ± 6.8, 38.9 ± 7.5, and 47.2 ± 8.1 pg/mL, respectively; $P < 0.001$), indicating intensified Th1-mediated immune activation with advancing disease. Similarly, IL-17 levels showed a significant stepwise elevation across severity groups (23.5 ± 5.9, 29.6 ± 6.7, and 35.8 ± 7.2 pg/mL; $P < 0.001$), reflecting enhanced Th17-driven inflammatory responses associated with increased tissue involvement and disease burden.

Table 2
Serum levels of interferon-gamma and interleukin-17 in pulmonary tuberculosis patients and controls

Biomarker	TB patients (n = 70) mean ± SD	Controls (n = 25) mean ± SD	P-value
IFN-γ, pg/mL	38.7 ± 9.6	18.4 ± 5.2	<0.001
IL-17, pg/mL	29.1 ± 7.8	12.6 ± 4.1	<0.001

Table 3
Variation of interferon-gamma and interleukin-17 levels according to disease severity in pulmonary tuberculosis patients

Biomarker	Mild TB (n = 24)	Moderate TB (n = 26)	Severe TB (n = 20)	P-value
IFN-γ, pg/mL	31.4 ± 6.8[a]	38.9 ± 7.5[ab]	47.2 ± 8.1[b]	<0.001
IL-17, pg/mL	23.5 ± 5.9[a]	29.6 ± 6.7[ab]	35.8 ± 7.2[b]	<0.001

Correlation analysis demonstrated significant positive associations between both IFN-γ and IL-17 levels and the duration of symptoms, ESR, and CRP, indicating that increasing cytokine concentrations are closely linked to prolonged disease course and heightened systemic inflammation. IFN-γ showed moderate positive correlations with symptom duration (r = 0.42), ESR (r = 0.47), and CRP (r = 0.51), while IL-17 exhibited similar trends. Conversely, both cytokines were negatively correlated with BMI, suggesting that enhanced inflammatory and immune activation is associated with disease-related weight loss and poor nutritional status in pulmonary tuberculosis patients.

Table 4
Correlation between inflammatory biomarkers and clinical-laboratory parameters in pulmonary tuberculosis patients

Variable	IFN-γ (r / P-value)	IL-17 (r / P-value)
Duration of symptoms, weeks	0.42 / 0.001	0.38 / 0.002
ESR, mm/hr	0.47 / <0.001	0.41 / 0.001
CRP, mg/L	0.51 / <0.001	0.45 / <0.001
BMI, kg/m²	−0.36 / 0.003	−0.32 / 0.006

ROC curve analysis demonstrated strong diagnostic accuracy for both IFN-γ and IL-17 in distinguishing pulmonary tuberculosis patients from healthy controls. IFN-γ showed high sensitivity (82.9%) and specificity (80.0%) with an AUC of 0.88, while IL-17 yielded comparable performance (AUC = 0.84). Notably, the combined use of IFN-γ and IL-17 significantly improved diagnostic power, achieving higher sensitivity (90.0%), specificity (84.0%), and the largest AUC (0.92), indicating a synergistic effect and supporting their combined utility as robust biomarkers for TB detection.

Table 5
Diagnostic performance of IFN-γ and IL-17 for discriminating pulmonary tuberculosis patients from controls

Biomarker	Cut-off value, pg/mL	Sensitivity, %	Specificity, %	AUC (95% CI)	P-value
IFN-γ	≥ 26	82.9	80.0	0.88 (0.80–0.95)	<0.001
IL-17	≥ 20	78.6	76.0	0.84 (0.75–0.92)	<0.001
IFN-γ + IL-17	–	90.0	84.0	0.92 (0.86–0.97)	<0.001

Discussion

The current research offers immunological and clinical data showing that pulmonary tuberculosis (PTB) is defined by a complex of inflammatory cytokine responses that are dominated by Th1- and Th17-like pathways. The internal validity of the biomarker comparisons is supported by the fact that the groups (age, sex, residence, and smoking status; P > 0.05) are demographically comparable, minimizing the possibility that the differences in cytokines were caused by baseline confounders. Conversely, the BMI of PBT patients was significantly less (P = 0.001), which is in line with the catabolic and inflammatory activity of active TB. Biologically, TB patients lose weight due to the elevated energy expenditure at rest, diminished appetite, and muscle proteolysis and lipolysis, which ties systemic inflammation to unhealthy nutritional condition and disease burden.

One of the findings was a significant increase of IFN-γ and IL-17 in patients with TB compared to the controls (all P < 0.001). This trend is consistent with the existing immunopathogenic models whereby IFN-γ is the key effector cytokine of Th1 immunity, which activates macrophage, increases phagolysosome maturation, enhanced antigen presentation, and antimicrobial effector functions that are essential to control *Mycobacterium tuberculosis.* In modern mechanistic studies of AML and myeloid malignancies, the importance of reconstituting immune responses and tissue microenvironment by chronic inflammatory cytokine networks has been highlighted (Zhang et al., 2018; Feng et al., 2021). This idea can be applied to TB wherein sustained release of cytokine by persistent antigenic stimulation can be maintained.

Active engagement of Th17 pathway is also supported by the fact that IL-17 is increased in PTB patients. The IL-17 enhances the local inflammation by inducing chemokines (e.g., CXCL1/CXCL8), which attracts neutrophils and strengthens cellular transport into infected lung tissue. Literature based on cytokine networks suggests that there is seldom a unidirectional activity of immune signaling. Iinstead, pro- and anti-inflammatory mediators are dynamically regulated in response to antigen burden and tissue injury (Akhter et al., 2023). This is clinically important in TB since IL-17 may be protective in early life by enhancing containment, but may be destructive when overexpressed by being associated with neutrophil-dominated inflammation and lung damage. Therefore, the presence of increased IL-17 that we have detected fits well into a dual process of inflammation especially in the presence of the disease in the active stages (Chamoun et al., 2018).

This interpretation is reinforced by the severity stratified results. Both IFN-γ and IL-17 increased gradually between mild and severe TB (P < 0.001), indicating that cytokine increases follow the disease severity and perhaps the bacillary load. The biological plausibility of this finding is that due to the further accumulation of bacterial replication and tissue involvement, the antigen presentation and stimulation of innate immunity may become more intense, further extending Th1/Th17 polarization and cytokine. Likewise, severity-dependent immune restructuring has been reported in malignant inflammatory microenvironments, an inflammatory condition can be used to further risk stratification (Gaines, 2022); similarly, the inflammatory signature of TB can be a measurable correlate of clinical severity. There are, however, other findings in chronic inflammatory disorders that indicate that IFN is not necessarily proportional to the disease severity due to immune fatigue, pathways or compartments of cytokine responses (local lung vs. peripheral blood) (Ward & John, 2021; Landy et al., 2024). This may explain interstudy differences especially in cases where timing, disease stage or even status of treatment of the sample varies.

In the TB cohort, correlation analysis indicated positive associations of both cytokines, duration of symptoms, ESR and CRP, but negative associations with BMI. Such data suggests that IFN-7 and IL-17 are evidence of long term systemic inflammation and a long term process of the disease and also associate immune activation with caused nutritional degradation. CRP is a known acute-phase reactant, although, it is vulnerable to false highs and non-specificity in the inflammatory conditions (Liu et al., 2022). Consequently, the reported cytokine-CRP relationship confirms the idea that IFN-γ and IL-17 present immunologically specific data in addition to the traditional acute-phase ones. The negative correlation with BMI favours a mechanistic mode of action where chronic inflammation leads to TB wasting by mediating metabolic dysregulation via cytokines (Baluku et al., 2024; Arif et al., 2025).

Lastly, strong diagnostic results for both cytokines were yielded by ROC analyses, which showed better results combined together (AUC 0.92). This indicates additive or synergies in information sensing embodied by the integration of Th1 (IFN-γ) and Th17 (IL-17) axes in line with cytokine network models highlighting multi-marker responses to interventions as opposed to the effects of an individual mediator (Ludi et al., 2023). The differences in the reported cut-offs and AUCs among studies are not surprising since the differences in assays platforms (ELISA kits, calibration), the heterogeneity of the populations (age, comorbidities), and the distribution of the phenotype of the disease can alter biomarker distributions. However, our findings indicate the usefulness of the dual-cytokine profiling as an adjunctive tool to TB risk stratification and monitoring.

Conclusion

Sustained activation of Th1 and Th17 immune pathways in response to *Mycobacterium tuberculosis* leads to pulmonary tuberculo-

sis being linked with significant increases in IFN-γ and IL-17. Enhanced activation of macrophages and intracellular killing of bacteria under the influence of increased IFN-gamma and in the case of IL-17 enhanced neutrophil recruitment and local inflammation. The increase of these cytokines , wqhich is dependent on severity, indicates the growing weight of antigens and tissue infiltration. The fact that they are closely related to inflammatory markers and nutritional depletion underlines their mechanism usefulness and complementary application as accurate biomarkers of disease activity and severity.

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