Al-Kindy College Medical Journal (KCMJ)

Research Article

Assessment of Interleukin-30 in Iraqi Female Patients with Hashimoto's Thyroiditis

Nadia H. Mohammed[1], Anmar Al-Taie[2*], Sinan B. Alrifai[3]

[1] Department of Microbiology, College of Medicine, Mustansiriyah University, Baghdad, Iraq
[2] Clinical Pharmacy Department, Faculty of Pharmacy, Istinye University, Istanbul, Türkiye
[3] Supervision and Scientific Evaluation Authority, Ministry of Higher Education and Scientific Research, Baghdad, Iraq
* Corresponding author's email: altaiianmar@gmail.com

Article history:
Received 20 May 2025
Accepted 20 October 2025
Available online 1 April 2026

DOI: 10.47723/yayh3b98

Keywords: Autoimmune thyroiditis; Hashimoto's thyroiditis; Interleukin-30; Thyroid gland

ABSTRACT

Background: Hashimoto's thyroiditis is a chronic lymphocytic autoimmune thyroid disease that results in gradual destruction of the thyroid gland. Interleukin-30 (IL-30) is one of the interleukins that controls inflammatory response since it enhances T-reg cells' suppressive function and induces IL-10 production. The aim of this study was to assess the serum level of IL-30 as an indicator for understanding the pathology of this autoimmune disorder.
Subjects and Methods: A single-center, case-control study was carried out on Hashimoto's Thyroiditis patients who were admitted to the department of medicine at Al-Yarmook Teaching Hospital and Baghdad Teaching Hospital, Baghdad, Iraq, between January 2024 and June 2024. Serum levels of thyroid-stimulating hormone (TSH), antithyroglobulin antibody (anti-TG Abs), antithyroid peroxidase antibody (TPO Ab), and IL-30 were measured in control (n=21) and patient (n=40) groups, all are determined by sandwich enzyme-linked immunosorbent assay (ELISA) kits.
Results: Serum TSH, TPO Ab, and TG Ab are significantly higher in the patient group ($p<0.0001$), whereas serum IL-30 decreases significantly ($p<0.0001$). There was a significant correlation between serum IL-30 and serum TPO Abs levels ($p=0.001$), while there was a negative correlation between the severity of hypothyroidism and serum IL-30 ($P<0.05$).
Conclusion: Serum IL-30 level is associated with the pathogenesis of autoimmune thyroiditis. There is a negative correlation between serum IL-30 and the degree of hypothyroidism. Moreover, a decrease in serum IL-30 level can be considered a predictor for autoimmune thyroiditis.

Introduction

Triiodothyronine [T3] and thyroxin [T4] are produced by the thyroid gland in response to thyroid-stimulating hormone [TSH] that is produced by the pituitary gland. These two hormones can regulate metabolism, growth, and development [1]. Hashimoto's Thyroiditis is the commonest chronic lymphocytic autoimmune thyroid disease in iodine- sufficient states, characterized by an inflammatory process with profound lymphocytic inflammation. Its prevalence ranges from 0.3 to 1.5 per 1000 people, and a female-to-male ratio of 7-10:1 [2]. In Iraq, females form about 82% of patients, and the prevalence seems to be the same as the global prevalence [3].

The thyroid's lymphocyte infiltration is the hallmark of this disease, which slowly destroys the glandular tissue. This decrease in thyroxin production, hence, leads to the development of autoantibodies against thyroglobulin [TG], a glycoprotein crucial to thyroxin synthesis, and thyroid peroxidase [TPO] [4]. Genetic predisposition, along with

environmental factors, plays a part in the occurrence of this condition, even if the exact immune mechanism of attack on thyroid tissue is unclear. The outcome of these circumstances is loss of tolerance to self-antigens.

Cellular immunity contributes to thyroid autoimmune etiology and the subsequent production of autoantibodies [5]. Thyroid-specific antigens, particularly TG and TPO, serve as a source of self-antigenic peptides. Native T cells are activated and undergo clonal growth when antigen-presenting cells [APCs], mainly dendritic cells, expose thyroid-specific autoantigens to native T cells. Cytotoxic T cells, macrophages, and B lymphocytes penetrate and gather in the thyroid gland, whereas auto-reactive T and B cells develop in the draining lymph nodes [6,7].

Thyrocytes are eventually lost due to several immune destruction mechanisms, including antibody-dependent, cytokine-mediated, and apoptotic cytotoxicity. This loss is caused by the generation of auto-reactive T cells, B cells, and antibodies, which results in thyroiditis and hypothyroidism. Cytokines, and interleukins play a crucial role in the development of various chronic immune and inflammatory diseases. They maintain the physiological and biological activities of tissues and cells with a proper body immune state. Over- or under-expression of these immune mediators may precipitate inflammatory reactions or autoimmune disorders [8]. Interleukin 30 (IL-30), also known as IL27-p28, may modulate immune response. It antagonizes interleukin 6 (IL-6) when it attaches to the soluble interleukin 6 receptor (IL-6-R) and thus suppresses the production of Th17 and Th1 cells from native T cells [9]. IL-30 also antagonizes GP130 signaling by binding to GP130. This will affect the signalling of many cytokines, such as IL-6, interleukin 11 (IL-11), and interleukin 27 (IL-27). Therefore, IL-30 controls the inflammatory response and immune-reactive T cells, as it enhances Treg suppressive function and induces IL-10 production [10]. Other interleukins (IL-6, IL-17, IL-23) were assessed in previous studies [11,12]. Accordingly, IL-30 may play a regulatory role as a modulator of cytokine and interleukin balance, with particular attention to its association with Hashimoto's thyroiditis compared to other diseases. From this perspective, the aim of this study was to assess serum IL-30 levels in patients with autoimmune thyroiditis, which may serve as a valuable indicator of the pathogenesis of autoimmune thyroid disease and as a potential diagnostic biomarker reflecting disease activity.

Subjects and Methods

This was a case-control study enrolled among patients whom newly diagnosed with autoimmune thyroiditis. The study was performed at the outpatient clinic department of medicine and surgery at Al-Yarmook Teaching Hospital and Baghdad Teaching Hospital, Baghdad, Iraq, between January and June 2024. The study was approved by the Research Ethics Committee, College of Medicine, Mustansiriyah University, Baghdad, Iraq (Ref. no. 67/17.02.2025).

Patients attending the outpatient department of medicine and surgery clinic who met the inclusion criteria were eligible to enroll in this study. Every participant who agreed to enroll in this study was fully informed about the study's purpose prior to enrollment and completed a written informed consent form. Inclusion criteria included patients over the age of 18 years with a new diagnosis of autoimmune thyroiditis confirmed by clinical examination under the supervision of medical specialists, serological evaluation confirmed by laboratory tests of increased TSH and TPO & TG antibodies, and histopathological diagnosis confirmed by a medical report of lymphocytic thyroiditis following thyroidectomy. Patients taking systemic immune-suppressive medications or chemotherapy, pregnant women, patients with malignancy, patients with other autoimmune diseases and thyroid diseases (De Quervain's thyroiditis, Riedle's thyroiditis, radaition induced thyroiditis), and those with chronic diseases were all excluded.

Baseline demographic and medical data of the participants were collected from the medical records to draw up a detailed clinical data for the study participants, which included age, family history of thyroid disorders, including autoimmune thyroiditis, and the presence of comorbidities. The participants were divided into 2 groups, in which the first one was designated as a control group, included twenty-one apparently healthy participants with no diagnosis of autoimmune thyroid disease or other autoimmune or chronic medical conditions. The second group designated as patients group, included forty patients with only new diagnosis of autoimmune thyroid disease without other autoimmune or chronic medical conditions. The patients group was subdivided into three groups according to the readings of serum TSH level: Group I based on serum TSH level 0.45-4.5 mU/L (normal); Group II based on serum TSH level 4.5-10 mU/L (mild underactive thyroid or subclinical hypothyroidism); and Group III based on serum TSH level >10 mU/L (overt hypothyroidism).

A venous blood sample (5 ml) was withdrawn from each individual aseptically. Samples of blood were poured into a gel tube, allowed to clot at 37°C for 20 to 30 minutes, and then centrifuged for 15 minutes at 1500 g to separate serum. Serum samples were stored in an Eppendorf tube at -20°C.

Participants within both groups underwent an assessment of serum levels of thyroid-stimulating hormone (TSH), T3, T4, antithyroglobulin antibody (ATGA), antithyroid peroxidase antibody (TPO Ab), and IL-30, which were quantitatively assessed using the sandwich enzyme-linked immunosorbent assay (ELISA) kits (Catalogue no. MBS3800854 for antithyroid peroxidase Ab TPO, Catalogue no. MBS268340 for antithyroglobulin antibody TG, Catalogue no. ab100660 for thyroid-stimulating hormone TSH, and Catalogue no. MBS269915 for IL-30, MyBiosource USA). The used kits have detection ranges of 0.1-200 IU/mL for anti-TPO Ab, 1.5-100 IU/mL for ATGA, and 78-5000 pg/mL for IL-30.

Microsoft Office Excel 2017 and the Statistical Package for the Social Sciences (SPSS) version 23.0 were used to analyze the data of the study. Data were assessed using Shapiro test and were normally distributed. While data were expressed as numbers and percentages, numerical variables were expressed as mean and standard deviation. The differences between the control and patient groups were assessed using an independent t-test. Significant differences among means were assessed using a one-way ANOVA and the least significant differences (LSD) post hoc test. The chi-square test was performed to determine significant variations in proportions. P-values < 0.05 are considered statistically significant.

Results

All of the enrolled participants were females. Out of the enrolled participants who met the inclusion criteria of the study, 21 participants were identified within the control group with a mean age of 38 ± 10.01 years, and 40 participants were within the patient group with a mean age of 38.5 ± 7.28 years.

Table 1 compares the serum levels of different biochemical parameters related to thyroid function between the control and patients group. There were statistically significantly higher levels in these parameters between the patient group compared to the control group regarding serum TSH hormone (6±2.35 vs. 2.6±0.59 mU/L, P<0.0001); serum TPO Ab (142±64.75 vs. 5±1.57 IU/L, P<0.0001); and serum TG Ab (72±32.13 vs. 3±1.60, P<0.0001). However, there was a statistically significant difference in serum IL-30 levels between the patient and control groups (109.6±57.52 vs. 537±83.53 pg/mL, P<0.0001). Although statistically non-significant, patients within the age group above 41 years (42.5%) were more susceptible to Hashimoto's Thyroiditis, as shown in Table 2.

Table 1: Changes in serum levels of different biochemical parameters related to thyroid function between the control and patient group

Variable	Control Group N=21 Mean± SD	Patient Group N=40 Mean± SD	P-value
Serum TSH mU/L	2.61±0.59	6.12±2.35	0.0001***
Serum TPO IU/ml	5.00±1.57	142.21±64.75	0.0001***
Serum TG IU/ml	3.05±1.60	72.11±32.13	0.0001***
Serum IL-30 (pg/mL)	537.12±83.53	109.60±57.52	0.0001***

Table 2: Distribution of patients' age groups that are more susceptible to Hashimoto's Thyroiditis

Patients' Age (years)	Patients' group N= (%)	P-value
≤ 35	13 (32.50)	
36-40	10 (25.00)	0.40 NS†
≥41	17 (42.50)	

NS=non-significant; †=Chi-square test

Table 3 shows the association between serum autoantibodies and the different TSH level categories. There was a significant decrease in serum TPO Ab levels with increasing serum TSH levels (192.60±5.98 IU/L, 113.88±60.71 IU/L, 78.00±66.68 IU/L; P<0.05). Similarly, the decrease in serum TG Abs levels was significant, accompanied by an increase in serum TSH levels (11.10±2.10 IU/L, 6.07±1.34 IU/L, 3.86±0.71 IU/L; P<0.05). Likewise, serum IL-30 levels were also significantly decreased with an increase in serum TSH levels (171.98±66.67 pg/mL, 136.30±56.3 pg/mL, 99.13±9.33 pg/mL; P<0.05).

The study findings also revealed a statistical correlation between serum levels of TPO Abs, anti-TG Abs, and IL-30. A statistically significant correlation was noticed between serum levels of IL-30 and TPO Abs (P=0.001), as shown in Table 4 and Figures 1 and 2.

The heat map (Figure 3) illustrates the correlation between serum IL-30 and thyroid function parameters. IL-30 showed a negative correlation with TSH and a positive correlation with T3 and T4, indicating that reduced IL-30 is associated with hypothyroidism severity. IL-30 demonstrated an inverse association with TG antibodies, while the correlation with TPO antibodies was weak, suggesting that autoantibody titers do not consistently reflect thyroid dysfunction. Strong negative correlations between TSH and thyroid hormones (T3, T4) and positive correlations between TPO and TG antibodies were observed, consistent with expected physiological and immunological relationships. Overall, the data support a potential protective role of IL-30 in maintaining thyroid function and modulating autoimmune activity.

Table 3: Association of serum thyroid-stimulating hormone with the serum autoantibodies

Serum TSH	Patient Group Serum TPO (Mean± SD)	Patient Group Serum TG (Mean± SD)	Patient Group Serum IL-30 (Mean± SD)
0.45-4.5 mU/L	192.60±5.98^{a}	11.10±2.18^{a}	171.98±66.67^{a}
4.5-10 mU/L	113.88±60.71^{b}	6.07±1.34^{b}	136.30±56.30^{ab}
>10 mU/L	78.00±66.68^{b}	3.86±0.71^{c}	99.13±9.33^{b}
LSD	71.81	1.79	68.02

Means with a different letter in the same column are significantly different (P< 0.05)

Table 4: Pearson correlation between serum autoantibodies and IL-30

Parameter	TG	IL-30
TPO	0.35	-0.49
P-value	0.02	0.001
TG		-0.19
P-value		0.24

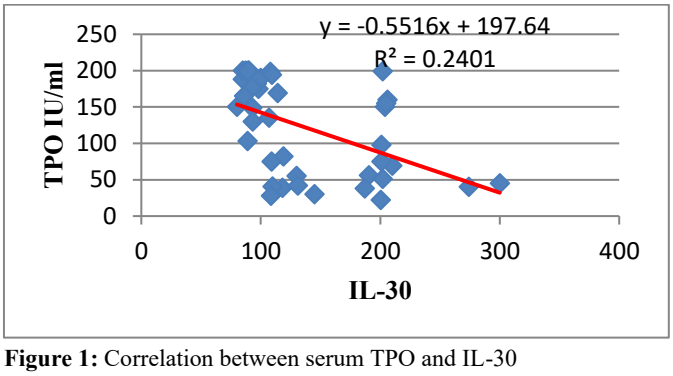

Figure 1: Correlation between serum TPO and IL-30

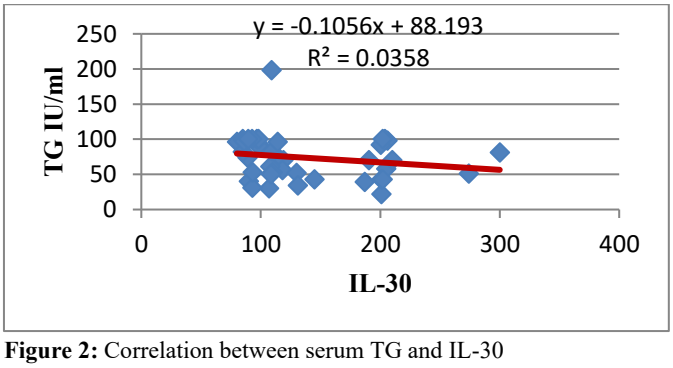

Figure 2: Correlation between serum TG and IL-30

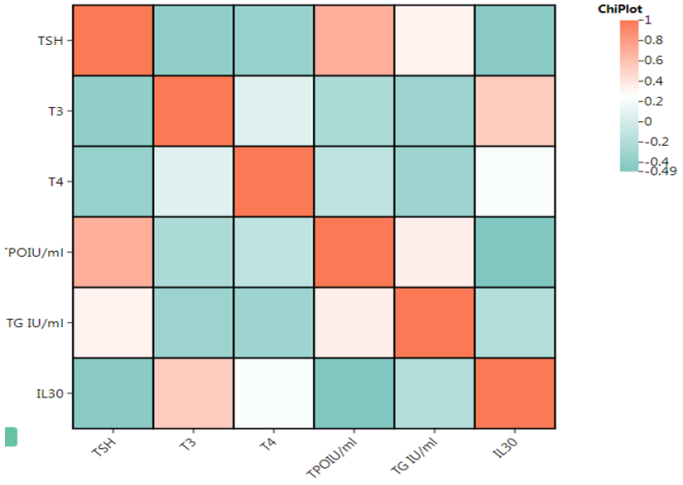

Figure 3: Correlation between serum IL-30 levels and biochemical parameters related to thyroid function

Discussion

Autoimmune thyroiditis is one of the cell-mediated autoimmune diseases for which the etiology is uncertain [1]. Overall, the data support a potential protective role of IL-30 in maintaining thyroid function and modulating autoimmune activity. Several cytokines, including IL-17, IL-23, IL-28, and IL-29, have important role in the pathogenesis of autoimmune thyroiditis [12-14]. The deficiency in IL-39, which is a newly discovered member of the IL-12 family cytokine, documented to be associated with the development of thyroid autoimmune diseases, can be considered a predictive biomarker [15].

IL-27 is also one of the IL-12 family cytokines that regulates immunological responses, including autoimmunity. IL-30, also named as the IL-27p28 subunit, was first identified as a specific subunit of IL-27 that may be secreted independently of Ebi3, a subunit that combines IL-30 to generate bioactive IL-27. Furthermore, IL-30 itself might counteract IL-27 by acting as a negative regulator [16].

In the present study, serum IL-30 levels were significantly reduced in patients compared with controls and showed a negative correlation with TSH, reflecting hypothyroidism severity. These findings are consistent with reports by Do et al. (2017) and Min et al. (2021), which demonstrated that IL-30 can antagonize IL-27 signaling in regulatory T cells (Tregs), thereby impairing their suppressive function. Reduced IL-30 may therefore exacerbate autoimmune inflammation [10,17]. Serum IL-30 levels also paralleled autoimmune antibodies (TPO, TG), decreasing further with rising TSH, particularly when TSH >10 mU/L (overt hypothyroidism). This suggests that downregulation of IL-30 is linked to thyroid autoimmunity and progression of thyroid dysfunction [15]. These observations highlight the complex and sometimes contradictory roles of IL-30 in immunity. For example, while elevated IL-27 and IL-30 have been proposed as diagnostic and activity biomarkers in rheumatoid arthritis [18], IL-30 deficiency in experimental models increased disease severity, as seen in IL-30-deficient mice with aggravated experimental autoimmune encephalomyelitis [16].

Regarding thyroid antibodies, the study showed that serum TPO and TG antibody levels decreased as TSH increased. This indicates that antibody titers do not always track clinical course or severity, as thyroid dysfunction in Hashimoto's thyroiditis is driven mainly by cellular rather than humoral mechanisms [19]. IL-30 exhibits both pro- and anti-inflammatory activities in viral and autoimmune diseases. It modulates cytokine pathways via GP130, particularly IL-6 signaling, acting as a natural GP130 antagonist. By doing so, IL-30 suppresses macrophage activation and limits pro-inflammatory responses. Since IL-6 and IL-27 also signal through GP130, tight regulation of IL-30 is required. Dysregulation of this balance promotes autoimmune inflammation [10,20].

IL-30 additionally protects against inflammation-induced liver injury [10,21]. It forms complexes with various partners. The IL-30/Ebi3 heterodimer (IL-27) carries out anti-inflammatory and regulatory functions, initially thought to mimic IL-12 by inducing Th1 responses via STAT1-mediated IL-12Rβ2 upregulation. IL-30 may also pair with cytokine-like factor-1 (CLF1), secreted by dendritic cells. The IL-30/CLF1 complex enhances NK cell activity, suppresses T-cell proliferation, and promotes IL-17 and IL-10 production, explaining its dual pro- and anti-inflammatory properties [22]. Tormo et al. reported that IL-30/CLF1 also acts on B cells, promoting plasma cell differentiation and antibody production. Reduced IL-30 may therefore enhance IL-30/CLF1 activity, leading to increased TG and TPO antibody production [23]. Since autoimmune thyroiditis is largely a cellular immune response dependent on IL-12 and IFN-γ, the induction of IL-30 by IL-12 through IFN-γ/STAT1 signaling suggests that therapeutic upregulation of IL-30 may help modulate disease progression [24].

The study further identified a significant association between IL-30 and anti-TPO antibodies. Both TPO and TG antibodies contribute to disease pathogenesis, although anti-TPO is more prevalent and more strongly predictive of thyroid dysfunction [25]. Rising TSH levels, reflecting hypothyroidism, indicate thyroid tissue destruction primarily via cell-mediated mechanisms [24,26]. This may explain why decreases in IL-30 coincide with reduced antibody levels and progression to overt hypothyroidism [27].

Limitations

To the best of our knowledge, this study presents a comprehensive investigation of the role of IL-30 in the pathogenesis of Hashimoto's thyroiditis by assessing serum levels in affected female patients. Nevertheless, limitations of our study were noted and should be considered in future studies. First, the study was conducted at a single center with a relatively limited sample size. While the sample size is reasonable for preliminary findings, future studies with more participants that include a larger cohort may be necessary to validate the results and ensure broader applicability, thereby confirming and extending our observations. Second, all participants were female, which raises questions about the generalizability of the results to a male population. Therefore, future studies that include male participants would be necessary to determine whether the findings can be generalized across both sexes. Therefore, future longitudinal studies are required to monitor IL-30 level changes throughout the disease course and to assess changes in response to various therapeutic interventions. This will provide further insight into its potential role in disease monitoring and management.

Conclusion

The current study revealed that a low serum level of IL-30 is associated with the pathogenesis of autoimmune thyroiditis. A negative correlation was found between serum IL-30 and the degree of hypothyroidism, as reflected in the serum TSH level. Therefore, a deficiency in serum IL-30 might be a predictive biomarker for patients with autoimmune thyroiditis.

Funding

This study did not receive any specific funding from public, commercial, or non-profit organizations

Conflict of Interest

The author declares no conflicts of interest related to this work.

Data availability

Data are available upon reasonable request.

Author Contributions

NHM contributed to conception, study design, interpretation of results, and revision and proofreading of the manuscript. AA contributed to data acquisition, data analysis, interpretation of results, drafting of the manuscript, and revision and proofreading. SBA contributed to the study design, revision, and proofreading. All authors read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

All authors meet the ICMJE criteria for authorship and agree to be accountable for all aspects of the work.

ORCID

Nadia Mohammed 0000-0001-9234-3198
Anmar Al-Taie 0000-0002-2183-6830
Sinan Alrifai 0000-0003-1585-2401

References

[1] Bellan M, Andreoli L, Mele C, Sainaghi PP, Rigamonti C, Piantoni S, De Benedittis C, Aimaretti G, Pirisi M, Marzullo P: Pathophysiological Role and Therapeutic Implications of Vitamin D in Autoimmunity: Focus on Chronic Autoimmune Diseases. Nutrients 2020, 12(3):789. https://doi.org/10.3390/nu12030789

[2] Qian Zhu, Qian-Han Xu, Ting Xie, Li-Ling Wang, Hong Liu, Kahindo P. Muyayalo, Xiao-Bo Huang, Si-Jia Zhao, Ai-Hua Liao: Recent insights into the impact of immune dysfunction on reproduction in autoimmune thyroiditis. Clin Immunol 2021, 224: 108663. https://doi.org/10.1016/j.clim.2020.108663

[3] Al-Hassani AJ, Al-Zamely HA, Hameed ZA. Incidence of Hashimoto's disease and its relationship to age, sex, smoking and blood groups. NTU Journal of Pure Sciences. 2021;4(1):63-73.

[4] Ralli M, Angeletti D, Fiore M, D'Aguanno V, Lambiase A, Artico M, de Vincentiis M, Greco A: Hashimoto's thyroiditis: An update on pathogenic mechanisms, diagnostic protocols, therapeutic strategies, and potential malignant transformation. Autoimmun Rev 2020, 19(10):102649. https://doi.org/10.1016/j.autrev.2020.102649

[5] Bogusławska J, Godlewska M, Gajda E, Piekiełko-Witkowska A: Cellular and molecular basis of thyroid autoimmunity. Eur Thyroid J 2022, 11(1):e210024. https://doi.org/10.1530/ETJ-21-0024

[6] Luty J, Ruckemann-Dziurdzińska K, Witkowski JM, Bryl E: Immunological aspects of autoimmune thyroid disease - Complex interplay between cells and cytokines. Cytokine 2019, 116:128-133. https://doi.org/10.1016/j.cyto.2019.01.003

[7] Koehler VF, Filmann N, Mann WA: Vitamin D Status and Thyroid Autoantibodies in Autoimmune Thyroiditis. Horm Metab Res 2019, 51(12):792-797. https://doi.org/10.1055/a-1023-4181

[8] Tonel G, Conrad C: Interplay between keratinocytes and immune cells--recent insights into psoriasis pathogenesis. Int J Biochem Cell Biol 2009, 41(5):963-968. https://doi.org/10.1016/j.biocel.2008.10.022

[9] Stumhofer JS, Tait ED, Quinn WJ 3rd, Hosken N, Spudy B, Goenka R, Fielding CA, O'Hara AC, Chen Y, Jones ML, Saris CJ, Rose-John S, Cua DJ, Jones SA, Elloso MM, Grötzinger J, Cancro MP, Levin SD, Hunter CA: A role for IL-27p28 as an antagonist of gp130-mediated signaling. Nat Immunol 2010, 11(12):1119-1126. https://doi.org/10.1038/ni.1957

[10] Min B, Kim D, Feige MJ. IL-30† (IL-27A): A familiar stranger in immunity, inflammation, and cancer. Exp Mol Med 2021, 53(5):823-834. https://doi.org/10.1038/s12276-021-00630-x

[11] Alaa Ibrahim Hashim, Dr. Ibtisam Habeeb Saeed: An Association of IL-6 and TNF Alpha Levels with Hashimoto Thyroiditis in Najaf Province/ Iraq. Thi-Qar Medical Journal TQMJ 2024, (2). https://doi.org/10.32792/jmed.v28i2.548

[12] Gerenova J, Manolova I, Stanilova S. Serum Levels of Interleukin - 23 and Interleukin - 17 in Hashimoto's Thyroiditis. Acta Endo (Buc) 2019, 15 (1): 74-79. https://doi.org/10.4183/aeb.2019.74

[13] Arpaci D, Karakas Celik S, Can M, Cakmak Genc G, Kuzu F, Unal M, Bayraktaroglu T. Increased Serum Levels of IL-28 and IL-29 and the Protective Effect of IL28B rs8099917 Polymorphism in Patients with Hashimoto's Thyroiditis. Immunol Invest 2016, 45(7):668-78. https://doi.org/10.1080/08820139.2016.1208215

[14] Esfahanian F, Ghelich R, Rashidian H, Jadali Z. Increased levels of serum interleukin-17 in patients with Hashimoto's thyroiditis. Indian J Endocrinol Metab 2017, 21(4):551-554. https://doi.org/10.4103/ijem.IJEM_412_16

[15] Weng L, Huang G, Gong L, Xu J, Mao Y, Li Y, Li M. Low levels of serum IL-39 are associated with autoimmune thyroid disease. J Clin Lab Anal. 2022, 36(4):e24284. https://doi.org/10.1002/jcla.24284

[16] Kim D, Kim S, Kang MS, Yin Z, Min B. Cell type specific IL-27p28 (IL-30) deletion in mice uncovers an unexpected regulatory function of IL-30 in autoimmune inflammation. Sci Rep 2023, 13: 1812. https://doi.org/10.1038/s41598-023-27413-w

[17] Do J, et al. Treg-specific IL-27Ralpha deletion uncovers a key role for IL-27 in Treg function to control autoimmunity. Proc. Natl Acad. Sci. USA. 2017;114:10190–10195. https://doi.org/10.1073/pnas.1703100114

[18] Tawfeeq AF, Al-Hadithi HS, Gorial FI. Serum Levels of IL-27 and IL-30 as Predictor of Disease Activity and Treatment Response in Patients with Rheumatoid Arthritis: A Case-Control Study. AJMS 2024, 7(2): 115–119. https://doi.org/10.54133/ajms.v7i2.1449

[19] Mehanathan PB, Erusan RR, Shantaraman K, Kannan SM. Antithyroid Peroxidase Antibodies in Multinodular Hashimoto's Thyroiditis Indicate a Variant Etiology. J Thyroid Res 2019; 2019:4892329. https://doi.org/10.1155/2019/4892329

[20] Gong H, Ma S, Chen J, Yang B, Liu S, Liu X, Han J, Wu X, Lei L, Yin Z, Sun H, Yu D, Liu H, Xu Y, Wu D. Dendritic cell-derived IL-27 p28 regulates T cell program in pathogenicity and alleviates acute graft-versus-host disease. Signal Transduct Target Ther 2022, 7(1):319. https://doi.org/10.1038/s41392-022-01147-z

[21] Dibra D, Cutrera J, Xia X, Kallakury B, Mishra L, Li S. Interleukin-30: a novel antiinflammatory cytokine candidate for prevention and treatment of inflammatory cytokine-induced liver injury. Hepatology 2012, 55(4):1204-14. https://doi.org/10.1002/hep.24814

[22] Crabé S, Guay-Giroux A, Tormo AJ, Duluc D, Lissilaa R, Guilhot F, Mavoungou-Bigouagou U, Lefouili F, Cognet I, Ferlin W, Elson G, Jeannin P, Gauchat JF. The IL-27 p28 subunit binds cytokine-like factor 1 to form a cytokine regulating NK and T cell activities requiring IL-6R for signaling. J Immunol 2009, 183(12):7692-702. https://doi.org/10.4049/jimmunol.0901464

[23] Tormo AJ, Meliani Y, Beaupré LA, Sharma M, Fritz JH, Elson G, Crabé S, Gauchat JF. The composite cytokine p28/cytokine-like factor 1 sustains B cell proliferation and promotes plasma cell differentiation. J Immunol 2013, 191(4):1657-65. https://doi.org/10.4049/jimmunol.1201595

[24] Zhang J, Tang H, Wu H, Pang X, Jin R, Zhang Y. Thymic dendritic cell-derived IL-27p28 promotes the establishment of functional bias against IFN-γ production in newly generated CD4+ T cells through STAT1-related epigenetic mechanisms. Elife. 2025 May 14;13:RP96868. https://doi.org/10.7554/eLife.96868

[25] Balucan FS, Morshed SA, Davies TF. Thyroid autoantibodies in pregnancy: their role, regulation and clinical relevance. J Thyroid Res 2013, 2013:182472. https://doi.org/10.1155/2013/182472

[26] Kilgore AM, Welsh S, Cheney EE, Chitrakar A, Blain TJ, Kedl BJ et.al. IL-27p28 Production by XCR1+ Dendritic Cells and Monocytes Effectively Predicts Adjuvant-Elicited CD8+ T Cell Responses. Immunohorizons 2018; 2(1):1-11. https://doi.org/10.4049/immunohorizons.1700054

[27] Zaletel K, Gaberscek S. Hashimoto's thyroiditis: from genes to the disease. Current genomics. 2011 Dec 1;12(8):576-88. http://dx.doi.org/10.2174/138920211798120763