

Alterations of mucosal-associated invariant T cells in dermatomyositis

S. Ye¹, J. Du^{2,3}, Y. Zhang¹, W. Wei^{2,3}, Y. Li^{2,3}

¹Department of Medical Records Management, Department of Neurology, Tianjin Huanhu Hospital, Tianjin Key Laboratory of Cerebral Vascular and Neurodegenerative Diseases, Tianjin;

²Department of Rheumatology and Immunology, Tianjin Medical University General Hospital, Tianjin Key Clinical Specialty, Tianjin; ³Tianjin Clinical Research Center for Rheumatic and Immune Diseases, Tianjin Science and Technology Bureau, Tianjin, China.

Abstract

Objective

The aim of this study is to investigate the frequency and functional changes of mucosal-associated invariant T (MAIT) cells in the peripheral blood of patients with dermatomyositis (DM).

Methods

Peripheral blood mononuclear cells (PBMCs) from 23 untreated DM patients and 32 healthy controls (HC) were analysed via flow cytometry to assess MAIT cell frequency, activation status, chemokine receptor expression, and cytokine production.

Results

The frequency of circulating MAIT cells was significantly decreased in the DM group compared with the HC group, while the proportion of CD69⁺ and PD-1⁺ MAIT cells was significantly increased, especially in patients with melanoma differentiation-associated gene 5-positive dermatomyositis (MDA5-DM). Moreover, the expression of the chemokine receptors CCR2 and CCR5 on MAIT cells was reduced in DM patients. In addition, the proportion of IFN- γ ⁺ MAIT cells was significantly elevated in DM patients.

Conclusion

The reduced frequency of MAIT cells in DM may be associated with excessive activation. The increased secretion of IFN- γ by MAIT cells may contribute to DM pathogenesis.

Key words

MAIT cells, dermatomyositis, activation, chemotaxis, cytokines

Siyuan Ye, MD*
 Jun Du, MD*
 Ying Zhang, MD
 Wei Wei, MD
 Yanmei Li, MD, PhD

*Contributed equally.

Please address correspondence to:
 Wei Wei,

Department of Rheumatology
 and Immunology,
 Tianjin Medical University
 General Hospital,
 145 Anshan Street, Heping District,
 350052 Tianjin, China.

E-mail: tjweiwei2003@163.com

and to:

Yanmei Li
 E-mail: liyanmei0617@163.com

Received on July 5, 2025; accepted in
 revised form on October 7, 2025.

© Copyright CLINICAL AND
 EXPERIMENTAL RHEUMATOLOGY 2026.

Introduction

Dermatomyositis (DM) is a systemic autoimmune disease primarily affecting the skin and muscles. The distinctive skin features include Gottron's papules, heliotrope rash with periorbital oedema, V-neck erythema, and shawl sign. Muscular symptoms involve subacute progressive proximal muscle weakness and elevated serum creatine kinase (CK) levels. Common extra-muscular manifestations include cardiac involvement, interstitial lung disease (ILD), and malignancy (1). Histologically, muscle biopsy typically reveals perivascular and perifascicular inflammation, predominantly involving B lymphocytes, CD4⁺ T cells, and macrophages (2). Currently, the aetiology and pathogenesis of DM are still unclear. Susceptibility genes, environmental stressors, immune and non-immune mechanisms, and abnormal signal transduction via the interferon pathway are associated with the pathogenesis of DM (3). The innate immune response, especially interferons and interferon regulatory proteins, plays an important role in the process of DM (4).

Mucosal-associated invariant T (MAIT) cells were recently identified as innate-like lymphocytes belonging to the $\alpha\beta$ T cell subset. Unlike conventional T cells, MAIT cells possess a semi-invariant T cell receptor (TCR) composed of a conserved TCR α chain, V α 7.2-J α 33 in humans, and a limited TCR β repertoire (5). Human MAIT cells are phenotypically defined as CD3⁺ TCRV α 7.2⁺ CD161^{hi} cells and can be identified using MR1 tetramers (6). MAIT cells are the most abundant innate-like T cells, comprising up to 10% of peripheral blood T cells in healthy individuals (7). Their versatile immunological properties make them a potential therapeutic target in immunomodulation. MAIT cells have been extensively studied in infectious and autoimmune diseases (8). In infections, MAIT cells are often depleted from peripheral blood and accumulate at infection sites, suggesting that MAIT cells may be recruited to infection sites to exert their anti-infection effects (9, 10). Their roles have also been demonstrated in inflammatory bowel disease,

multiple sclerosis, systemic lupus erythematosus, and type 1 diabetes (11). However, few studies have investigated the changes of MAIT cells in DM. This study explored the frequency, phenotype and functional alterations of MAIT cells in patients with DM to clarify their clinical significance.

Materials and methods

Study subjects

From January 2021 to June 2023, 23 patients newly diagnosed with DM at Tianjin Medical University General Hospital were enrolled. The diagnosis met the 2020 ENMC criteria for DM (12). Among them, 13 were anti-MDA5 antibody-positive DM (MDA5-DM), and 10 were anti-MDA5 antibody-negative (non-MDA5-DM). The exclusion criteria were as follows: (1) the presence of other autoimmune diseases such as systemic lupus erythematosus; (2) severe comorbidities such as hypertension or diabetes; and (3) current infection. 32 age- and sex-matched healthy individuals were recruited as controls (HC). The exclusion criteria for HCs included autoimmune, metabolic, or infectious diseases. Ethical approval was obtained from the institutional review board (IRB2021-YX-005-01), and written informed consent was provided by all participants. Demographic and clinical data including sex, age, disease duration, symptoms, and laboratory parameters such as creatine Kinase (CK), erythrocyte sedimentation rate (ESR), c-reactive protein (CRP) and ferritin, as well as chest CT imaging were collected.

Surface staining of PBMCs

Peripheral blood (5 mL) was collected in EDTA tubes. Peripheral blood mononuclear cells (PBMCs) were isolated via density gradient centrifugation, counted, and adjusted to 10⁹/mL. A 100 μ L aliquot was stained with 1 μ L of Live/Dead dye (BioLegend, USA), incubated for 15 mins in the dark, then washed and resuspended. Cells were stained with 5-OP-RU tetramer (NIH, USA) and a panel of surface antibodies against CD3, CCR2, CCR5, CD69 and PD-1 (BioLegend, USA). Data were acquired via flow cytometry (BD

Funding: this work is supported by Tianjin Health Research Project (TJWJ2023QN005), Tianjin Key Clinical Specialty and Tianjin Key Medical Discipline Construction Project (TJYXZDXK-3-011C).

Competing interests: none declared.

LSRFortessa, USA) and analysed with FlowJo software version 10.8 (Tree Star, Ashland, OR, USA).

Intracellular cytokine staining

PBMCs were stimulated with leukocyte activation cocktail (BD) for 5 hours at 37°C. Cells were stained for viability and surface markers CD3, TCRV α 7.2, and CD161 (BioLegend, USA), fixed, permeabilised, and stained intracellularly for IFN- γ and TNF- α (BioLegend, USA). Data were acquired by flow cytometry.

Statistical analysis

Statistical analyses were conducted using SPSS 19.0 and GraphPad Prism 9. Parametric data were compared using independent-sample t-tests; non-parametric data were analysed via the Mann-Whitney U-test. Chi-square tests were used for categorical variables. Correlations were assessed with Pearson correlation. A *p*-value <0.05 was considered statistically significant.

Results

Characteristics of the subjects

The clinical characteristics of the subjects are described in Table I and II. Compared with non-MDA5-DM patients, MDA5-DM patients presented significantly lower serum CK levels and a higher incidence of interstitial lung disease (ILD) (Table II).

Reduced frequency of MAIT cells in peripheral blood of DM patients

MAIT cells were identified as CD3⁺5-OP-RU Tetramer⁺ (Fig. 1A). The frequency of circulating MAIT cells (as a percentage of CD3⁺ T cells) was significantly reduced in the DM group compared to the HC group (0.67±0.09% vs. 2.32±0.34%, *p*<0.0001). No significant difference was observed between MDA5-DM and non-MDA5-DM patients (0.82±0.13% vs. 0.49±0.06%) (Fig. 1B). Additionally, no clear correlation was found between MAIT cell frequency and levels of CK, CRP, or ferritin (Fig. 1C). No significant difference was observed in MAIT cell frequency between DM patients with interstitial lung disease and those without ILD (Fig. 1D).

Table I. Characteristics of recruited donors for flow cytometry.

	HC (n=32)	DM (n=23)
Age (years, range)	54.66 (24-72)	57.48 (24-79)
Sex (male/female)	14/18	11/12
Duration (months, range)	NA	3.37 (0.5-24)
CK (U/L, range)	NA	3118.39 (13-22887)
CRP (mg/dl, range)	NA	0.97 (0.10-3.08)
Ferritin (ng/ml, range)	NA	1219.31 (41.57-3589.58)
ILD (%)	NA	69.56 (16/23)

CK: creatine kinase; CRP: C reactive protein, ILD: interstitial lung disease; NA: not applicable; HC: healthy controls; DM: dermatomyositis.

Table II. Characteristics of patients with dermatomyositis for flow cytometry.

	MDA5-DM (n=13)	Non-MDA5-DM (n=10)
Age (years, range)	56.77 (33-75)	58.40 (24-79)
Sex (male/female)	5/8	6/4
Duration (months, range)	3.47 (0.5-24)	3.25 (0.5-11)
CK (U/L, range)	1195.62 (13-9963)	5618 (48-22887)*
CRP (mg/dl, range)	1.14 (0.10-3.40)	0.75 (0.17-1.48)
Ferritin (ng/ml, range)	1654.11 (70.84-3589.58)	654.06 (41.57-1122.32)
ILD (%)	92.31 (12/13)	40 (4/10)**

CK: creatine kinase; CRP: C reactive protein, ILD: interstitial lung disease.

* *p*<0.05; ** *p*<0.01.

Changes of activation markers and chemokine receptors on circulating MAIT cells

To explore the potential mechanisms underlying the reduction of MAIT cells in DM, we analysed the expression of the activation markers CD69 (early activation marker) and PD-1 (late activation marker), as well as the chemokine receptors CCR2 and CCR5. The proportions of CD69⁺ and PD-1⁺ MAIT cells were significantly elevated in the DM group compared with the HC group (CD69⁺: 56.67±3.46% vs. 41.0±2.56%, *p*=0.0023; PD-1⁺: 41.55±3.59% vs. 20.65±2.91%, *p*<0.0001) (Fig. 2A). MDA5-DM patients exhibited even higher activation levels compared to non-MDA5-DM patients (CD69⁺: 61.57±4.67% vs. 45.69±3.72%, *p*=0.02; PD-1⁺: 47.85±3.92% vs. 33.35±5.71%, *p*=0.04) (Fig. 2B). The proportion of MAIT cells expressing CCR2 and CCR5 was significantly lower in the DM group than in the HC group (CCR2⁺: 64.48±2.51% vs. 84.21±2.32%, *p*<0.0001; CCR5⁺: 56.49±4.02% vs. 78.43±2.77%, *p*<0.0001) (Fig. 2C). However, within the DM subgroup, the expression of CCR2 and CCR5 on MAIT cells was significantly greater in MDA5-DM patients than

in non-MDA5-DM patients (CCR2⁺: 70.45±2.96% vs. 56.72±2.88%, *p*=0.0038; CCR5⁺: 63.95±5.24% vs. 46.79±4.97%, *p*=0.0031) (Fig. 2D).

Altered cytokine secretion by MAIT cells

The proportion of IFN- γ ⁺ MAIT cells was significantly greater in the DM group than in the HC group (33.92±4.39% vs. 15.25±2.37%, *p*=0.003), whereas the proportion of TNF- α ⁺ MAIT cells was reduced, though not statistically significant (8.74±1.99% vs. 17.58±4.02%, *p*=0.07) (Fig. 3A). No significant differences in IFN- γ ⁺ or TNF- α ⁺ MAIT cell proportions were found between MDA5-DM and non-MDA5-DM patients (Fig. 3B).

Discussion

The pathogenesis of dermatomyositis remains poorly understood, with immune dysregulation believed to be a central driver of disease progression. Both innate and adaptive immunity contribute to DM pathophysiology, with lymphocytes and their secreted cytokines playing pivotal roles (13). MDA5-DM represents a distinct subset of DM characterised by mild muscle involvement but a high risk of rapidly progressive

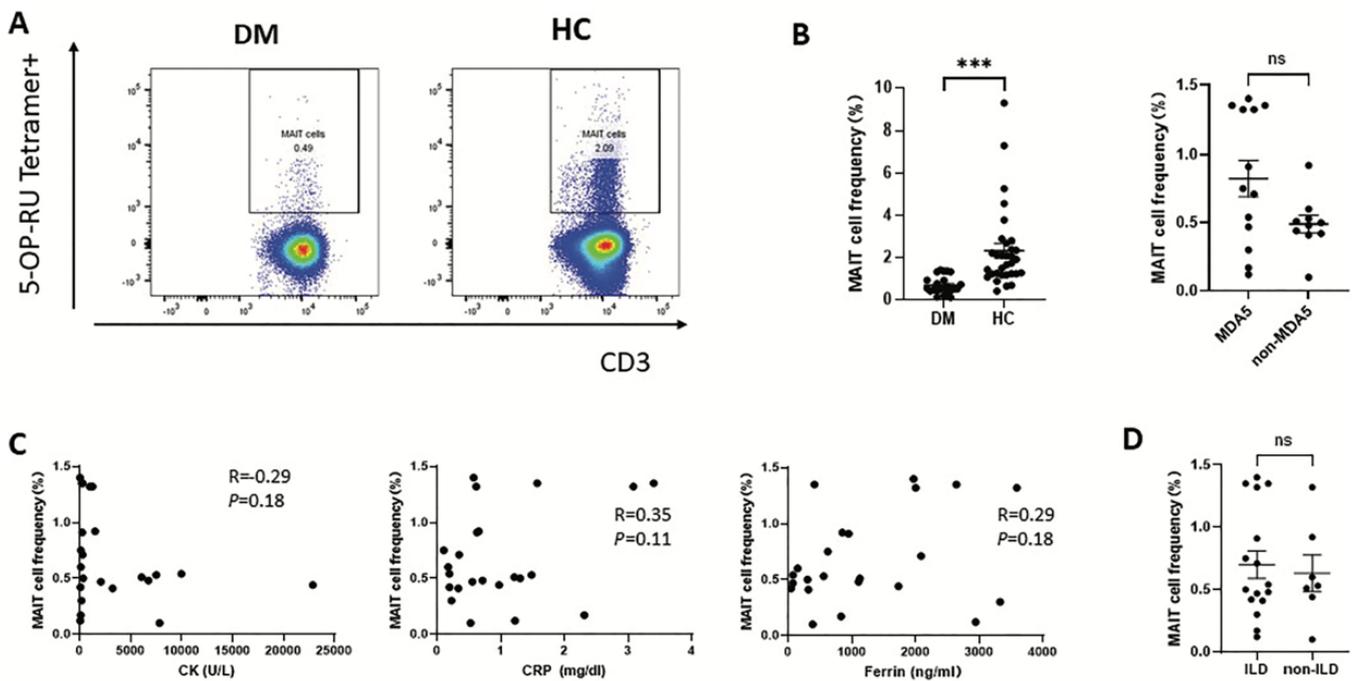


Fig. 1. Changes of peripheral blood MAIT cell frequency in DM patients.

A: Representative flow plots of MAIT cells in HCs and DM patients. **B:** MAIT cell frequency comparison between HC and DM, and between MDA5-DM and non-MDA5-DM patients. **C:** Correlations between MAIT cell frequency and clinical parameters (CK, CRP, ferritin). **D:** Comparison of MAIT cell frequency between DM patients with and without ILD.

* $p < 0.05$, *** $p < 0.001$; ns: not significant.

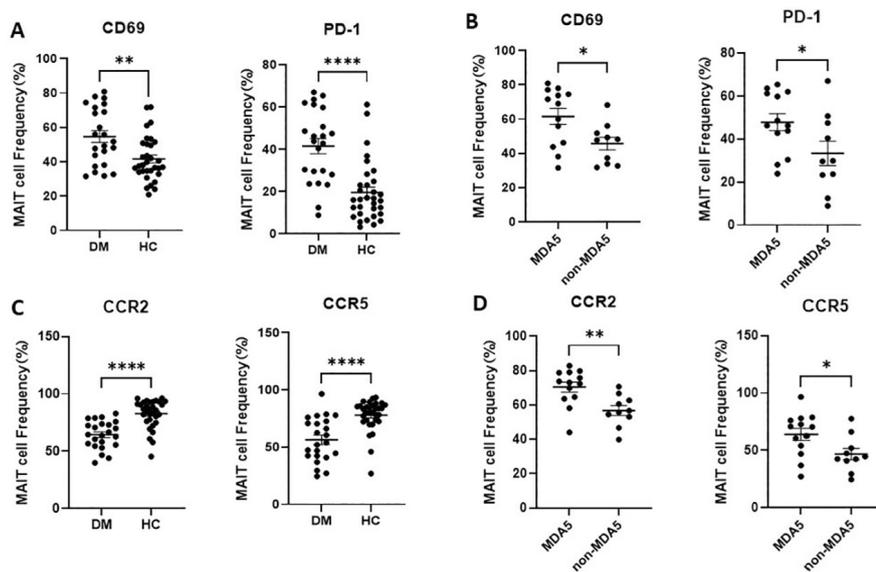


Fig. 2. Expression of activation markers and chemokine receptors on MAIT cells in DM group.

A: CD69 and PD-1 expression on MAIT cells in DM vs. HC. **B:** Comparison between MDA5-DM and non-MDA5-DM. **C:** CCR2 and CCR5 expression in DM vs. HC. **D:** Comparison between MDA5-DM and non-MDA5-DM.

* $p < 0.05$, ** $p < 0.01$, **** $p < 0.0001$.

ILD (14). MAIT cells, possessing a semi-invariant TCR that recognises riboflavin-derived microbial metabolites, display heterogeneous distribution in tissues and respond differently to various stimuli (15-17). They can be classi-

fied into MAIT1 (IFN- γ -producing) and MAIT17 (IL-17-producing) subsets and are capable of producing cytotoxic molecules (e.g. granzyme B, perforin) and diverse cytokines like Th1, Th2, and Th17 cells (18, 19).

MAIT cells act as a bridge between innate and adaptive immunity and play key roles in various autoimmune diseases (20). One study reported a reduced MAIT cell frequency in peripheral blood during active DM, which tended to normalise after treatment (21). These reductions correlate with increased expression of activation and exhaustion markers such as CD25, CD39, and CTLA-4, although they are not associated with clinical scores or biomarkers. Consistent with this finding, our study demonstrated a marked reduction in the percentage of peripheral blood MAIT cells in untreated DM patients without a correlation with clinical parameters. There were no significant differences in MAIT cell frequency between MDA5-DM and non-MDA5-DM patients, suggesting that MAIT cells may be involved in the disease process of DM patients with different antibody subtypes. The reduced frequency of MAIT cells in DM may result from activation-induced exhaustion or migration to inflamed tissues. MAIT cells express various chemokine receptors, including CCR9, CCR6, and CXCR6, enabling tissue homing (22). Endothelial cells

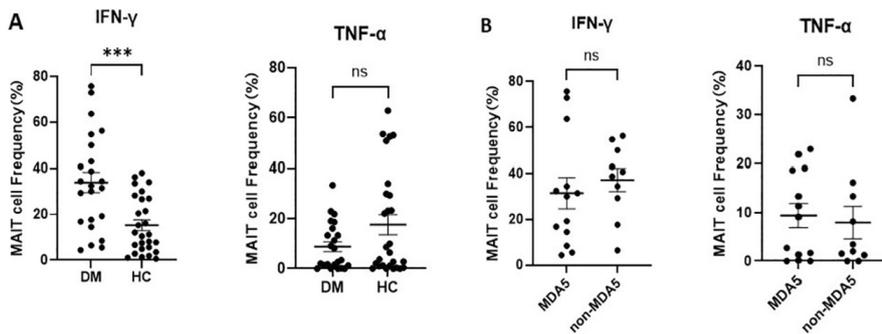


Fig. 3. Cytokine production by MAIT cells in DM.

A: IFN- γ and TNF- α production in MAIT cells from DM vs. HC. **B:** Comparison between MDA5-DM and non-MDA5-DM.

*** $p < 0.001$; ns: not significant.

in affected DM tissues reportedly express high levels of CCL2 and CCL5 (23, 24). Some studies have found that serum level of CCL2, the ligand for CCR2, was elevated in patients with DM (25, 26). In our study, CCR2 and CCR5 expression on peripheral blood MAIT cells was decreased in DM, suggesting impaired chemotactic capacity, although further confirmation in affected tissues (*e.g.* skin, muscle, and lung) is needed. Elevated expression of CD69 and PD-1 also indicates heightened activation and potential exhaustion of MAIT cells. This may represent an adaptive response to chronic inflammation, as MAIT cells are often recruited to inflamed tissues in autoimmune diseases to exert cytotoxic effects and secrete pro-inflammatory mediators (27). Notably, MDA5-DM patients exhibited higher activation and chemokine receptor expression, which coincided with the high prevalence of ILD and implied possible MAIT cell migration to the lungs. Similarly, it has been found that CD8 T cells from patients with MDA5-DM show significant exhausted phenotype than non-MDA5-DM patients and HCs (28). MAIT cell accumulation has been observed in various pulmonary diseases (29), and recent studies have shown that activated MAIT cells exhibit anti-fibrotic effects in murine bleomycin-induced lung injury (30). Whether MAIT cells infiltrate MDA5-DM-affected lungs and their precise role warrants further investigation. Activated MAIT cells secrete multiple cytokines. Our data revealed increased IFN- γ and decreased TNF- α produc-

tion in DM, although the latter was not statistically significant. Prior studies have shown that hyperactivation of type I interferon signalling correlated with disease activity in DM, with up-regulation of interferon-stimulated genes in PBMCs, muscle, and skin (31, 32). Currently, several studies also identified that the regulation of IFN- γ is also altered in DM as evidenced by elevated expression of IFN- γ in serum and in affected tissues such as lung and muscle (33, 34). The roles of both type I and IFN- γ extend beyond their conventional function of inhibiting viral replication. They play a significant role in cell differentiation, growth and survival, and elicit many immunostimulatory effects in DM (35). Therefore, MAIT cell-derived IFN- γ may contribute to DM pathogenesis. Inhibitors targeting the JAK-STAT pathway downstream of interferon signalling have shown clinical efficacy in DM, particularly MDA5-DM (36-38).

Conclusion

In summary, our study demonstrated that the frequency of MAIT cells is significantly reduced in the peripheral blood of patients with dermatomyositis, accompanied by enhanced activation and increased IFN- γ secretion, particularly in MDA5-DM patients. The expression of chemokine receptors CCR2 and CCR5 is decreased, suggesting limited chemotactic potential, although this requires validation in affected tissues. Owing to the limited sample size, further studies with larger cohorts are needed to substantiate these findings.

Acknowledgments

The MR1 tetramer technology was developed jointly by Dr James McCluskey, Dr Jamie Rossjohn, and Dr David Fairlie, and the material was produced by the NIH Tetramer Core Facility as permitted to be distributed by the University of Melbourne, Australia.

References

- SCHMIDT J: Current classification and management of inflammatory myopathies. *J Neuromuscul Dis* 2018; 5(2): 109-29. <https://doi.org/10.3233/jnd-180308>
- REED AM, ERNSTE F: The inflammatory milieu in idiopathic inflammatory myositis. *Curr Rheumatol Rep* 2009; 11(4): 295-301. <https://doi.org/10.1007/s11926-009-0041-1>
- FINDLAY AR, GOYAL NA, MOZAFFAR T: An overview of polymyositis and dermatomyositis. *Muscle Nerve* 2015; 51(5): 638-56. <https://doi.org/10.1002/mus.24566>
- HORNUNG T, WENZEL J: Innate immune-response mechanisms in dermatomyositis: an update on pathogenesis, diagnosis and treatment. *Drugs* 2014; 74(9): 981-98. <https://doi.org/10.1007/s40265-014-0240-6>
- TREINER E, DUBAN L, BAHRAM S *et al.*: Selection of evolutionarily conserved mucosal-associated invariant T cells by MR1. *Nature* 2003; 422(6928): 164-69. <https://doi.org/10.1038/nature01433>
- REANTRAGOON R, CORBETT AJ, SAKALA IG *et al.*: Antigen-loaded MR1 tetramers define T cell receptor heterogeneity in mucosal-associated invariant T cells. *J Exp Med* 2013; 210(11): 2305-20. <https://doi.org/10.1084/jem.20130958>
- DUSSEAU M, MARTIN E, SERRIARI N *et al.*: Human MAIT cells are xenobiotic-resistant, tissue-targeted, CD161hi IL-17-secreting T cells. *Blood* 2011; 117(4): 1250-59. <https://doi.org/10.1182/blood-2010-08-303339>
- TOUBAL A, NEL I, LOTERSZTAJN S, LEHUEN A: Mucosal-associated invariant T cells and disease. *Nat Rev Immunol* 2019; 19(10): 643-57. <https://doi.org/10.1038/s41577-019-0191-y>
- SALOU M, FRANCISZKIEWICZ K, LANTZ O: MAIT cells in infectious diseases. *Curr Opin Immunol* 2017; 48: 7-14. <https://doi.org/10.1016/j.coi.2017.07.009>
- JOUAN Y, GUILLON A, GONZALEZ L *et al.*: Phenotypical and functional alteration of unconventional T cells in severe COVID-19 patients. *J Exp Med* 2020; 217(12). <https://doi.org/10.1084/jem.20200872>
- MECHELLI R, ROMANO S: MAIT Cells and microbiota in multiple sclerosis and other autoimmune diseases. *nat rev immunol* 2021; 9(6). <https://doi.org/10.3390/microorganisms9061132>
- MAMMEN AL, ALLENBACH Y, STENZEL W, BENVENISTE O: 239th ENMC International Workshop: Classification of dermatomyositis, Amsterdam, the Netherlands, 14-16 December 2018. *Neuromuscul Disord* 2020; 30(1): 70-92. <https://doi.org/10.1016/j.nmd.2019.10.005>

13. FRANCO C, GATTO M, IACCARINO L, GHIRARDELLO A, DORIA A: Lymphocyte immunophenotyping in inflammatory myositis: a review. *Curr Opin Rheumatol* 2021; 33(6): 522-28. <https://doi.org/10.1097/bor.0000000000000831>
14. LU X, PENG Q: Anti-MDA5 antibody-positive dermatomyositis: pathogenesis and clinical progress. *Nat Rev Rheumatol* 2024; 20(1): 48-62. <https://doi.org/10.1038/s41584-023-01054-9>
15. CORBETT AJ, ECKLE SB, BIRKINSHAW RW *et al.*: T-cell activation by transitory neo-antigens derived from distinct microbial pathways. *Nature* 2014; 509(7500): 361-65. <https://doi.org/10.1038/nature13160>
16. KAMMANN T, CAI C: MAIT cell heterogeneity across paired human tissues reveals specialization of distinct regulatory and enhanced effector profiles. *Sci Immunol* 2024; 9(99). <https://doi.org/10.1126/sciimmunol.adn2362>
17. VORKAS CK, KRISHNA C, LI K: Single-cell transcriptional profiling reveals signatures of helper, effector, and regulatory MAIT cells during homeostasis and activation. *J Immunol* 2022; 208(5): 1042-56. <https://doi.org/10.4049/jimmunol.2100522>
18. VAN WILGENBURG B, SCHERWITZL I, HUTCHINSON EC *et al.*: MAIT cells are activated during human viral infections. *Nat Commun* 2016; 7: 11653. <https://doi.org/10.1038/ncomms11653>
19. KELLY J, MINODA Y, MEREDITH T *et al.*: Chronically stimulated human MAIT cells are unexpectedly potent IL-13 producers. *Immunol Cell Biol* 2019; 97(8): 689-99. <https://doi.org/10.1111/imcb.12281>
20. LI Y, DU J, WEI W: Emerging roles of mucosal-associated invariant T cells in rheumatology. *Front Immunol* 2022; 13: 819992. <https://doi.org/10.3389/fimmu.2022.819992>
21. CASSIUS C, BRANCHTEIN M, BATTISTELLA M *et al.*: Persistent deficiency of mucosal-associated invariant T cells during dermatomyositis. *Rheumatology* (Oxford) 2020; 59(9): 2282-86. <https://doi.org/10.1093/rheumatology/kez564>
22. KELLER AN, CORBETT AJ, WUBBEN JM, MCCLUSKEY J, ROSSJOHN J: MAIT cells and MR1-antigen recognition. *Curr Opin Immunol* 2017; 46: 66-74. <https://doi.org/10.1016/j.coi.2017.04.002>
23. DE PAEPE B, CREUS KK, DE BLEECKER JL: Chemokine profile of different inflammatory myopathies reflects humoral versus cytotoxic immune responses. *Ann NY Acad Sci* 2007; 1109: 441-53. <https://doi.org/10.1196/annals.1398.050>
24. CIVATTE M, BARTOLIC C, SCHLEINITZ N *et al.*: Expression of the beta chemokines CCL3, CCL4, CCL5 and their receptors in idiopathic inflammatory myopathies. *Neuropathol Appl Neurobiol* 2005; 31(1): 70-79. <https://doi.org/10.1111/j.1365-2990.2004.00591.x>
25. ODA K, KOTANI T, TAKEUCHI T *et al.*: Chemokine profiles of interstitial pneumonia in patients with dermatomyositis: a case control study. *Sci Rep* 2017; 7(1): 1635. <https://doi.org/10.1038/s41598-017-01685-5>
26. WANG Y, ZHENG Y, ZHAO Y *et al.*: Comparison of cytokine/chemokine profiles between dermatomyositis and anti-synthetase syndrome. *Front Neurol* 2022; 13: 1042580. <https://doi.org/10.3389/fneur.2022.1042580>
27. WATERH LTER A, WUNDERLICH M, TURNER JE: MAIT cells in immune-mediated tissue injury and repair. *Eur J Immunol* 2023; 53(12): e2350483. <https://doi.org/10.1002/eji.202350483>
28. XU YT, CAO ZY, LIN S *et al.*: Expansion of exhausted CD8⁺ T cells associates with increased pulmonary fungal infection risk in anti-melanoma differentiation associated gene 5 dermatomyositis. *Clin Exp Rheumatol* 2024; 42(2): 237-45. <https://doi.org/10.55563/clinexp/rheumatol/e51d3m>
29. KAMIKI J, GORGULHO CM, L RIAS JR, MAEURER MJ: Mucosal-associated invariant T-cells in pulmonary pathophysiology. *Curr Opin Pulm Med* 2025; 31(3): 202-10. <https://doi.org/10.1097/mcp.0000000000001163>
30. ZHANG X, LI S, LASON W *et al.*: MAIT cells protect against sterile lung injury. *Cell Rep* 2025; 44(2): 115275. <https://doi.org/10.1016/j.celrep.2025.115275>
31. GREENBERG SA: Dermatomyositis and type I interferons. *Curr Rheumatol Rep* 2010; 12(3): 198-203. <https://doi.org/10.1007/s11926-010-0101-6>
32. WALSH RJ, KONG SW, YAO Y *et al.*: Type I interferon-inducible gene expression in blood is present and reflects disease activity in dermatomyositis and polymyositis. *Arthritis Rheum* 2007; 56(11): 3784-92. <https://doi.org/10.1002/art.22928>
33. GIRIŞ M, DURMUŞ H, YETIMLER B *et al.*: Elevated IL-4 and IFN- γ levels in muscle Tissue of patients with dermatomyositis. *In Vivo* 2017; 31(4): 657-60. <https://doi.org/10.21873/invivo.11108>
34. ISHIKAWA Y, IWATA S, HANAMI K *et al.*: Relevance of interferon-gamma in pathogenesis of life-threatening rapidly progressive interstitial lung disease in patients with dermatomyositis. *Arthritis Res Ther* 2018; 20(1): 240. <https://doi.org/10.1186/s13075-018-1737-2>
35. THUNER J, COUTANT F: IFN- γ : an overlooked cytokine in dermatomyositis with anti-MDA5 antibodies. *Autoimmun Rev* 2023; 22(10): 103420. <https://doi.org/10.1016/j.autrev.2023.103420>
36. PAIK JJ, CASCIOLA-ROSEN L: Study of tofacitinib in refractory dermatomyositis: an open-label pilot study of ten patients. *Arthritis Rheumatol* 2021; 73(5): 858-65. <https://doi.org/10.1002/art.41602>
37. BECKETT M, TAN J, BONNARDEAUX E, DUTZ J: Tofacitinib therapy in refractory inflammatory myositis: a retrospective cohort study of 41 patients. *Rheumatology* (Oxford) 2024; 63(5): 1432-36. <https://doi.org/10.1093/rheumatology/kead404>
38. WU W, GUO B, SUN W *et al.*: Effectiveness and safety of tofacitinib versus calcineurin inhibitor in interstitial lung disease secondary to anti-MDA5-positive dermatomyositis: a multi-centre cohort study. *Eur Respir J* 2025; 65(5): 2401488. <https://doi.org/10.1183/13993003.01488-2024>