

PULMONARY TUBERCULOSIS TREATMENT AND ITS EFFECT ON LEPTIN LEVELS AMONG TRIBAL AND NON-TRIBAL POPULATIONS OF SOUTHERN RAJASTHAN

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ABSTRACT

Objective: Pulmonary tuberculosis (PTB) remains a leading cause of morbidity and mortality globally, disproportionately affecting poor and malnourished populations. Malnutrition, usually noted among tribal groups, exacerbates vulnerability to infections. Adipose tissue is usually used to secrete the hormone leptin, which is closely related to immunological responses, strength balance, and nutritional health. This study aimed to analyze the modifications in serum leptin levels among tribal and non-tribal PTB patients in Southern Rajasthan throughout extensive and continuation levels of anti-tubercular therapy (ATT).

Methods: A complete of 207 newly diagnosed PTB patients (123 tribal and 84 non-tribal) and 207 healthy controls were enrolled in this comparative, analytical study. Patient's anthropometric measurements (specifically body mass index [BMI]) and serum leptin levels had been recorded before remedy and again after 3 months of ATT. Statistical evaluation among the tribal and non-tribal populations was done using an unpaired t-test. $p < 0.05$ is considered statistically significant.

Results: The mean BMI of patients significantly increased from $15.10 \pm 2.04 \text{ kg/m}^2$ before treatment to $19.12 \pm 2.21 \text{ kg/m}^2$ after treatment ($p < 0.05$) and was remarkably lower as compared to healthy controls, $21.07 \pm 2.64 \text{ kg/m}^2$. Before starting treatment, patients' mean serum leptin levels and pleural fluid leptin levels were considerably lower at $1.98 \pm 0.71 \text{ ng/mL}$ and $1.62 \pm 1.11 \text{ ng/mL}$, respectively. Following medication, there was a significant increase of $4.20 \pm 1.0 \text{ ng/mL}$. Before treatment, the mean BMI $14.99 \pm 1.85 \text{ kg/m}^2$ levels among tribal patients were lower as compared to the non-tribal patients, 15.23 ± 2.29 . Further, serum leptin levels among the tribal population, before treatment were $1.77 \pm 0.66 \text{ ng/mL}$ and after treatment were 4.03 ± 1.05 as compared to the non-tribal group, $15.23 \pm 2.29 \text{ kg/m}^2$ and $2.29 \pm 0.68 \text{ ng/mL}$, respectively.

Conclusion: ATT significantly improves nutritional markers and leptin levels in PTB patients. Notably, tribal populations exhibit a greater deficit at baseline, underscoring the role of malnutrition in disease progression and the need for targeted nutritional interventions.

Keywords: Pulmonary tuberculosis, Leptin, Tribal populations, Malnutrition, Anti-tubercular therapy, Southern Rajasthan.

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INTRODUCTION

Tuberculosis (TB) remains a prime worldwide health subject notwithstanding advances in diagnosis, remedy, and prevention [1]. TB document represents that the high burden of TB in low- to middle-income nations is closely related to social determinants, together with malnutrition, poverty, and limited healthcare get entry to [2]. In many areas of India, tribal populations are especially prone to TB because of their socioeconomic constraints, lower literacy rates, and geographical isolation [3]. Due to the persistently high TB incidence in southern Rajasthan, which is home to a sizable tribal population, public health initiatives have been implemented to improve prognosis, adherence to treatment, and normal affected person outcomes [4].

One of the most common headaches in TB is weight reduction or "intake," historically identified as a trademark of the disease [5]. In malnourished individuals, weakened immune responses exacerbate susceptibility to infection and can avert recovery. The 16-kDa hormone leptin, which is mostly released by white adipose tissue, functions in the brain to change normal body weight, power expenditure, and food intake [2]. Body mass index (BMI) is connected with leptin levels, which rise in overweight individuals and fall in wasting individuals [6]. Leptin is a multipurpose hormone that does more than just control weight and hunger. It affects neuroendocrine functions, angiogenesis, bone formation, reproduction, hematopoiesis, and immunology in addition to controlling food intake and energy homeostasis [7]. Two opposing mechanisms may influence leptin plasma concentrations in TB patients: The host's acute inflammatory

response, which raises leptin levels and may result in anorexia, appetite suppression, and decreased body mass, and chronic inflammation, which reduces leptin production by causing loss of body fat mass [6,8].

Despite rising evidence regarding leptin's position in TB, distinctly little research has specifically investigated tribal versus non-tribal variations in leptin changes for the duration of anti-tubercular therapy. Hence, studying leptin fluctuations from pre-remedy to the intensive and continuation phases can shed light on the effectiveness of trendy anti-TB regimens in enhancing not only bacterial clearance but also metabolic and immunological parameters.

This examines objectives to (a) evaluate modifications in leptin levels in pulmonary TB (PTB) sufferers before and after 3 months of anti-tubercular therapy (ATT), (b) examine those adjustments between tribal and non-tribal populations, and (c) elucidate the ability clinical implications of leptin level changes on disease development and healing. With the aid of that specializing in a region of high TB incidence and a population characterized with the aid of stated dietary demanding situations, this work underscores the wider significance of integrating dietary support with biomedical treatments to optimize patient outcomes [9].

METHODS

Study design and setting

The study's objective was to evaluate the leptin status of patients receiving anti-TB treatment and those who had recently received a

TB diagnosis. The study was conducted in TB patients attending the outpatient department as well as in patients of the Department of Chest Medicine, medicine at PIMS, Udaipur, Rajasthan.

This cross-sectional study included 414 samples. After ethical approval from the Institutional Ethics Committee and informed consent was obtained from the participants before enrollment.

Participants and sampling

Inclusion criteria

(1) Newly diagnosed smear-positive or clinically shown PTB patients aged between 18 and 60 years; (2) Willingness to take part and offer informed consent; (3) No previous records of anti-TB remedy.

Exclusion criteria

(1) Patients with comorbid situations along with diabetes, human immunodeficiency virus/acquired immunodeficiency syndrome, malignancies, persistent renal failure, or any immune-compromising disease; (2) Pregnant or lactating women; (three) individuals on corticosteroids or immune-suppressive therapy.

A total of 414 participants were recruited. A detail history was taken from patients who got newly diagnosed with PTB. The patients were informed about the study in their own language. Participant signed informed consent was obtained. The patients were measured for height (h) while standing straight and barefoot, and for weight (w) using a digital standing scale while wearing very little clothing. BMI was calculated using the standard components as per the MS Excel 2007 Software. Statistical analysis was done using the Statistical Package for the Social Sciences Software.

Weight (kg)

BMI = Height (m)²

BMI categories were defined as underweight (<18.5 kg/m²), normal weight (18.5–24.9 kg/m²), overweight (25–29.9 kg/m²), and obese (≥30 kg/m²).

Laboratory investigations

Peripheral venous blood samples (5 mL) were drawn from all participants under aseptic conditions. Before analysis, the serum was centrifuged and kept at -80°C. Serum and pleural fluid leptin levels were quantified using a commercially available enzyme-linked immunosorbent assay kit. In addition, sputum smears for acid-fast bacilli, chest radiographs, and other routine investigations were performed for PTB patients.

Treatment and follow-up

All PTB patients received standard ATT under direct observation as per national guidelines. The therapy consisted of 2 months of intensive treatment (isoniazid, rifampicin, pyrazinamide, and ethambutol), followed by 4 months of continuation therapy (isoniazid and rifampicin). Serum leptin levels and anthropometric measurements were reassessed 3 months after therapy concluded.

Statistical analysis

Information was entered right into a spreadsheet and analyzed the usage of suitable statistical software program. Continuous variables were expressed as mean±standard deviation, and categorical variables were presented as proportions or frequencies. Mean BMI and leptin levels before and after treatment, as well as across tribal and non-tribal agencies, are assessed using the unpaired t-test. A p<0.05 was considered statistically significant, and p<0.001 was taken as rather huge.

RESULTS

Overall demographics and baseline characteristics

This study included a total of 207 patients. Of the 207 PTB patients, 123 (59.42%) had been from tribal populations and 84 (40.58%) had

been from non-tribal populations. The implied age of PTB patients changed into 45.2±14.6 years (range 18–60). Out of the 207 patients, 143 were male and 64 were female, with a mean age of 43.227±14.89 years. PTB and healthy controls had similar distributions by age and gender. The minimum age of the patients was 18 years, and their maximum age was 60 years. The mean age of the patients was 43.22±14.89. The mean height of the patients was 1.69±0.08 m. Table 1 represents the changes in mean weight of the PTB subjects was 43.48±5.60 kg. The mean BMI of TB patients before and after 3 months of treatment was 15.09±2.04 kg/m² and 19.12±2.21 kg/m², respectively, which was considerably lower than compared of healthy control, 21.07±2.64 kg/m². The mean BMI levels among tribal and non-tribal populations before treatment were 14.98±1.85 kg/m² and 15.23±2.29 kg/m² and after 3 months of treatment were 18.34±1.89 kg/m² and 20.26±2.18 kg/m² and among healthy controls, it was 21.30±2.65 kg/m² and 20.78±2.62 kg/m². Male and female patients have mean BMIs of 14.70±1.79 and 15.96±2.30 kg/m², respectively (Fig. 1).

Fig. 1 compared the levels of Serum Leptin and Pleural Fluid among Healthy control, before treatment and Undergoing Treatment group and shows the differences in their mean values in Serum and Pleural Fluid as 5.79 ± 2.04, 1.98 ± 0.72, 4.20 ± 1.00 and 1.62 ± 1.11 Before treatment.

Fig. 2. Shows the mean BMI levels among Tribal and Non-Tribal Population before treatment was 14.98 ± 1.85 kg/m² and 15.23 ± 2.29 kg/m² and after 3 months of treatment was 18.34 ± 1.89 kg/m² and 20.26 ± 2.18 kg/m² and among healthy controls it was 21.30 ± 2.65 kg/m² and 20.78 ± 2.62 kg/m². Male and female patients have mean BMIs of 14.70 ± 1.79 and 15.96 ± 2.30 kg/m², respectively.

Malnutrition status amongst PTB patients

Fig. 3 represents that out of, 207 newly diagnosed PTB patients, 118 (94.6%) patients fall under category of malnutrition. Before treatment, approximately 57.00% (n=118) of the tribal patients were suffering from malnutrition compared to the non-tribal patients being 37.68% (n=78) and about 11 patients (5.3%) had BMI more than 18.5.

Tribal versus non-tribal differences

BMI differences

Mean BMI among newly diagnosed PTB tribal patients was 14.98±1.85 kg/m², which was markedly lower than the non-tribal patients, 15.23±2.29 kg/m². Further, mean BMI levels after treatment among tribal patients were 18.34±1.89 kg/m² whereas among non-tribal patients, it was 20.26±2.18 kg/m².

Differences in leptin levels

Fig. 4 shows that prior to therapy, the mean leptin levels of tribal patients were lower (1.77±0.66 ng/mL) than those of non-tribal patients (2.29±0.68 ng/mL). After 3 months of therapy, leptin levels increased in both tribal and non-tribal groups, 4.03±1.05 ng/mL and 4.44±0.86 ng/mL, although tribal levels remained generally lower (Fig. 2).

These findings suggest that malnutrition disproportionately affects tribal communities, which may compromise their immune response and increase their susceptibility to TB.

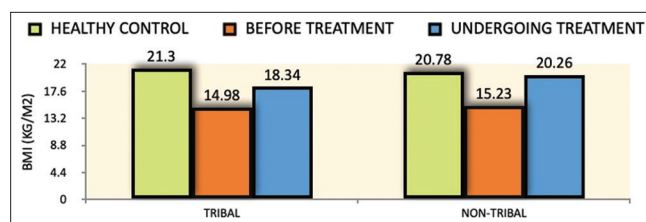


Fig. 1: Comparison of Mean value of Serum Leptin and Pleural Fluid Leptin across three groups, healthy controls and PTB Patients before treatment and undergoing treatment

Table 1: Comparison of Mean value of Serum Leptin, BMI and Pleural Fluid Leptin across three groups, healthy controls and PTB Patients before treatment and undergoing treatment

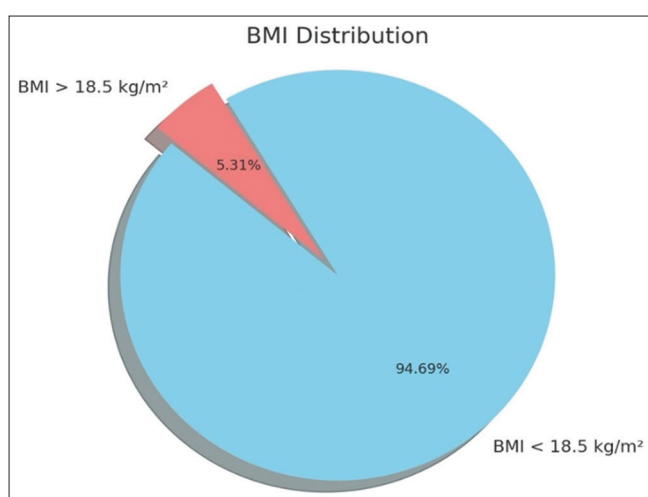
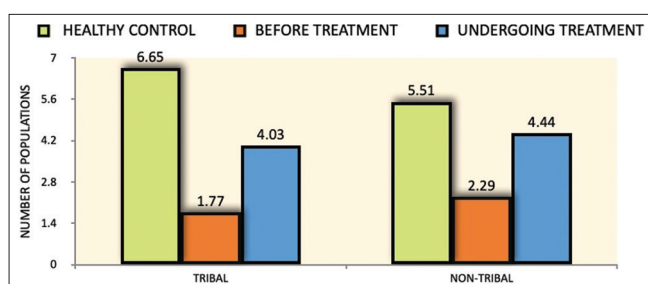
Variables	Control (mean±SD)	Before treatment (mean±SD)	Undergoing treatment (mean±SD)	F-statistic	p-value (<0.05)
BMI (kg/m ²)	21.07±2.64	15.09±2.04	19.19±2.22	358.70	0.000
Serum leptin (ng/mL)	5.79±2.04	1.98±0.72	4.20±1.00	399.6	0.000
Pleural fluid leptin (ng/mL)	-	1.62±1.11	-	-	-

BMI: Body mass index, PTB: Pulmonary tuberculosis, SD: Standard deviation

Table 2: Correlation of BMI Vs Serum Leptin among PTB Patients before treatment and Undergoing treatment

Groups	Parameters	Correlation coefficient (r)	Significance (p-value)
Before treatment	Serum leptin versus BMI	0.26	0.0001
Undergoing treatment	Serum leptin versus BMI	0.136	0.051 NS

BMI: Body mass index, PTB: Pulmonary tuberculosis. *Correlation among BMI and serum leptin levels before treatment and during treatment *p≤0.05, statistically significant. **Highly significant. NS=not significant p>0.05

**Fig. 2: The distribution of body mass index among newly diagnosed pulmonary tuberculosis patients those are falling under malnutrition****Fig. 3: Comparison of mean value of serum leptin among tribal and non-tribal population across three groups, healthy controls, and pulmonary tuberculosis patients before treatment and undergoing treatment**

Narrative summary of key findings (paragraphs)

Table 2 represents the correlation between BMI and Leptin Levels among PTB patients before treatment and Undergoing treatment. Figs. 5 and 6 shows correlation graph between BMI and Leptin level in Pretreatment show strong positive correlation with $r=0.26$, $*p=0.001$

this indicates that anti-tubercular therapy (ATT) markedly enhanced nutritional and metabolic status among PTB patients, as evidenced by significant increases in BMI and serum leptin levels from baseline to the during course of treatment. Despite these gains, patients' post-treatment levels still lagged those of healthy controls, indicating that longer-term nutritional and clinical follow-up is essential.

Tribal communities were notably disadvantaged at the outset, with lower BMIs and leptin levels suggesting severe undernutrition. Although improvement was observed after 3 months of therapy, the tribal cohort continued to exhibit comparatively lower markers than both non-tribal patients and healthy controls. This difference underscores the critical need for comprehensive public health strategies that integrate nutritional support with ongoing TB management, particularly in tribal-dominated areas.

DISCUSSION

According to a recent statewide survey in India, which was done to estimate the burden of TB cases from 2019 to 2021, the crude prevalence of infection among cases involving people aged 15 and older was 31.3%. There were 316 microbiologically verified pulmonary cases in a population of at least 15 years old. An estimated 312 cases of all types of TB were found for every lakh people [10]. This study elucidates the impact of ATT on serum leptin levels in PTB patients from tribal and non-tribal communities in Southern Rajasthan. Notably, the marked differences have been observed among newly diagnosed PTB patients which highlight how socioeconomic status and nutritional disparities can shape immunological responses and treatment outcomes. Malnutrition, particularly prevalent among tribal populations, is well-documented to weaken immunity, thus increasing the risk of TB acquisition and complicating recovery [1]. The recovery of BMI and leptin levels in our cohort underscores the beneficial effect of successful TB therapy in improving systemic nutritional indices – an observation consistent with other investigations linking heightened leptin concentration to immune reconstitution during treatment [5].

Our findings also align with the role of leptin as both an energy-regulating hormone and an immune modulator. Leptin deficiency has been associated with dampened T-lymphocyte responses and impaired macrophage function, rendering patients more susceptible to *Mycobacterium TB* [2]. Nonetheless, it is crucial to note that leptin is not a standalone marker; inflammatory cytokines, other adipokines, and micronutrient levels also influence disease trajectory and recovery [11].

Tribal communities continue to exhibit lower leptin levels and BMI compared to non-tribal counterparts' post-treatment, indicating that 3 months of standardized therapy may not suffice to rectify chronic malnutrition. Cultural practices, limited access to healthcare, and food insecurity likely contribute to the persistence of this nutritional gap. Therefore, incorporating targeted nutritional interventions – ranging from protein supplementation to micronutrient fortification – might further accelerate recovery and improve long-term outcomes [4].

According to research findings given by Mexitalia *et al.*, and Yamborisut *et al.*, before and after treatment, the intensive phase ended, the leptin level rose, and it continued to rise during the continuation period. Our studies also resemble the same findings, longitudinal monitoring has shown that leptin concentrations continue to rise through the

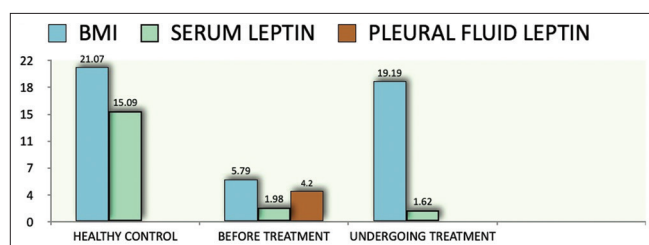


Fig. 4: Comparison of mean value of serum leptin and pleural fluid leptin across three groups, healthy controls and pulmonary tuberculosis patients before treatment and undergoing treatment

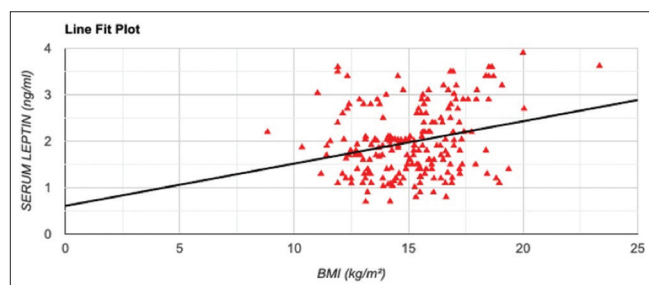


Fig. 5: Correlation of body mass index versus serum leptin among pulmonary tuberculosis patients before treatment ($r=0.26$)

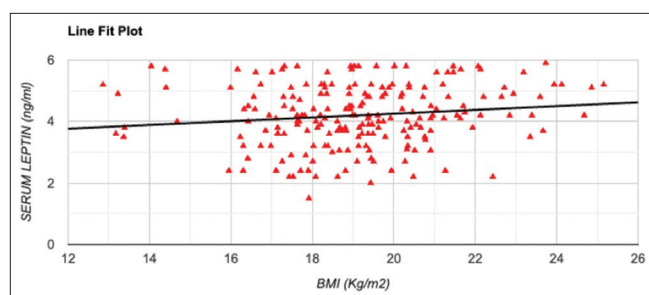


Fig. 6: Correlation of body mass index and serum leptin among pulmonary tuberculosis undergoing treatment ($r=0.136$) not significant

continuation phase (months 3–6) of standard therapy, paralleling progressive gains in BMI and body-fat percentage [12,13]. Similar stepwise increases have been documented in Indonesian pediatric cohorts, where mean leptin values nearly doubled between the second and 6th months of treatment [12,13]. Adult series from Turkey and Europe likewise report 60–90% elevations in circulating leptin over the same interval, correlating with weight restoration and improved immune markers [6,14]. Our findings correlating with the study given by Benoit *et al.*, which confirmed that proinflammatory cytokine production was higher during the continuation phase of therapy, the concentration of leptin increased more during the intense phase due to the reduction in the number of bacteria and the acute phase response [15].

A meta-analysis pooling 12 case–control studies confirmed a significant post-treatment rise in leptin (standardized mean difference=0.70, 95% confidence interval 0.51–0.89) [16], while endocrine-profiling work demonstrates that cytokine-driven hormonal recovery – including leptin – persists throughout the full 6-month regimen [17]. Collectively, these data reinforce the present finding that sustained restoration of leptin is a hallmark of therapeutic success and may serve as a surrogate indicator of nutritional convalescence.

Another consideration is the genetic and physiological diversity among different ethnic groups. Certain polymorphisms in genes regulating leptin production or leptin receptor sensitivity may influence an

individual's vulnerability to TB and response to treatment. Future research could explore how genetic factors, together with socio-economic variables, shape leptin patterns in tribal and non-tribal populations. Furthermore, longitudinal designs extending beyond 3 months would clarify whether leptin normalization persists or further improves over time and whether it correlates with reduced relapse rates.

In conclusion, this study gives compelling evidence that anti-tubercular remedy is useful not only for scientific and microbiological therapy but also for enhancing dietary and metabolic parameters. According to our findings, leptin levels and BMI increased when anti-TB medicine was administered. We concluded that the elevated leptin levels might contribute to patients' increased appetites and improved nutritional intake. A further investigation carried out in Thailand that discovered a positive correlation between leptin levels and BW supports our findings [13]. However, disparities stay, among tribal populations, pointing to the need of included, culturally touchy interventions. Through leveraging leptin as a capacity biomarker for dietary and immune recuperation, clinicians and public health policymakers can refine TB control protocols to cope with every infection management and the critical dimensions of malnutrition.

CONCLUSION

In summary, this study demonstrates that powerful PTB remedy will significantly increase BMI and serum leptin levels, even though each stay decreases than in wholesome controls. Tribal PTB sufferers exhibit a pronounced deficit at baseline, indicating a dual undertaking of infectious ailment and severe undernutrition. These findings emphasize the critical position of incorporated procedures – encompassing anti-tubercular tablets, ordinary nutritional assessments, and culturally tailor-made interventions – to ensure comprehensive care and higher lengthy-time period results, specifically in resource-constrained tribal areas of Southern Rajasthan.

ETHICAL APPROVAL

This study was approved by the Institutional ethical committee with Reference No. STU/IEC/2022/93, Sai Tirupati University, Udaipur, Rajasthan.

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AUTHOR CONTRIBUTION

Supriya, P. Satyanarayan carried out the design of data, acquisition of data, analysis and interpretation of data, statistical analysis of data, research article drafting, and revising the article.

CONFLICTS OF INTEREST

No conflict of interest.

SOURCE OF FUNDING

None.

REFERENCES

1. Flynn JL, Chan J. Immunology of tuberculosis. *Annu Rev Immunol*. 2001;19:93-129. doi: 10.1146/annurev.immunol.19.1.93, PMID 11244032
2. Ahima RS, Flier JS. Leptin. *Annu Rev Physiol*. 2000;62:413-37. doi: 10.1146/annurev.physiol.62.1.413, PMID 10845097
3. World Health Organization. Global Tuberculosis Report. Geneva: World Health Organization; 2022. doi: 10.1016/s2666-5247(22)00359-7
4. Chauhan LS, Tonsing J. Revised national TB control programme in India. *Tuberculosis (Edinb)*. 2005;85(5-6):271-6. PMID 16253562

5. Macallan DC. Malnutrition in tuberculosis. *Diagn Microbiol Infect Dis.* 1999;34(2):153-7. doi: 10.1016/s0732-8893(99)00007-3, PMID 10354866
6. Schwenk A, Hodgson L, Rayner CF, Griffin GE, Macallan DC. Leptin and energy metabolism in pulmonary tuberculosis. *Am J Clin Nutr.* 2003;77(2):392-8. doi: 10.1093/ajcn/77.2.392, PMID 12540399
7. Park HK, Ahima RS. Physiology of leptin: Energy homeostasis, neuroendocrine function and metabolism. *Metabolism.* 2015;64(1):24-34. doi: 10.1016/j.metabol.2014.08.004, PMID 25199978
8. Zheng Y, Ma A, Wang Q, Han X, Cai J, Schouten EG, *et al.* Relation of leptin, ghrelin and inflammatory cytokines with body mass index in pulmonary tuberculosis patients with and without type 2 diabetes mellitus. *PLoS One.* 2013;8(11):e80122. doi: 10.1371/journal.pone.0080122, PMID 24260344
9. Bhargava A, Chatterjee M, Jain Y, Chatterjee B, Kataria A, Bhargava M, *et al.* Nutritional status of adult patients with pulmonary tuberculosis in rural central India and its association with mortality. *PLoS One.* 2013;8(10):e77979. doi: 10.1371/journal.pone.0077979, PMID 24205052
10. Ministry of Health. Family Welfare Government of India. India TB Report; 2023. Available from: <https://tbcindia.mohfw.gov.in/2023/06/06/india-tb-report-2023>
11. Margetic S, Gazzola C, Pegg GG, Hill RA. Leptin: A review of its peripheral actions and interactions. *Int J Obes Relat Metab Disord.* 2002;26(11):1407-33. doi: 10.1038/sj.ijo.0802142, PMID 12439643
12. Mexitalia M, Dewi YO, Pramono A, Anam MS. Effect of tuberculosis treatment on leptin levels, weight gain, and percentage body fat in Indonesian children. *Korean J Pediatr.* 2017;60:118-23. doi: 10.3345/kjp.2017.60.4.118
13. Yamborisut U, Riabroy N, Phonrat B, Tungtrongchitr R. Serum leptin levels and body composition in obese Thai children. *Southeast Asian J Trop Med Public Health.* 2009;40(3):544-52. PMID 19842442
14. Buyukoglan H, Gulmez I, Kelestimur F, Kart L, Oymak FS, Demir R, *et al.* Leptin levels in various manifestations of pulmonary tuberculosis. *Mediators Inflamm.* 2007;2007:64859. doi: 10.1155/2007/64859, PMID 17497033
15. Benoit SC, Clegg DJ, Seeley RJ, Woods SC. Insulin and leptin as adiposity signals. *Recent Prog Horm Res.* 2004;59:267-85. doi: 10.1210/rp.59.1.267, PMID 14749506
16. Ye M, Bian LF. Association of serum leptin levels and pulmonary tuberculosis: A meta-analysis. *J Thorac Dis.* 2018;10(2):1027-36. doi: 10.21037/jtd.2018.01.70, PMID 29607177
17. Tsegaye Y, Admassu W, Edao A, Kinde S, Gentu M, Negash M, *et al.* Alteration of endocrine hormones and antibody responses in different spectrum of tuberculosis disease. *Front Immunol.* 2022 Feb 25;13:849321. doi: 10.3389/fimmu.2022.849321, PMID 35281036