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The role of thymic stromal lymphopoietin (TSLP): A predictive biomarker for severity and poor control in asthmatic Iraqi patients

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ABSTRACT

Thymic stromal lymphopoietin is an epithelial-derived alarmin that triggers and enhances type 2 airway inflammation. In this case–control study, adults with asthma (n=90) and healthy controls (n=90) were enrolled. Patients were subclassified by Global Initiative for Asthma (GINA)-based severity (moderate n=56; severe n=34) and control (controlled n=31; partly controlled n=34; uncontrolled n=25). Serum Thymic stromal lymphopoietin (TSLP) was measured using ELISA technology. Non-parametric comparisons between moderate and severe asthmatic patients were conducted using the Mann–Whitney U-test and comparisons across asthma control categories were conducted using the Kruskal–Wallis with Dunn's post-hoc tests. Receiver operating characteristic (ROC) analysis was used to estimate diagnostic accuracy. Serum TSLP was significantly higher in severe compared with moderate asthma (median 43.0 vs 26.4 pg/mL; $p<0.0001$). Across control categories, TSLP differed significantly ($p<0.0001$), with higher values in partly controlled and uncontrolled asthma compared with controlled asthma (post-hoc: Partly controlled asthma > controlled patients and uncontrolled asthma patients > controlled patients; uncontrolled asthma patients ≈ Partly controlled asthma). ROC analysis showed excellent discrimination between

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patients and healthy controls (AUC=0.907; 95% CI 0.855–0.945). A cut-off >19.21 pg/mL yielded 78.89% sensitivity and 92.22% specificity. High levels of serum TSLP in asthma patients who get worse and lose control suggest that it could be a good biomarker for severe and poorly managed asthma in Iraqi adults. Adding serum TSLP to multi-marker panels may improve phenotyping and risk classification.

Keywords: asthma, thymic stromal lymphopoietin (TSLP), serum biomarker, asthma control, GINA

INTRODUCTION

Asthma is a chronic inflammatory airway illness that affects people in different ways. It causes different respiratory symptoms, makes it hard to breathe, and makes the airways more sensitive[1]. Modern asthma management focuses on both present symptom control and future risks, such as exacerbations, accelerated lung function decline, and treatment-related adverse effects, as outlined in the Global Initiative for Asthma (GINA) framework[2-4].

Thymic stromal lymphopoietin (TSLP) is a short-chain, four α-helical bundle type one interleukin-2 family cytokine that is synthesized by thymic stromal cells. It was initially described in 1994 and shares similarities with IL-7. It is an alarmin cytokine, similar to IL-25 and IL-33. The primary sources of TSLP secretion are cells of the epithelial layer, airway smooth muscle cells, keratinocytes, stromal cells, fibroblasts, mast cells, monocytes and macrophages, granulocytes, and dendritic cells[5,6].

TSLP expression is heightened in asthmatic airways and corresponds with the concentrations of type 2-attractive chemokines and disease severity. It is classified as an “alarmin”, and bronchial connective tissue and immune cells have been shown to secrete TSLP upon exposure to respiratory viruses, pollutants from the air, allergens, and stimulation such as IL-4, IL-13, and (Tumor Necrosis Factor-α)[7]. There is now a substantial body of evidence indicating that TSLP serves as a critical intermediary between immune cells and structural cells in the airway, in addition to its effects on specific immune cells. The airway can undergo airway remodeling as a result of the dysregulation of structural cells in asthma. This process involves the thickening of the reticular basement membrane, goblet cell hyperplasia, subepithelial fibrosis, and hyperplasia[8].

Importantly, data regarding serum TSLP levels in Middle Eastern populations are scarce. Given the potential influence of genetic background, environmental exposures, and healthcare practices on inflammatory profiles, population-specific evaluation of serum TSLP is warranted. Moreover, determining whether circulating TSLP reflects clinically relevant outcomes such as disease severity and asthma control may enhance its utility as a non-invasive biomarker in routine clinical practice[9–11]. Therefore, the present study aimed to evaluate serum TSLP levels in adult Iraqi patients with asthma and to investigate their association with disease severity and level of asthma control.

METHODOLOGY

Study design and population

This study was designed as a case-control investigation conducted at the Chemistry and Biochemistry Department, College of Medicine, Al-Nahrain University, Baghdad, Iraq. Adult patients with asthma were consecutively obtained from outpatient clinics and at Al-Imamin Alkadmeen Hospital City, alongside age- and sex-matched apparently healthy controls. The diagnosis of asthma was determined in accordance with the GINA guidelines[12], which are predicated on objective evidence of changing airflow limitation and characteristic symptoms. A total of 90 asthmatic patients and 90 healthy controls were enrolled in the study. Asthmatic patients were further stratified according to disease severity and level of asthma control, allowing evaluation of serum TSLP across clinically relevant subgroups.

Clinical classification of asthmatic patients

Patients were classified according to the severity and the controlling status of asthma.

Asthma severity

Asthma severity was classified retrospectively according to GINA recommendations, based on the minimum treatment required to achieve disease control. Patients were categorized into:

- **Moderate asthma (n=56)**
- **Severe asthma (n=34)**

as defined by the need for high-dose inhaled corticosteroids with additional controller therapy to maintain control or persistent lack of control despite optimized treatment[13]the level of control must be monitored on a periodic basis to determine whether therapy should be maintained or adjusted (stepped up if necessary, stepped down if possible.

Asthma control

Asthma control was assessed using GINA symptom-based criteria, incorporating daytime symptoms, nocturnal awakenings, activity limitation, and reliever medication use. Patients were classified into:

- **Controlled asthma (n=31)**
- **Partly controlled asthma (n=34)**
- **Uncontrolled asthma (n=25)**

Asthma Control Test (ACT) scores were used as supportive clinical indicators where applicable[14].

Inclusion and exclusion criteria

Adults aged 18–70 years with a confirmed diagnosis of asthma were eligible for participation. Exclusion criteria included diabetes mellitus, chronic kidney or liver disease, chronic obstructive pulmonary disease, acute respiratory infections, autoimmune or systemic inflammatory diseases, active smoking, pregnancy, and recent systemic corticosteroid use within four weeks before enrollment, to minimize confounding inflammatory effects[15].

Blood sampling and laboratory analysis

Venous blood samples of (five milliliters) were obtained under aseptic conditions. The samples were centrifuged at 3000 rpm for 15 minutes after being allowed to coagulate. The serum was separated and stored at -80°C until it was analyzed. The manufacturer's instructions were followed to quantify serum TSLP concentrations using a commercially available enzyme-linked immunosorbent assay ELISA kit from China. The TSLP concentrations were determined from standard calibration curves, and the optical density was detected at 450 nm using a reader for microplates.

Ethical considerations

The Institutional Review Board (IRB) of the College of Medicine, Al-Nahrain University granted ethical approval for the study on March 10, 2025, under decision number 225/3/2. The nature and objectives of the investigation were disclosed to all participants, and verbal informed consent was obtained before recruitment. All procedures complied with the ethical norms outlined in the Declaration of Helsinki.

Statistical analysis

Data distribution was assessed for normality by the Shapiro-Wilk test and found to be non-normally distributed. Continuous variables were expressed as mean

± standard deviation and median with interquartile range (IQR). Comparisons between two groups (moderate vs severe asthma) were performed using the Mann–Whitney U test. Comparisons across more than two groups (asthma control categories) were conducted using the Kruskal–Wallis test followed by Dunn's post-hoc analysis[16,17].

Receiver operating characteristic (ROC) curve analysis was applied to assess the diagnostic and discriminatory performance of serum TSLP, including area under the curve (AUC), optimal cut-off value, sensitivity, specificity, and 95% confidence intervals. A p-value<0.05 was considered statistically significant[18].

RESULTS and DISCUSSION

Baseline characteristics

The study included 180 participants (90 asthmatic patients and 90 healthy controls). No significant differences were observed between groups with respect to age, Body Mass Index (BMI), and sex distribution as illustrated in Tables 1 and 2.

Table 1. Sex distribution of study population

Gender	Patient (n, %)	Controls (n, %)	Chi-square	p-value
Male	35 (38.9%)	37 (41.1%)	0.023	0.879
Female	55 (61.1%)	53 (58.9%)	-	-

Legend: Data presented as frequency (n) and percentage (%). Statistical comparison performed using the Chi-square test.

Table 2. Baseline demographic and clinical characteristics of asthmatic patients and healthy controls

Parameter	Control (n=90)	Patient (n=90)	Statistical Analysis
Age (years)			
Median	49.00	45.00	p=0.265
Mean ± SD	48.30 ± 12.69	46.00 ± 14.87	
SEM	1.338	1.567	
BMI (kg/m²)			
Median	29.35	29.90	p=0.242
Mean ± SD	29.36 ± 3.43	30.16 ± 5.55	
SEM	0.362	0.585	

Serum TSLP levels in asthma versus healthy controls

Serum TSLP concentrations were significantly higher in asthmatic patients (Mean = 31.32 ± 12.72 pg/mL; Median = 30.10 pg/mL) compared to controls (Mean = 13.87 ± 3.92 pg/mL; Median = 14.06 pg/mL; $p<0.001$) which represents more than a twofold elevation in the asthmatic group as illustrated in Table 3.

Table 3. Comparison of serum TSLP levels between asthmatic patients and controls

Parameter	Patient (n=90)	Control (n=90)	Statistical Analysis
TSLP pg/mL			
Median	30.1	14.06	p<0.001
Mean ± SD	31.32 ± 12.72	13.87 ± 3.91	
SEM	1.341	0.413	

Serum TSLP levels according to asthma severity

Results illustrated in Table 4 and Figure 1 showed that TSLP levels were significantly higher in severe asthma (Median = 43.0 pg/mL; Mean = 39.8 pg/mL) than in moderate asthma (Median = 26.4 pg/mL; Mean = 26.2 pg/mL; $p<0.0001$). This corresponds to a 51.9% increase (≈ 1.52-fold) in the mean concentration.

Table 4. Serum TSLP levels in moderate versus severe asthma

Parameter	Severe (n=34)	Moderate (n=56)	p-value
TSLP pg/mL			
Median (IQR)	43.0 (23.6)	26.4 (16.1)	<0.0001
25^{th}–75^{th}	28.6 – 52.2	17.9 – 33.9	
Mean ± SD	39.8 ± 12.5	26.2 ± 9.8	

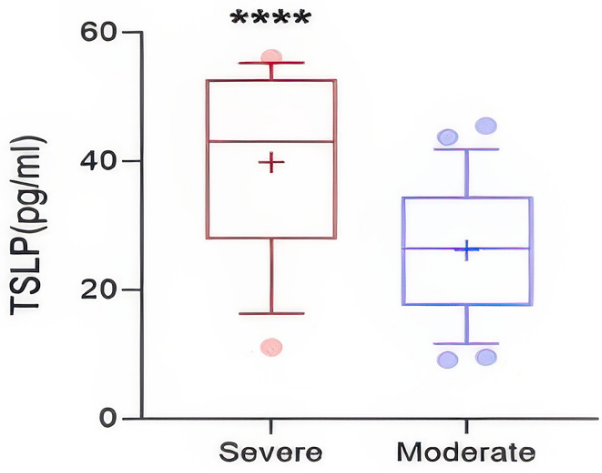

Figure 1. Box-plot representation of serum TSLP according to asthma severity

Serum TSLP according to asthma control

Results demonstrated in Table 5 and Figure 2 showed that serum TSLP levels differed significantly across asthma control categories (Kruskal–Wallis, $p<0.0001$). Patients in the partially controlled group exhibited the highest TSLP concentrations (median = 38.9 pg/mL, IQR = 11.5), followed by the uncontrolled group (median = 31.8 pg/mL, IQR = 18.3), whereas the controlled group showed the lowest levels (median = 19.7 pg/mL, IQR = 10.9). Post-hoc analysis demonstrated that partially controlled > controlled and uncontrolled > controlled.

Table 5. Serum TSLP levels according to asthma control status

Parameter	Controlled (n=31)	P. Controlled (n=34)	Uncontrolled (n=25)	p-value
TSLP pg/mL				
Median (IQR)	19.7 (10.9) a	38.9 (11.5) b	31.8 (18.3) b	<0.0001
25^{th}–75^{th}	14.54 - 25.4	33.5 - 45.0	24.9 - 43.3	
Mean ± SD	20.4 ± 7.7	39.2 ± 9.1	33.9 ± 12.7	

Values with similar letters non-significantly different.

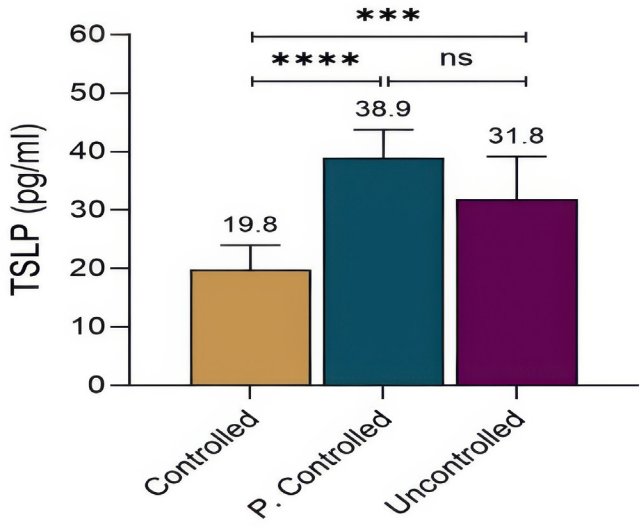

Figure 2. Levels of serum TSLP across asthma control categories

Serum TSLP according to ICS/LABA uses

Serum TSLP levels were compared between asthmatic patients receiving inhaled corticosteroid/long-acting β2-agonist (ICS/LABA) therapy and those not receiving ICS/LABA treatment (Table 6). Patients using ICS/LABA (n=68) demonstrated lower serum TSLP concentrations compared with non-users (n=22). The median TSLP level in ICS/LABA users was 28.51 pg/mL (IQR = 19.5), whereas non-users exhibited a higher median level of 36.97 pg/mL (IQR = 11.4). Similarly, the mean ± SD of serum TSLP was lower in the ICS/LABA group (30.1 ± 12.9 pg/mL) compared with patients not receiving ICS/LABA therapy (35.2 ± 11.3 pg/mL). However, this difference did not reach statistical significance (p=0.077).

Table 6. Serum level of TSLP of asthmatic patients at ICS/LABA use

Parameter	Yes (n=68)	No (n=22)	p-value
TSLP pg/mL			
Median (IQR)	28.51 (19.5)	36.97 (11.4)	0.077
25^{th}–75^{th}	19.6 - 39.1	29.4 - 40.8	
Mean ± SD	30.1 ± 12.9	35.2 ± 11.3	

Diagnostic performance of serum TSLP

ROC curve analysis as illustrated in Figure 3, demonstrated excellent diagnostic accuracy of serum TSLP for distinguishing asthmatic patients from healthy controls (AUC = 0.907; 95% CI 0.855–0.945). A cut-off value >19.21 pg/mL provided high specificity (92.22%) and acceptable sensitivity (78.89%).

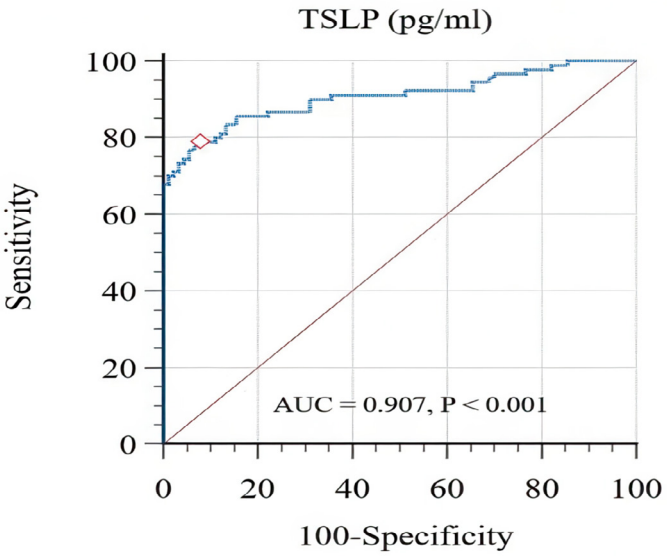

Figure 3. Receiver operating characteristic (ROC) curve for (TSLP) in classifying patients from controls

The present study demonstrates that serum TSLP is significantly elevated in adult Iraqi patients with asthma and is strongly associated with both disease severity and loss of asthma control. These findings support the concept that circulating TSLP reflects upstream epithelial inflammatory activity and may serve as a clinically relevant biomarker in adult asthma[19].

The marked elevation of serum TSLP in severe asthma compared with moderate disease aligns with mechanistic evidence indicating persistent epithelial injury and continuous alarmin release in severe phenotypes[20]. TSLP is known to amplify dendritic cell–mediated Th2 polarization and ILC2 activation, leading to enhanced production of IL-5 and IL-13, eosinophilic inflammation, and airway remodeling—hallmarks of severe asthma[21,22].

Importantly, serum TSLP levels also increased with worsening asthma control. Patients with partly controlled and uncontrolled asthma exhibited

significantly higher TSLP concentrations than controlled patients. This observation suggests that inadequate symptom control is associated with persistent epithelial activation and ongoing inflammatory signaling, even in patients receiving controller therapy. Similar patterns have been described in adult asthma cohorts, where circulating TSLP remained elevated despite inhaled corticosteroid treatment, potentially reflecting partial corticosteroid insensitivity of epithelial alarmin pathways[22,23].

The excellent diagnostic performance of serum TSLP (AUC 0.907) observed in this study further supports its clinical relevance. High specificity at the identified cut-off value indicates that elevated serum TSLP may serve as a reliable "rule-in" biomarker for asthma, particularly in settings where access to spirometry or advanced airway sampling is limited. Nevertheless, the moderate sensitivity underscores that TSLP should be interpreted within a broader biomarker context rather than as a standalone diagnostic tool.

Discrepancies among published studies regarding serum TSLP levels may be attributed to differences in age groups, disease duration, assay platforms, and population characteristics. Pediatric studies, for example, reported elevated serum TSLP in asthma but inconsistent correlations with lung function or phenotype[24,25]. In contrast, adult cohorts, particularly those enriched for moderate-to-severe disease, appear more likely to demonstrate clinically meaningful associations between circulating TSLP and disease burden[11,21].

The present study adds novel population-specific evidence from Iraq, a region in which biomarker-based asthma phenotyping remains underexplored. By demonstrating consistent associations between serum TSLP, disease severity, and asthma control, these findings support the integration of TSLP into multi-marker strategies aimed at risk stratification and personalized asthma management. Moreover, the results provide biological plausibility for the observed efficacy of anti-TSLP biologic therapies across diverse asthma phenotypes[26,27].

In conclusion, serum TSLP is significantly elevated in adult asthma and increases with both disease severity and poor asthma control. Its strong discriminatory performance supports its potential role as a predictive biomarker reflecting upstream epithelial inflammatory activity. These findings reinforce the biological and clinical relevance of TSLP in adult asthma and support further exploration of serum TSLP within biomarker-guided management strategies.

STATEMENT OF ETHICS

The Institutional Review Board (IRB) of the College of Medicine, Al-Nahrain University granted ethical approval for the study on March 10, 2025, under decision number 225/3/2. The nature and objectives of the investigation were disclosed to all participants, and verbal informed consent was obtained before recruitment. All procedures complied with the ethical norms outlined in the Declaration of Helsinki.

CONFLICT OF INTEREST STATEMENT

The authors have no conflicts of interest to declare.

AUTHOR CONTRIBUTIONS

Design and planning, RJM, HAA; data collection, AYS, HAA; data analysis, AYS, RJM; literature search and writing, AYS, RJM; supervision, RJM, HAA; editing and proofreading, RJM, HAA.

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REFERENCES

1. Hoque F. Asthma management essentials: GINA recommendations key takeaways. J Integr Heal, 2024;3(1):185–187. Doi: 10.51219/JIH/Farzana-Hoque/32

2. Tho NV, Quan VTT, Dung DV, Phu NH, Dinh-Xuan AT, Lan LTT. GINA implementation improves asthma symptoms control and lung function: a five-year real-world follow-up study. J Pers Med, 2023;13(5):809. Doi: 10.3390/jpm13050809

3. Abashar IBH, Salih OAMM, Joda TAA, Alsedig MFA, Omer AT, Ahmed HA, et al. Assessment of the implementation of global initiative for asthma (GINA) 2019 guidelines for treatment of mild asthma among pediatric registrars: a quasi-interventional study. J Asthma Allergy, 2024;17:339–347. Doi: 10.2147/JAA.S442573

4. Muhsen SA, AL-Saeed HH, ALOmrani AAA. Study of the association between prolidase enzyme kinetics and severity of asthma in children. Indian J Public Heal Res Dev, 2019;10(6):836–842. Doi: 10.5958/0976-5506.2019.01383.4

5. Mitchell PD, O'Byrne PM. Biologics and the lung: TSLP and other epithelial cell-derived cytokines in asthma. Pharmacol Ther, 2017;169:104–112. Doi: 10.1016/j.pharmthera.2016.06.009

6. Verstraete K, Peelman F, Braun H, Lopez J, Van Rompaey D, Dansercoer A, et al. Structure and antagonism of the receptor complex mediated by human TSLP in allergy and asthma. Nat Commun, 2017;8:14937. Doi: 10.1038/ncomms14937

7. Roan F, Obata-Ninomiya K, Ziegler SF. Epithelial cell-derived cytokines: more than just signaling the alarm. J Clin Invest, 2019;129(4):1441–1451. Doi: 10.1172/JCI124606

8. Fehrenbach H, Wagner C, Wegmann M. Airway remodeling in asthma: what really matters. Cell Tissue Res, 2017;367:551–569. Doi: 10.1007/s00441-016-2566-8

9. Ko H-K, Cheng S-L, Lin C-H, Lin S-H, Hsiao Y-H, Su K-C, et al. Blood tryptase and thymic stromal lymphopoietin levels predict the risk of exacerbation in severe asthma. Sci Rep, 2021;11:8425. Doi: 10.1038/s41598-021-86179-1

10. Alturaiki W. High plasma levels of the TSLP cytokine in Saudi patients with chronic stable asthma. J King Saud Univ Sci, 2022;34:102271. Doi: 10.1016/j.jksus.2022.102271

11. Ranjbar M, Matloubi M, Assarehzadegan M-A, Fallahpour M, Sadeghi F, Soleyman-Jahi S, et al. Association between two single nucleotide polymorphisms of thymic stromal lymphopoietin (TSLP) gene and asthma in Iranian population. Iran J Allergy Asthma Immunol, 2020;19(4):362–372. Doi: 10.18502/ijaai.v19i4.4111

12. Bateman ED, Hurd SS, Barnes PJ, Bousquet J, Drazen JM, FitzGerald JM, et al. Global strategy for asthma management and prevention: GINA executive summary. Eur Respir J, 2008;31(1):143–178. Doi: 10.1183/09031936.00138707

13. Bleecker ER, Al-Ahmad M, Bjermer L, Caminati M, Canonica GW, Kaplan A, et al. Systemic corticosteroids in asthma: a call to action from World Allergy Organization and Respiratory Effectiveness Group. World Allergy Organ J, 2022;15(12):100726. Doi: 10.1016/j.waojou.2022.100726

14. Reddel HK, Bacharier LB, Bateman ED, Brightling CE, Brusselle GG, Buhl R, et al. Global initiative for asthma strategy 2021: executive summary and rationale for key changes. Am J Respir Crit Care Med, 2022;205(1):17–35. Doi: 10.1164/rccm.202109-2205PP

15. Dobler CC, Morrow AS, Beuschel B, Farah MH, Majzoub AM, Wilson ME, et al. Pharmacologic therapies in patients with exacerbation of chronic obstructive pulmonary disease: a systematic review with meta-analysis. Ann Intern Med, 2020;172(6):413–422. Doi: 10.7326/M19-3007

16. Manna MJ, Baqir LS, Abdulamir HA. Verapamil versus tamoxifen in treatment of experimentally induced infertility. J Res Pharm, 2024;28(5):1485–1491. Doi: 10.29228/jrp.826

17. Jarari AM, Farag AMMM, Abdulamir HA, Pathak RM. Ferritin levels and erythrocyte lipid peroxidation in acute myocardial infarction in diabetic patients. Jamahiriya Med J, 2010;10(2):101–105.

18. Çorbacıoğlu ŞK, Aksel G. Receiver operating characteristic curve analysis in diagnostic accuracy studies: a guide to interpreting the area under the curve value. Turk J Emerg Med, 2023;23(4):195–198. Doi: 10.4103/tjem.tjem_182_23

19. García-Reyes MM, Zumaya-Pérez LC, Pastelin-Palacios R, Moreno-Eutimio MA. Serum thymic stromal lymphopoietin (TSLP) levels in atopic dermatitis patients: a systematic review and meta-analysis. Clin Exp Med, 2023;23:4129–4139. Doi: 10.1007/s10238-023-01147-5

20. Parnes JR, Molfino NA, Colice G, Martin U, Corren J, Menzies-Gow A. Targeting TSLP in Asthma. J Asthma Allergy, 2022;15:749–765. Doi: 10.2147/JAA.S275039

21. Smolinska S, Antolín-Amérigo D, Popescu F-D, Jutel M. Thymic Stromal Lymphopoietin (TSLP), its isoforms and the interplay with the epithelium in allergy and asthma. Int J Mol Sci, 2023;24(16):12725. Doi: 10.3390/ijms241612725

22. Ebina-Shibuya R, Leonard WJ. Role of thymic stromal lymphopoietin in allergy and beyond. Nat Rev Immunol, 2023;23:24–37. Doi: 10.1038/s41577-022-00735-y

23. Suzukawa M, Ohta K, Tashimo H, Narumoto O, Hashimoto H, Kita T, et al. High serum TSLP is characteristic of late-onset, long-duration, eosinophilic asthma. Allergy, 2026;81(2):402–412. Doi: 10.1111/all.70109

24. Orzołek I, Kowalczyk P, Rybka-Frączek A, Storman M, Dąbrowska M. Thymic stromal lymphopoietin expression in different biological specimens in asthma: a systematic review and meta-analysis. Front Allergy, 2025;6:1703989. Doi: 10.3389/falgy.2025.1703989

25. Vrsalović R, Korošec P, Štefanović IM, Bidovec-Stojkovič U, Čičak B, Harjaček M, et al. Value of thymic stromal lymphopoietin as a biomarker in children with asthma. Respir Med, 2022;193:106757. Doi: 10.1016/j.rmed.2022.106757

26. Matera MG, Rogliani P, Calzetta L, Cazzola M. TSLP inhibitors for asthma: current status and future prospects. Drugs, 2020;80(5):449–458. Doi: 10.1007/s40265-020-01273-4

27. Gauvreau GM, O'Byrne PM, Boulet L-P, Wang Y, Cockcroft D, Bigler J, et al. Effects of an anti-TSLP antibody on allergen-induced asthmatic responses. N Engl J Med, 2014;370(22):2102–2110. Doi: 10.1056/NEJMoa1402895