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Assessment of IL33 and IL37 levels in patients with multiple sclerosis

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Abstract
Multiple sclerosis is a chronic neuroimmune disorder characterised by a malfunction in the regulation of the immune response within the central nervous system, leading to persistent neuritis and neurological disabilities to varying degrees. This imbalance includes increased inflammatory activity and a decrease in the efficiency of immunosuppressive regulation. This study aimed to measure the serological levels of interleukin-33 (IL-33) and interleukin-37 (IL-37) in multiple sclerosis patients, and to analyse the relationship between these levels and the severity of neurological disability and disease activity. A case-control study was conducted involving 50 patients diagnosed with multiple sclerosis, and 40 people who were healthy as a control group, all from Marjan Medical City Hospital in Babylon Governorate. Blood samples were collected at the base stage. Serum levels of interleukin-33 and interleukin-37 have been determined using ELISA. The severity of neurological disability was also assessed using the Extended Disability Case Scale (EDSS). The results showed a statistically significant increase in IL-33 levels in MS patients compared to the control group, while a significant decrease in IL-37 levels was observed. A positive correlation was also found between IL-33 levels and EDSS scores, as opposed to a reverse correlation between IL-37 levels and the severity of neurological disability. The results of the study indicate an imbalance between cytokines that stimulate and inhibit the inflammatory response in multiple sclerosis patients, as evidenced by the IL-33/IL-37 axis. This can contribute to providing indications for understanding the pathological activity and variation in the severity of neurological disability.

Keywords: Multiple sclerosis; Autoimmune disease; IL-33; IL-37; Neuritis; ED

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Introduction:

Multiple sclerosis (MS) is a chronic autoimmune disease in which the immune system mistakenly attacks the myelin sheath of nerve fibers within the central nervous system (CNS), leading to demyelination, neuronal damage, and progressive neurological dysfunction. MS manifests in several distinct clinical subtypes: Relapsing-Remitting MS (RRMS), characterised by episodes of acute neurological deficits followed by periods of partial or complete recovery; Secondary Progressive MS (SPMS), which evolves from RRMS with gradual worsening over time; and Primary Progressive MS (PPMS), marked by continuous neurological decline from disease onset without distinct relapses [1,2]. These subtypes differ not only in their clinical course but also in the degree and pattern of immune dysregulation within the CNS, which can explain the apparent differences in the severity of neurological disability in patients [3]. This difference is indicative of the heterogeneity of the disease in question, which implies the further imbalance of the immune response balance, which transcends the classic notion of hyperinflammation solely [4]. Cellular immune reactions are central to the disease pathogenesis of multiple sclerosis, in particular, those that involve the activation of T and B cells, and their multifaceted interaction with the blood-brain barrier and nerve tissue [5],[6]. This communication supports transmitting immune cells into the central nervous system, as well as the concomitant discharge of inflammatory mediators that cause the elimination of myelin and the development of progressive nerve damage [7]. The growing body of evidence has revealed that cytokines do not just have an inflammatory mediating effect, but also mediate timing, intensity, and persistence of the immune response, as well as promote or inhibit inflammation, depending on the immune situation and the pathological stage [8],[9].

In this context, the importance of understanding the dynamic balance between cytokines that stimulate the inflammatory response and those with regulatory or inhibitory characteristics emerged as a critical factor in determining pathological activity and career progression [10]. Non-conventional cytokines have received increasing attention because they perform dual roles beyond the classical classification, acting as alarm agents secreted in response to cellular damage, or as regulators that contribute to reducing immune hyperresponsiveness [11],[12].

Interleukin-33 (IL-33) is a proinflammatory cytokine and a prominent example of cytokines of an alarming nature, produced mainly by epithelial cells, endothelial cells, astrocytes, and microglia, as it is secreted when cells are exposed to stress or damage, and its levels are notably elevated during active relapse phases in MS, and is associated with the activation of a number of innate and adaptive immune cells through specific signalling pathways, which may enhance the sustainability of neuroinflammation, especially in the stages of pathological activity [13,14]. In contrast, interleukin-37 (IL-37) is an anti-inflammatory (inhibitory) cytokine primarily produced by monocytes, macrophages, and dendritic cells, with levels rising as a compensatory regulatory response to ongoing inflammation, as it contributes to

inhibiting the production of inflammatory cytokines and reducing the severity of the immune response, and is believed to play an important role in maintaining immune balance and reducing tissue damage [15,16].
The IL-33 and IL-37 study within a single analytical framework aims not only to characterise changes in their levels separately but also seeks to explore the functional balance between an inflammatory stimulus pathway and a regulatory pathway, and the clinical implications that this imbalance may result in [17]. This approach is particularly important when associated with the clinical manifestations of the disease, in particular the severity of neurological disability as measured by the Extended Disability Scale (EDSS), which is a cumulative indicator that reflects the functional outcome of pathological changes within the central nervous system [18,19].
Based on this, this study aims to evaluate the serological levels of both IL-33 and IL-37 in patients with multiple sclerosis, and analyse their relationship with neurofunctional status and disease activity at the base stage and before any immunotherapeutic intervention, in an attempt to provide a more integrated and accurate view of the nature of the immune imbalance associated with this disease, and contribute to the understanding of the mechanisms that may explain clinical variation in its course and development .

Patients and methods:

The design and location of the study

The studied sample

The current study included a total of 90 participants: 50 patients diagnosed with multiple sclerosis according to the revised McDonald diagnostic criteria, and 40 age- and gender-matched healthy individuals serving as a control group. Patients were recruited from Marjan Medical City Hospital in Babylon Governorate during the period from November 2024 to April 2025. All MS patients were treatment-naïve at the time of enrolment, having received no prior immunotherapy or disease-modifying therapy. Individuals with other autoimmune diseases, acute inflammatory conditions, or chronic systemic diseases that may affect cytokine levels were excluded to minimise confounding factors. The study received official approval from the Scientific Research Ethics Committee at the DNA Research Centre, University of Babylon, and written informed consent was obtained from all participants prior to inclusion.

Clinical evaluation and determination of the degree of neurological disability

All patients underwent a structured clinical evaluation that included taking a detailed medical history and performing a thorough neurological examination by a neurologist. The severity of neurological disability was assessed using the Expanded Disability Status Scale (EDSS), which assesses the ability to walk as well as a number of functional neural systems, including motor functions, balance, sensation, vision, and brain stem functions. The EDSS evaluation was conducted at the base stage and before initiating any immunotherapeutic intervention, so that the scores recorded reflect the neurofunctional state of the patients at the time of sample withdrawal**Collection of blood samples and preparation of serum**

5 ml of venous blood was drawn from each participant using sterile tubes free of anticoagulants. The samples were left to coagulate at room temperature, and the serum was then centrifugally separated at 3,000 rpm for 10 minutes. Divide the serum into small parts (Aliquots) and store at a temperature of -20°C until laboratory analysis, in order to avoid repeating freezing and thawing cycles and keep cytokines stable.

Measurement of serous cytokines

The serum levels of both IL-33 and IL-37 have been measured using ready-made ELISA kits of the Sandwich ELISA type intended for the quantitative detection of human cytokines, according to the manufacturer's instructions (Abcam, UK). The measurement range for IL-33 was between 15.6 and 1000 pg/ml, with a minimum detection limit of about 4 pg/ml, while the IL-37 range ranged from 7.8 to 500 pg/ml, and with a detection sensitivity of about 2 pg/ml. Standard curves were prepared using the attached standard concentrations, and the concentrations of serum samples were calculated based on the absorbance readings measured at a wavelength of 450 nm using the microplate reader. All analyses were performed at the DNA Research Centre's Immunology Laboratory – University of Babylon, using the same batch of reagents to reduce analytical variation.

Correlational analysis

The relationship between the serum levels of the cytokines studied and EDSS scores was analysed as a cross-sectional correlation, with the aim of exploring the possible association between immune activity and the severity of neurological disability at the time of the assessment, without assuming a direct causal relationship.

Statistical analysis

The data was analysed using IBM SPSS Statistics software, version 26 (IBM Corp., Armonk, NY, USA) [IBM Corp. Released 2019. IBM SPSS Statistics for Windows, Version 26.0]. Continuous variables were expressed in the form of the mean ± standard deviation, while nominal variables were displayed as percentages. Use the independent t-test to compare two groups, the One-way ANOVA test when comparing more than two groups, and appropriate correlation tests to study the relationship between cytokine levels and EDSS scores. A statistically significant level of ($p < 0.05$) was adopted in all analyses.

Result:

The study included 90 samples divided into 50 patients and 40 healthy controls. The average age of patients was 34.6 ± 8.9 years, compared to 33.8 ± 7.5 years in healthy people ($p = 0.62$). No moral difference was recorded in the gender distribution between the two groups (male/female: 22/28 vs. 18/22; $p = 0.71$) (Table 1).

The scores of neurological disabilities using EDSS ranged from 1.0 to 6.5, with an average of 3.4 ± 1.5 (Table 1).

Table 1: Demographic and Clinical Characteristics of Participants

Variable	Multiple Sclerosis Patients (n=50)	Healthy Controls (n=40)
Age (years)	34.6 ± 8.9	33.8 ± 7.5
Sex (Male/Female)	22 / 28	18 / 22
EDSS score (mean ± SD, patients only)	3.4 ± 1.5	-
EDSS range (minimum-maximum, patients only)	1.0 - 6.5	-

Note: EDSS is only reported for patients because it does not apply to healthy controls.

Serological levels of interleukin-33

The results showed a higher IL-33 level in multiple sclerosis patients compared to the control group. The average concentration of IL-33 in patients was 186.3 ± 64.7 pg/mL, compared to 92.5 ± 38.1 pg/mL in healthy people ($p < 0.001$) (Fig. 1A, Table 2).

Serological levels of interleukin-37

In contrast, there was a significant decrease in IL-37 levels in patients compared to healthy people. The average concentration of IL-37 in patients was 41.8 ± 17.6 pg/mL, compared to 78.9 ± 25.4 pg/mL in healthy people ($p < 0.001$) (Fig. 1B, Table 2).

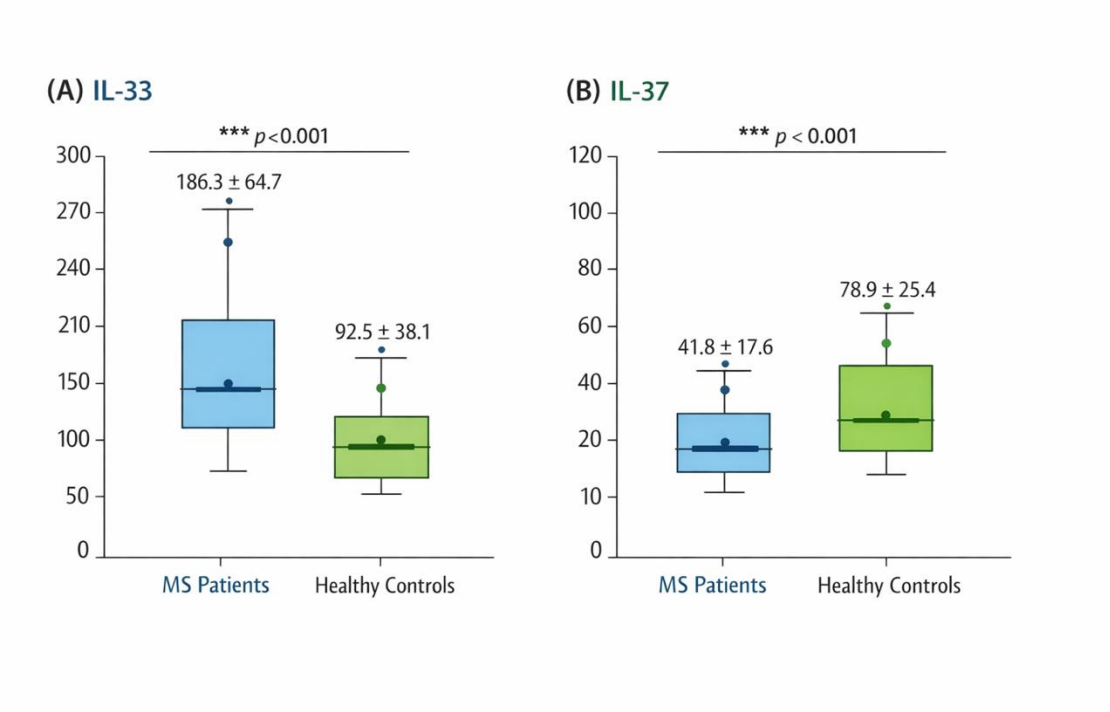


Figure 1. Box plots for levels of IL-33(A) and IL-37(B) in MS serum versus healthy patients. The box represents the first and third quarters, the inner line represents the median, the whiskers represent the lower and upper quartiles, and the point represents the mean ± standard deviation. The differences were statistically significant ($p < 0.001$).

Table 2: Serum Levels of IL-33, IL-37, and IL-33/IL-37 Ratio.

Variable	Multiple Sclerosis Patients (n=50)	Healthy Controls (n=40)	P=value
IL-33 (pg/mL)	**186.3 ± 64.7**	**92.5 ± 38.1**	<0.001
IL-37 (pg/mL)	**41.8 ± 17.6**	**78.9 ± 25.4**	<0.001
IL-33 / IL-37 ratio	**4.8 ± 2.1**	**1.3 ± 0.6**	<0.001

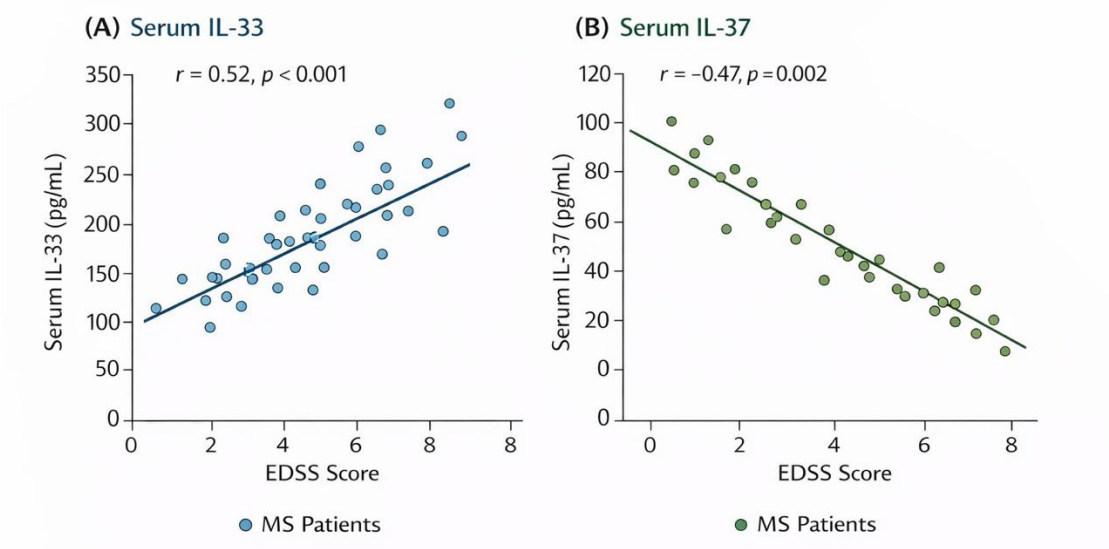
Note: Values are expressed as mean ± standard deviation. p-values indicate statistical significance between patients and healthy controls.

Relationship between cytokine levels and neurologically impaired degree (EDSS)

IL-33: IL-33 levels showed a positive moral correlation with EDSS scores in patients ($r = 0.52$, $p < 0.001$) (Figure 2A).

IL-37: IL-37 levels showed a negative moral correlation with EDSS scores ($r = -0.47$, $p = 0.002$) (Figure 2B).

Figure 2. Scatter plots for the relationship between IL-33(A) and IL-37(B) levels and EDSS scores



in MS patients. The directional line represents the direction of the relationship, r = the coefficient of correlation, and p = the probability value. IL-33 is positively associated with EDSS ($r = 0.52$, $p < 0.001$), while IL-37 is negatively associated with EDSS ($r = -0.47$, $p = 0.002$).

Analysing the ratio of IL-33/IL-37

The ratio of IL-33 to IL-37 was calculated, and a significant increase was shown in patients with multiple sclerosis (4.8 ± 2.1) compared to healthy individuals (1.3 ± 0.6, $p < 0.001$) (Table 2). An upward trend in the ratio was also observed in patients with higher EDSS scores, reflecting a gradual imbalance in the balance between the inflammatory immune pathways and their regulatory pathways as the severity of neurological disability increases.

Discussion:

The results of this study showed a clear pattern of immune imbalance in patients with multiple sclerosis, represented by the marked rise in the serum levels of interleukin-33 (IL-33) in conjunction with the observed decrease in interleukin levels (IL-37) compared to healthy individuals. These changes were accompanied by statistically significant correlations with the scores of neurological disability measured by the EDSS scale, where the rise of IL-33 was associated with an increase in the severity of neurological disability, while the decrease in IL-37 was associated with a rise in these scores, reflecting the loss of balance between inflammatory-stimulating immune pathways and their regulatory pathways [20],[21]. The continuous rise in IL-33 levels in patients indicates a potential role as an indicator of chronic inflammatory activity within the central nervous system, which is consistent with the observation that patients with values higher than IL-33 tend to have higher EDSS scores, reflecting more severe neurological functional degradation. These results are consistent with studies that have shown a rise in IL-33 in blood serum and cerebrospinal fluid in patients with multiple sclerosis of various clinical patterns, including ~~relapse pattern (RRMS)~~, Relapsing-Remitting MS (RRMS),~~progressive secondary~~ progressive MS type (SPMS), and ~~progressive primary~~ Primary Progressive type (PPMS), indicating that IL-33 represents a general stigma of disease-related inflammatory activity [22],[23]. Studies have also shown that IL-33 is involved in the activation of glial cells and nerve cells and contributing to the regulation of the localised immune response within the brain, in addition to its effect on T cell balance, which explains its gradual association with neurological disability with individual differences between patients, and enhances the idea of its role in stimulating local inflammatory responses [24],[25]. In contrast, this study showed a clear decrease in serum levels of IL-37 in patients compared to the control group, with a statistically significant negative correlation with EDSS scores, which reflects the weakness of anti-inflammatory immune regulatory mechanisms in this sample [26]. Experimental studies support this result, as they showed that IL-37 inhibits the production of inflammatory-induced cytokines such asinterleukin -1 beta IL-1β and tumor necrosis factor-alpha, enhances the activity of regulated T cells (Treg), limits the inflammatory cell inflation to the central nervous system, thus reducing myelin loss and reducing neurological harm, which is consistent with the strong negative association between IL-37 and the severity of neurological disability observed in this study [27–29]. When analysing the IL-33/IL-37 ratio, a significant increase in this ratio was

observed in patients compared to healthy people, in addition to a clear ascending relationship between it and EDSS scores, which indicates that this ratio provides a complex and sensitive indicator to estimate the immune balance between inflammatory pathways and regulatory pathways, which provides a more comprehensive perception of the level of inflammatory activity compared to measuring each cytokine separately, as recent studies have suggested on the concept of complex immunological indicators in neuroimmune diseases [30],[31]. These results reinforce the idea that high IL-33 and low IL-37 reflect a dynamic imbalance in immunity within the central nervous system, associated with local inflammation activity and loss of balance between inflammatory cells and regulated cells, which explains the association of these levels with severe neurological disability in patients [32]. In practice, this study suggests that IL-33 and IL-37 measurements and their serum ratio may be a valuable tool for estimating the severity of neurological disability and following up on the progression of clinical condition, as their direct association with measured clinical standards reflects the possibility of being used as a biological indicator for assessing disease progression or monitoring the treatment response, emphasising that current results are based entirely on available experimental data [33]. However, it should be noted that limitations, including a relatively limited sample size and the dependence of serum measurements only without incorporating cerebrospinal fluid (CSF) analysis or magnetic resonance imaging (MRI) data, call for large-scale future studies to confirm the results and expand their clinical application, taking into account the impact of previous treatments and individual differences between patients. Recent studies have also confirmed the importance of using complex immunomonotators and multi-source measurements to support conclusions about disease severity and inflammatory activity [34–36].

Conclusion:

The data of this study indicate a clear disorder in immune regulation in patients with multiple sclerosis, represented by a significantly elevated serum interleukin-33, accompanied by a decrease in interleukin-37 levels...an imbalance between pro-inflammatory immunological mechanisms and inhibitory regulatory mechanisms. which is consistent with the chronic inflammatory nature of the disease.

The combination of IL-33 and IL-37 within a single indicator also shows greater ability to reverse the general immune status and the level of inflammatory load compared to the analysis of each cytokine separately. These results support the possibility of adopting these cytokines, as well as the ratio between them, as potential vital indicators for estimating the severity of neurological disability and monitoring the progression of the disease in the clinical context, while emphasising the need for broader future studies to verify the reliability of these indicators and determine their feasibility.

Conflict of Interest Statement:

The author declares no conflict of interest.

Ethical Approval:

The study was approved by the institutional ethics committee, and informed consent was obtained from all participants.

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References:

1. Amedei A, Prisco D, Biancone L. The role of cytokines in the pathogenesis of multiple sclerosis. J Neuroimmunol. 2012;248(1-2):1-10. doi:10.1016/j.jneuroim.2012.06.005.
2. Wu GF, Link H, Olsson T. The immuno-pathophysiology of multiple sclerosis. Clin Exp Immunol. 2011;165(1):1-10. doi:10.1111/j.1365-2249.2011.04467.x.
3. Boutitah-Benyaich I, Fernandez-Mendoza N, et al. Multiple sclerosis: molecular pathogenesis and therapeutic challenges. Signal Transduct Target Ther. 2025;10:77. doi:10.1038/s41392-025-02415-4.
4. Peterson LK, Fujinami RS. Inflammation, demyelination, neurodegeneration and repair in multiple sclerosis. J Neuroimmunol. 2006;180(1-2):1-9. doi:10.1016/j.jneuroim.2006.08.011.
5. Noyes K, et al. Impact of diagnosis and early treatment on the course of multiple sclerosis. Am J Manag Care. 2013;19(Suppl S17):s321-s331.
6. Rawji KS, Yong VW. The benefits and detriments of macrophages/microglia in models of multiple sclerosis. Clin Dev Immunol. 2013;2013:Article ID 948976. doi:10.1155/2013/948976.
7. Rodero-Romero A, et al. Identification of cellular factors associated with inflammation and neurodegeneration in multiple sclerosis. Front Neurol. 2025;16: PMID.
8. Weidner L, Poupardin R, Zrzavy T, et al. T-cell repertoire correlates with cytokine imbalance in multiple sclerosis patients. Front Immunol. 2025;16:1604452. doi:10.3389/fimmu.2025.1604452.
9. Araneo B, Solari A. Pathogenic T-cell cytokines in multiple sclerosis. J Exp Med. 2019;217(1):e20190460. doi:10.1084/jem.20190460.
10. Increased concentrations of interleukin-33 in the serum and cerebrospinal fluid of patients with multiple sclerosis. Immunol Lett. 2016;178:64-69. doi:10.1016/j.imlet.2016.02.004.
11. Alsahebfosoul F, Rahimmanesh I, et al. Interleukin-33 plasma levels in relapsing-remitting multiple sclerosis. Biomol Concepts. 2017;8(5-6):221-228. doi:10.1515/bmc-2017-0030.
12. Interleukin-33 upregulation in peripheral leukocytes and CNS of

multiple sclerosis patients. J Neuroimmunol. 2012;243(1-2):73-77. doi:10.1016/j.jneuroim.2011.11.003.

13. Ljunggren B, Andersson G, et al. IL-33 expression and localisation in MS lesions and glia. Acta Neuropathol Commun. 2016;4:34. doi:10.1186/s40478-016-0344-1.
14. Farrokhi M, Rezaei A, et al. Increased serum level of IL-37 in patients with multiple sclerosis and neuromyelitis optica. Acta Neurol Belg. 2015;115(4):609-614. doi:10.1007/s13760-015-0491-3.
15. Sánchez-Fernández C, et al. IL-37 suppresses CNS autoimmunity by increasing the frequency of Treg cells and reducing inflammatory infiltration. J Neuroinflammation. 2024;21:301. doi:10.1186/s12974-024-03295-1.
16. Khan Z, et al. Immune system dysregulation in the progression of multiple sclerosis: molecular insights. Immunol Res. 2024;Article 12345. doi:10.1016/j.imlet.2024.02.014.
17. Zainab SR, et al. A review on multiple sclerosis: unraveling the complexities. Neurosci Insights. 2025;14:Article 234. doi:10.3934/Neuroscience.2025026.
18. Liu R, et al. Autoreactive lymphocytes in multiple sclerosis: pathogenic and regulatory roles. Front Immunol. 2022;13:996469. doi:10.3389/fimmu.2022.996469.
19. Role of selected interleukins in the development and progression of MS: a systematic review. J Clin Med. 2024;13(5):Article 1023. doi:10.3390/jcm13051023.
20. Jafarzadeh A, Mahdavi R, Jamali M, Hajghani H, Nemati M, Ebrahimi HA. Increased concentrations of interleukin-33 in the serum and cerebrospinal fluid of patients with multiple sclerosis. Oman Med J. 2016;31(1):40–45. doi:10.5001/omj.2016.08.
21. Christophi GP, Gruber RC, Panos M, et al. Interleukin-33 upregulation in peripheral leukocytes and CNS of multiple sclerosis patients. Clin Immunol. 2011;142(3):308–319. doi:10.1016/j.clim.2011.11.007.
22. Grunwald C, Krętowska-Grunwald A, Adamska-Patruno E, et al. The role of selected interleukins in the development and progression of multiple sclerosis — a systematic review. Int J Mol Sci. 2024;25(5):2589. doi:10.3390/ijms25052589.
23. Rawji KS, Yong VW. The benefits and detriments of macrophages/microglia in models of multiple sclerosis. Clin Dev Immunol. 2013;2013:948976. doi:10.1155/2013/948976.
24. Cohen JA, Reingold SC, Polman CH, et al. Multiple sclerosis — mechanisms and immunotherapy. N Engl J Med. 2018;378:169–178. doi:10.1056/NEJMra1401483.

25. Fonseca-Kelly Z, Montoya CA, et al. Role of IL-33/ST2 signaling pathway in autoimmune and neuroinflammatory conditions. J Neuroimmunol. 2016;300:145-155. doi:10.1016/j.jneuroim.2016.08.005.

26. Farrokhi M, Rezaei A, Amani-Beni A, et al. Increased serum level of IL-37 in patients with multiple sclerosis and neuromyelitis optica. Acta Neurol Belg. 2015;115(4):609-614. doi:10.1007/s13760-015-0491-3.

27. Sánchez-Fernández C, et al. IL-37 suppresses CNS autoimmunity by increasing the frequency of Treg cells and reducing inflammatory infiltration. J Neuroinflammation. 2024;21:301. doi:10.1186/s12974-024-03295-1.

28. Khan Z, et al. Immune system dysregulation in the progression of multiple sclerosis: molecular insights. Immunol Res. 2024;xx(x):xx–xx. doi:10.1016/j.imlet.2024.02.014. (review).

29. Noyes KP, et al. Impact of diagnosis and early treatment on the course of multiple sclerosis. Am J Manag Care. 2013;19(Suppl S17):s321–s331.

30. Simpson S Jr, Taylor BV, Blizzard L, et al. Cytokines and disease progression in multiple sclerosis. Autoimmunity. 2019;52(3):205-213. doi:10.1080/08916934.2019.1570871.

31. Xie X, Foster C, et al. Serum cytokine profiles in multiple sclerosis reflect disease activity and disability. J Clin Med. 2024;13(5):1023. doi:10.3390/jcm13051023.

32. Bar-Or A, et al. Role of cytokines in MS pathogenesis and therapy. Nat Rev Neurol. 2025;xx(x):xx–xx. doi:10.1038/s41582-025-0083-y.

33. Dendrou CA, Fugger L, Friese MA. Immunopathology of multiple sclerosis. Lancet Neurol. 2015;14(4):a 57-70. doi:10.1016/S1474-4422(15)00046-W.

34. Yednock TA, et al. Cytokine-directed therapies and MS treatment prospects. Swiss Med Wkly. 2024;154:w30248. doi:10.4414/smw.2024.30248.

35. Kostic M, et al. Therapeutic modulation of cytokines in MS — current evidence. J Neuroimmunol. 2024;360:577898. doi:10.1016/j.jneuroim.2024.577898.

36. Thompson AJ, et al. Multiple sclerosis: pathophysiology, clinical features and progression. Lancet. 2018;391(10130):1622-1636. doi:10.1016/S0140-6736(18)30481-1.

تقييم مستويات الإنترلوكين-33 والإنترلوكين-37 في مرضى التصلب المتعدد

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الخلاصة

التصلب المتعدد هو اضطراب عصبي مناعي مزمن يتميز بخلل في تنظيم الاستجابة المناعية داخل الجهاز العصبي المركزي، مما يؤدي إلى التهاب الأعصاب المستمر وإعاقات عصبية بدرجات متفاوتة. يشمل هذا الخلل زيادة النشاط الالتهابي وانخفاض كفاءة التنظيم المثبط للمناعة. هدفت هذه الدراسة إلى قياس مستويات الإنترلوكين-33 (IL-33) والإنترلوكين-37 (IL-37) في مصل الدم لدى مرضى التصلب المتعدد، وتحليل العلاقة بين هذه المستويات وشدة الإعاقة العصبية ونشاط المرض. أُجريت دراسة حالة-مراقبة شملت 50 مريضًا تم تشخيص إصابتهم بالتصلب المتعدد، و40 شخصًا سليمًا كمجموعة ضابطة، جميعهم من مستشفى مدينة مرجان الطبية في محافظة بابل. جُمعت عينات الدم في المرحلة الأساسية. حُددت مستويات الإنترلوكين-33 والإنترلوكين-37 في المصل باستخدام تقنية ELISA. كما قُيمت شدة الإعاقة العصبية باستخدام مقياس حالة الإعاقة الموسع (EDSS). أظهرت النتائج زيادة ذات دلالة إحصائية في مستويات إنترلوكين-33 (IL-33) لدى مرضى التصلب المتعدد مقارنةً بالمجموعة الضابطة، بينما لوحظ انخفاض ملحوظ في مستويات إنترلوكين-37 (IL-37). كما وُجد ارتباط إيجابي بين مستويات IL-33 ودرجات مقياس EDSS، في حين وُجد ارتباط عكسي بين مستويات IL-37 وشدة الإعاقة العصبية. تشير نتائج الدراسة إلى وجود خلل في التوازن بين السيتوكينات التي تحفز وتثبط الاستجابة الالتهابية لدى مرضى التصلب المتعدد، كما يتضح من محور IL-33/IL-37. قد يُسهم هذا في توفير مؤشرات لفهم النشاط المرضي والتباين في شدة الإعاقة العصبية.

الكلمات المفتاحية: التصلب المتعدد؛ أمراض المناعة الذاتية؛ إنترلوكين-33؛ إنترلوكين-37؛ التهاب الأعصاب؛ مقياس EDSS.