American Journal of Clinical Dermatology (2021) 22:339–347
https://doi.org/10.1007/s40257-020-00585-1

REVIEW ARTICLE

HLA-Cw1 and Psoriasis

Yi-Wei Huang[1] · Tsen-Fang Tsai[1,2]

Accepted: 27 December 2020 / Published online: 18 January 2021

Abstract

Psoriasis is a chronic inflammatory skin condition with regional and ethnic differences in its prevalence and clinical manifestations. *Human leukocyte antigen (HLA)-Cw6* is the disease allele conferring the greatest risk to psoriasis, but its prevalence is lower in Asian individuals. Recent studies have found associations between *HLA-Cw1* and some Asian populations with psoriasis, especially Southern Chinese. *HLA-Cw6* was associated with type I early-onset psoriasis, guttate psoriasis, Koebner phenomenon, and better response to methotrexate, interleukin (IL)-12/23, IL-17, and IL-23 targeting drugs. In contrast, *HLA-Cw1* positivity has been associated with erythrodermic psoriasis, pustular psoriasis, and the axial type of psoriatic arthritis. Furthermore, *HLA-Cw1* was more frequently associated with high-need patients who did not respond to conventional therapies. No known trigger factor nor autoantigen has been identified for *HLA-Cw1* positivity. However, *HLA-Cw1* has been linked to some viral agents. For example, cytotoxic T lymphocytes recognize multiple cytomegalovirus pp65-derived epitopes presented by HLA alleles, including HLA-C*01:02. In addition, cytomegalovirus can lead to severe exacerbation of psoriatic skin disease. The proposed interaction between viral infection, *HLA-Cw1*, and psoriasis is through the killer cell immunoglobulin-like receptors of natural killer cells. Given the diverse nature of psoriasis pathogenesis and the difference in *HLA-Cw* prevalence in different racial groups, more studies are needed to confirm the role of *HLA-Cw1* in psoriasis.

Key Points

Human leukocyte antigen (HLA)-Cw1 is a less recognized but important HLA-Cw allele associated with psoriasis in some Asian ethnicities.

Patients carrying *HLA-Cw1* tend to show higher disease activity, have an increased risk of developing erythrodermic psoriasis, and are more refractory to treatments.

1 Introduction

Psoriasis is a common inflammatory disease characterized by the infiltration of inflammatory cells into the epidermis and altered keratinocyte differentiation. Genes, immune dysregulations, and environmental triggers are the three critical factors in its pathogenesis. However, psoriasis is a heterogeneous disease presenting with different manifestations. Its prevalence varies from 0.09 to 5.1% [1]. Genome-wide association studies have identified more than 60 psoriasis susceptibility regions, which are believed to contribute to the activation of T helper-17 cells [2, 3]. In a meta-analysis of genome-wide association studies, 63 loci have been identified for European ancestry individuals [4]. Among all the psoriasis susceptibility genes, *human leukocyte antigen (HLA)-C*06:02* is the most significant risk allele. *HLA-C* is involved in the immune responses by presenting antigens to CD8+ T cells, the main inflammatory T cells that migrate into the epidermis, and by interacting with natural killer (NK) cell receptors [5, 6]. The impact of *HLA-Cw6* on psoriasis has been reviewed [7]. However, the prevalence of *HLA-C*06:02* varies widely, higher in Caucasian than in Asian individuals [7]. The frequencies of the *HLA-Cw6* allele were only 18.6% and 16.18% among Chinese patients with

✉ Tsen-Fang Tsai
tftsai@yahoo.com

[1] Department of Dermatology, National Taiwan University Hospital, No. 7 Chung San South Road, Taipei City, Taiwan

[2] Department of Dermatology, National Taiwan University College of Medicine, Taipei City, Taiwan

psoriasis [8, 9], while the prevalence was 45.9% in Finnish patients with psoriasis [10]. The effect of other *HLA-Cw* alleles on psoriasis is less studied. Recently, *HLA-C*01:02* and *HLA-A*02:07* were reported to confer a specific risk for psoriatic patients in Southern China [11]. *HLA-C*01* has also been identified as a common *HLA-C* in some other ethnicities [11–17].

There are ethnic differences in the presentation of psoriasis. Infection is a less common trigger for psoriasis in Asian individuals [18]. Atopic dermatitis in Chinese patients was shown to have more psoriasiform features [19]. A T helper-2-high psoriasis cluster has also been identified based on gene expression profiles of lesional skin specimens in a Chinese psoriasis population [20]. In specific ethnicities, some distinct characteristics were observed in *HLA-Cw1*-positive patients [21, 22]. Therefore, it is important to summarize the current reports regarding the role of *HLA-Cw*01* in the complex interplay between immunity and psoriasis.

2 Role of *HLA-Cw1* in the Immune System

Enhanced wound-healing abilities and a lower risk of infections, in particular leprosy, were reported in patients with psoriasis [23]. The resistance to infections has been partially attributed to the overexpressed antimicrobial peptides. One of which is believed to be LL-37, a cathelicidin peptide that is increased when the skin is exposed to external factors, such as skin trauma or infection [24]. LL-37 has antimicrobial activity and immunomodulatory functions, including induction of immune mediators and regulation of inflammatory responses, linking the antimicrobial defense system with the pathogenesis of psoriasis [24]. In psoriatic skin, LL-37 activates plasmacytoid dendritic cells by forming a complex with self-DNA, initiating interferon-alpha production [25]. Another study simulating the interaction confirmed the high binding affinities of smaller peptides derived from LL-37 to the *HLA-C*06:02* molecule [26]. The complex formed by LL-37 serves as an autoantigen, which interacts with a T-cell receptor, leading to the pathogenic T-cell response in psoriasis [26]. In addition, patients with *HLA-Cw*06:02* homozygotes showed significantly more improvement in the Psoriasis Area Severity Index, compared with heterozygous and *HLA-Cw*06:02*-negative patients after a tonsillectomy [27].

Other proposed autoantigens of psoriasis include a disintegrin and metalloproteinase with thrombospondin motifs (ADAMTS)-like protein 5 [28], and keratin 17 [29], a peptide constitutively expressed in hair follicles that shares extensive sequence homology with streptococcal M proteins [30]. Moreover, *HLA-C*06:02* has been linked with positive swabs indicating streptococcal throat infections and chronic or recurrent streptococcal tonsillitis [31, 32]. In contrast, *HLA-Cw*01:02* represents an HLA-C group 1 (HLA-C1) molecule and interacts with the KIR2DL2 and KIR2DL3 receptors, which normally inhibit the cytotoxicity of NK cells [33–35]. Briefly, *HLA-Cw*01:02*-binding peptides antagonize the inhibition mediated by KIR2DL2/KIR2DL3, leading to activation of NK cells [35]. In a meta-analysis of five articles, the KIR2DL2 polymorphism was significantly associated with psoriatic arthritis (odds ratio [OR] = 1.269, $p = 0.003$) [36]. Theoretically, the differences in *HLA-Cw* distribution may influence the clinical manifestations and types of trigger factors for psoriatic diseases [18, 37]. However, the immunological function of the *HLA-C* locus alleles was questioned from an evolutionary aspect [38], and there is significant linkage disequilibrium within the extended haplotype harboring *HLA-Cw*, with *A*02:07-C*01:02-B*46:01* present in the Southern China population [39, 40]. Thus, further studies are required to investigate the roles of *HLA-C*01* and its corresponding antigens/autoantigens in the pathogenesis of psoriasis.

3 *HLA-Cw1* Allele Frequency in Patients with Psoriasis

There is evidence supporting the association between *HLA-Cw1* and psoriatic diseases in different ethnic groups (Table 1), especially in Thai [12] and Southern China populations [11]. In Thai patients, the *HLA-Cw1* allele frequency of type I early-onset psoriasis compared with controls was 35% vs 16% ($p < 0.05$) [13]. Type II psoriasis, which was characterized by an age at onset greater than 40 years, also exhibited a higher allele frequency of Cw1 at 29% compared with controls at 16% ($p < 0.05$) [13].

In a study that compared the HLA susceptibility of 4493 patients with psoriasis and 4649 controls from Southern China, and 3657 patients with psoriasis and 5257 controls from Northern China, *HLA-C*06:02* and *HLA-C*07:04* were significantly increased in both patient groups. However, *HLA-C*01:02* (OR = 1.32, $p = 2.45 \times 10^{-7}$) and *HLA-A*02:07* (OR = 1.67, $p = 2.49 \times 10^{-11}$) were associated with an increased risk of psoriasis only in patients from Southern China using a logistic regression and stepwise conditional analysis [11]. *HLA-C*06:02* and *HLA-C*07:04* showed similarities in peptide binding preference [41], whereas *HLA-C*01* and *HLA-A*02* [42] have very similar binding motifs, suggesting possible yet unidentified antigen/autoantigens in the pathogenesis of patients with psoriasis with *HLA-Cw1* positivity. Interestingly, *HLA-A*02:07* was also reported to be associated with psoriasis in Japanese individuals [43].

Additionally, the *HLA-C*01* allele was significantly more frequent in Turkish patients with psoriasis than in control groups (8.1% vs 2.1%, $p = 0.02$), leading to an OR of 4.15 (95% confidence interval [CI] 1.15–15.01) [14]. In

Table 1 Summary of *HLA-Cw1* serotype frequency, positivity, and samples sizes of patients with psoriasis and non-psoriatic controls in different populations

References	Country	*HLA-Cw1* serotype frequency in patients (%)	*HLA-Cw1* serotype frequency in controls (%)	*HLA-Cw1* positivity in patients (%)	*HLA-Cw1* positivity in controls (%)	Number of patients	Number of controls
Cai et al. [11]	Southern China	12.7	16.8			3657	5257
Cardili et al. [88]	Brazil	1.6	1.7			125	202
Chiu et al. [54]	Taiwan	27	24			398	400
Choonhakarn et al. [13]	Thailand	26	16			140	300
Cibulova et al. [89]	Czech Republic	6.4	7.1			153	99
Gonzalez et al. [90]	Spain			10	7.3	110	177
Kim et al. [91]	South Korea	22.6	21.4			84	98
Nakagawa et al. [92]	Japan			14	27	56	100
Onsun et al. [14]	Turkey	8.1	2.1			150	145
Shaiq et al. [16]	Pakistan	4.73	2.90			351	593
Shawkatová et al. [93]	Slovakia	4.42	6.07			147	107
Szczerkowska Dobosz et al. [94]	Poland			2.4	5	41	80
Tiilikainen et al. [10]	Finland			13.5	14.3	37	483
Tsai et al. [9]	Taiwan	22.14	19.95			136	426
Zhang et al. [95]	China	7.5	10.1			166	204

one study that recruited Kuwait children, the association of *HLA-Cw1* with psoriasis was observed in 9 out of 50, resulting in an OR of 3.01 (95% CI 1.00–9.50) [15]. In the same study, among 25 patients with a positive family history, seven patients carried the *HLA-Cw1* allele, indicating an OR of 5.44 (95% CI 1.46–19.35) [15]. Another study focusing on the Pakistani population revealed an association between *HLA-Cw1* and psoriasis (OR = 1.66) [16]. Significant *HLA-C* allele associations were observed in Singapore Chinese patients with psoriasis, with an OR of 2.19 (95% CI 1.45–3.33) for *HLA-Cw1* [17]. However, no association between *HLA-Cw1* and Caucasian patients with psoriasis was observed [12].

4 *HLA-Cw1* and Clinical Presentations of Psoriasis

The strong connections between *HLA-Cw6* and psoriatic diseases have been well documented [7]. One review article highlighted that the allele is associated with early-onset psoriasis, guttate psoriasis, psoriatic arthritis, and Koebner phenomenon [7]. Patients with psoriatic arthritis with *HLA-Cw6* positivity more often have an early onset, and their cutaneous symptoms often develop before arthritis [7]. *HLA-Cw6*-positive patients have been shown in several studies to be more responsive to methotrexate [44, 45], anti-interleukin (IL)-12/23 [46, 47], anti-IL-17 [48, 49] and IL-23 drugs [50]. However, inconsistent data were reported in patients receiving secukinumab [51]. Moreover, anti-tumor necrosis factor agents showed less efficacy in patients with *HLA-Cw6*-positive psoriasis [48]. In comparison, *HLA-Cw1-B46* carriers were reported to be clinically distinct, showing a lower risk of disease, greater nail involvement, and a later age at onset [12]. Interestingly, the *HLA-Cw1-B46* haplotype imparts a risk for Asian individuals [12].

Erythrodermic psoriasis is a potentially fatal presentation of psoriasis. A higher frequency of severe psoriasis was reported in Asian individuals, [52] as well as erythrodermic psoriasis (OR = 5.56, $p = 0.018$) [37]. In Chinese patients with erythrodermic psoriasis, *HLA-C*01:02* was reported to be the most frequent *HLA-C* allele (34.4%).]21]. Regarding the allele frequencies of individuals carrying *HLA-C*01:02,* patients with plaque psoriasis (21.9%) had a significantly lower frequency than those with erythrodermic psoriasis (34.4%, $p = 0.02$), but was similar to healthy controls (21.2%) [21]. *HLA-Cw1* phenotype frequency is significantly increased in Japanese patients with generalized pustular psoriasis, standing at 46.2% compared with 22.2% in healthy controls [22]. Based on a study recruiting Thai patients with psoriasis, a significant increase of MICA*010 in patients with psoriasis represents a marker of the *HLA-B46-Cw1* haplotype [53].

The severity of psoriasis was also found to be associated with *HLA-Cw1* in a Chinese study. The *HLA-Cw1* allele was significantly increased in patients with moderate-to-severe psoriasis compared with all patients with psoriasis (32% vs 22%, $p = 0.023$) [54]. The authors also highlighted the elevated allele positivity of *HLA-Cw1-B46* compared with all patients with psoriasis (49% vs 29%, $p = 0.004$). The *HLA-Cw1-B46* allele was associated with early-onset (age <40 years) psoriasis ($p = 0.012$), but not late-onset psoriasis ($p = 0.065$) [54]. In the same study, differences in the Psoriasis Area Severity Index 50 response to alefacept at week 12 was significant between *HLA-Cw1*-positive and *HLA-Cw1*-negative individuals (0% vs 57%, $p = 0.026$).

5 *HLA-Cw1* and Psoriatic Arthritis

A high prevalence of *HLA-Cw1* antigens has been described in spondylarthritis since 1978, [55–58] and in patients with the spondylitis type of psoriatic arthritis [59]. The *HLA-Cw1* allele was increased in Spanish patients with psoriatic arthritis, especially the axial type, although this association may be secondary to the linkage between *HLA-B27.5* and *HLA-Cw1* [60]. In one study, genotyping was performed in 47 Chinese patients with active peripheral-type psoriatic arthritis despite conventional treatment, *HLA-Cw*07:02* was the most frequent allele (29.8%), followed by *HLA-C*01* (26.6%) [61]. Shao et al. described four phenotypes with a significant positive association with psoriatic arthritis, including *HLA-C*01*, **02*, **06*, and **12*, in a meta-analysis of European and Middle Eastern descent [62].

6 *HLA-Cw1* and Infectious Agents

Genetic variations in HLA may be the result of evolutionary adaptation in response to environmental stress, such as climate and the prevalence of infectious diseases [63]. *HLA-C* plays an essential role in the protection against cancers and viruses, and has also been implicated in rheumatic diseases, including psoriasis and psoriatic arthritis [64]. The HLA serotype may also play a role in the global coronavirus pandemic. In a recent Italian study, *HLA-B*44* and *HLA-C*01* were found to be positively and independently associated with COVID-19 with a growth rate of 16% per 1% point increase in *HLA-B*44* prevalence and of 19% per 1% point increase in *HLA-C*01* prevalence [65].

The proposed function of *HLA-C* in autoimmune and inflammatory diseases is to present antigens to T cells and to drive the innate immunity through binding activating or inhibitory receptors on NK cells [64]. *HLA-Cw1* was increased in patients with various clinical forms of tuberculosis, [66]. which is possibly due to the inhibition of NK cell activity through killer cell immunoglobulin-like receptors [35]. However, a meta-analysis of case-control studies failed to confirm this association [67]. Among the *HLA-Cw* homozygous subjects in a Han cohort seropositive with human immunodeficiency virus type 1, *HLA-Cw*01:02* was the predominant allele [68]. *HLA-Cw*01:02*-presented human immunodeficiency virus type 1 p24 peptide modulates the inhibitory receptor KIR2DL2, leading to functional inhibition of NK cells [6, 69]. Increased risks for tuberculosis onset as well as inflammatory reconstitution inflammatory syndrome were reported to be associated with the *KIR2DS2* gene, [70]. demonstrating the participation of HLA-C1 in the activation of NK cells [71].

The relationship between viral infection, psoriasis, and HLA is complicated. Human immunodeficiency virus infection increased the incidence of psoriasis in different reports [72–74]. Gambardella et al. [75] reported a patient presenting with cytomegalovirus (CMV), followed by a severe aggravation of psoriasis. Serology investigations of the patient revealed persistent positive IgM anti-CMV > 28 UA/mL, positive IgG anti-CMV, yet negative results for CMV-polymerase chain reaction and IgG anti-CMV avidity [75]. An interactive relationship between the severity of psoriasis and CMV infection has been proposed [76]. To be more specific, reduced circulating CMV-specific T cells were observed in patients with CMV-seropositive psoriasis who received effective anti-psoriatic treatment compared to CMV-positive healthy controls [76]. Cytotoxic T lymphocytes recognize multiple CMV pp65-derived epitopes presented by HLA alleles, including HLA-C*01:02 [77]. In an analysis based on each allotype in the *HLA-C* locus, the frequency of CMV *pp65*-specific CD8+ T cells secreting interferon-γ was the highest for *HLA-C*08:01*, followed by *HLA-C*01:02* [78].

Further evidence supporting the involvement of infectious agents in the pathogenesis of psoriasis through killer cell immunoglobulin-like receptors and NK cells lies in the treatment of psoriasis. In parallel to anti-psoriatic therapy, an enhanced proportion of acute activated CD8+ T cells were replaced by effector differentiated CD8+ T cells in CMV-seropositive patients with severe psoriasis [76]. In contrast to LL-37, a proposed autoantigen for *HLA-Cw6*, [26] there is no confirmed T-cell autoantigen for *HLA-Cw1* to our knowledge. The absence of obvious triggering antigens and the presence of highly prevalent (95%) CMV infection among patients with psoriasis [79] might explain the persistent of more refractory disease activity in patients with *HLA-Cw1* positivity. More research is needed to elucidate whether *HLA-Cw1* plays a role in triggering or influencing the disease severity of psoriasis.

Table 2 Summary of ethnicities, clinical features, autoantigens, ligands, infections, and comorbidities associated with *HLA-Cw1* and *HLA-Cw6* serotype

	HLA-Cw1	HLA-Cw6[a]
Ethnicities	Kuwait [15] Pakistan [16] Singapore Chinese [17] Southern Chinese [11] Thai [12] Turkish [14]	All ethnicities (higher in Caucasians)
Phenotypes	Erythrodermic psoriasis [21] Pustular psoriasis [22] Nail involvement [12] Positive family history [15]	Guttate psoriasis Photosensitive Early-onset (type I) Psoriatic arthritis
Treatments	Refractory to alefacept [54]	Good response to methotrexate, anti-IL-12/23, anti-IL-17[b], anti-IL-23 agents [50] Less responsive to anti-TNF agents [48]
Autoantigens	–	LL-37 ADAMTSL5 Keratin 17 [29, 30]
KIRs as ligands	KIR2DL2 [6, 69] KIR2DL3 [33–35]	KIR2DL1
Infections	CMV [76, 77]	Streptococcal pharyngitis HIV[b]
Comorbidities	Dyslipidemia [17, 80] Crohn's disease [81–83] Schizophrenia [84–87]	Overweight Atherosclerosis

ADAMTS a disintegrin and metalloproteinase with thrombospondin motifs, *CMV* cytomegalovirus, *HIV* human immunodeficiency virus, *HLA* human leukocyte antigen, *IL* interleukin, *KIRs* killer cell immunoglobulin-like receptors, *TNF* tumor necrosis factor

[a]Data of HLA-Cw6 were summarized from the review article of Chen et al. [7] unless otherwise specified

[b]Inconsistent evidence reported

7 *HLA-Cw1* and Other Comorbidities of Psoriasis

Psoriasis is linked to systemic inflammation and multiple comorbidities, which is often related to the severity of skin lesions and coexisting psoriatic arthritis [17, 80].

An increased cardiometabolic burden has been highlighted because of the risk of significant morbidities and mortalities. The association of *HLA-Cw1* and dyslipidemia was reported in Singapore Chinese patients [17]. Another Japanese cohort demonstrated *BTN2A1*, a gene within the major histocompatibility complex class I region, may play a role in the higher frequency of dyslipidemia in patients with psoriasis [80].

In a meta-analysis, patients with psoriasis showed a 2.53-fold risk of developing Crohn's disease and a 1.7-fold risk of developing ulcerative colitis, [81] which could be explained by shared susceptibility loci [82]. The *HLA-C*01* allotype has been correlated with an increased risk of Crohn's disease in a large genome-wide association study performed on Korean individuals [83]. In systemic reviews and meta-analyses, patients with psoriasis were also found to have a higher risk of schizophrenia [84] and vice versa [85]. In the Irish Schizophrenia Genomics Consortium, which recruited 1606 patients and 1794 controls, *HLA-C*01:02* was identified as the most significant HLA associated with schizophrenia [86]. A shared genetic risk between psoriasis and schizophrenia has been observed [87].

8 Conclusions

There is a growing understanding of the immunopathogenesis of immune-mediated inflammatory diseases, such as psoriasis. Involvement of a host gene polymorphism along with the interaction between infectious agents and killer cell immunoglobulin-like receptors of NK cells have been proposed based on investigations of viral infections such as CMV. A summary of characteristics of patients with psoriasis carrying *HLA-Cw1* in comparison to *HLA-Cw6* is provided in Table 2. *HLA-Cw6* is the most well-recognized HLA serotype, affecting susceptibility, phenotype, disease

course, and response to the treatment of psoriasis. Patients with *HLA-Cw6* also respond better to conventional treatments and some biologics despite more extensive plaques. *HLA-Cw1* is a less recognized but an important *HLA-C* serotype associated with psoriasis in some Asian ethnicities. Patients carrying *HLA-Cw1* tend to show higher disease activity, have an increased risk of developing erythrodermic psoriasis, and are more refractory to treatments. Compared to *HLA-Cw6* patients, there is a lack of identified exogenous triggers or autoantigens in *HLA-Cw1* patients with psoriasis. Future research is needed to elucidate the role of *HLA-Cw1* in psoriasis.

Declarations

Funding No funding was received for the preparation of this article.

Conflict of Interest Yi-Wei Huang has no conflicts of interest that are directly relevant to the content of this article. Tsen-Fang Tsai has conducted clinical trials or received honoraria for serving as a consultant for Abbvie, Boehringer Ingelheim, Bristol-Myers Squibb, Celgene, Eli Lilly, Galderma, Janssen-Cilag, Merck Sharp & Dohme, Novartis International AG, Pfizer Inc., and UCB Pharma.

Ethics Approval Not applicable.

Consent to Participate Not applicable.

Consent for Publication Not applicable.

Availability of Data and Material Not applicable.

Code Availability Not applicable.

Authors' Contributions All authors contributed to the study conception and design. The literature search and data analysis were performed by Yi-Wei Huang and Tsen-Fang Tsai. The first draft of the manuscript was written by Yi-Wei Huang and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

References

1. Michalek IM, Loring B, John SM. A systematic review of worldwide epidemiology of psoriasis. J Eur Acad Dermatol Venereol. 2017;31(2):205–12. https://doi.org/10.1111/jdv.13854.
2. Singh S, Pradhan D, Puri P, Ramesh V, Aggarwal S, Nayek A, et al. Genomic alterations driving psoriasis pathogenesis. Gene. 2019;683:61–71. https://doi.org/10.1016/j.gene.2018.09.042.
3. Capon F. The genetic basis of psoriasis. Int J Mol Sci. 2017;18(12):2526. https://doi.org/10.3390/ijms18122526.
4. Tsoi LC, Stuart PE, Tian C, Gudjonsson JE, Das S, Zawistowski M, et al. Large scale meta-analysis characterizes genetic architecture for common psoriasis associated variants. Nat Commun. 2017;8:15382. https://doi.org/10.1038/ncomms15382.
5. Prinz JC. Autoimmune aspects of psoriasis: heritability and autoantigens. Autoimmun Rev. 2017;16(9):970–9. https://doi.org/10.1016/j.autrev.2017.07.011.
6. Zipeto D, Beretta A. HLA-C and HIV-1: friends or foes? Retrovirology. 2012;9:39. https://doi.org/10.1186/1742-4690-9-39.
7. Chen L, Tsai T-F. HLA-Cw6 and psoriasis. Br J Dermatol. 2018;178(4):854–62. https://doi.org/10.1111/bjd.16083.
8. Chang YT, Tsai SF, Lee DD, Shiao YM, Huang CY, Liu HN, et al. A study of candidate genes for psoriasis near HLA-C in Chinese patients with psoriasis. Br J Dermatol. 2003;148(3):418–23. https://doi.org/10.1046/j.1365-2133.2003.05166.x.
9. Tsai TF, Hu CY, Tsai WL, Chu CY, Lin SJ, Liaw SH, et al. HLA-Cw6 specificity and polymorphic residues are associated with susceptibility among Chinese psoriatics in Taiwan. Arch Dermatol Res. 2002;294(5):214–20. https://doi.org/10.1007/s00403-002-0324-0.
10. Tiilikainen A, Lassus A, Karvonen J, Vartiainen P, Julin M. Psoriasis and HLA-Cw6. Br J Dermatol. 1980;102(2):179–84. https://doi.org/10.1111/j.1365-2133.1980.tb05690.x.
11. Cai M, Huang H, Ran D, Zheng X, Wen L, Zhu Z, et al. HLA-C*01:02 and HLA-A*02:07 confer risk specific for psoriatic patients in Southern China. J Invest Dermatol. 2019;139(9):2045-8.e4. https://doi.org/10.1016/j.jid.2019.02.027.
12. Stuart PE, Nair RP, Hiremagalore R, Kullavanijaya P, Kullavanijaya P, Tejasvi T, et al. Comparison of MHC class I risk haplotypes in Thai and Caucasian psoriatics shows locus heterogeneity at PSORS1. Tissue Antigens. 2010;76(5):387–97. https://doi.org/10.1111/j.1399-0039.2010.01526.x.
13. Choonhakarn C, Romphruk A, Puapairoj C, Jirarattanapochai K, Romphruk A, Leelayuwat C. Haplotype associations of the major histocompatibility complex with psoriasis in Northeastern Thais. Int J Dermatol. 2002;41(6):330–4. https://doi.org/10.1046/j.1365-4362.2002.01496.x.
14. Onsun N, Pirmit S, Ozkaya D, Çelik Ş, Rezvani A, Cengiz FP, et al. The HLA-Cw12 allele is an important susceptibility allele for psoriasis and is associated with resistant psoriasis in the Turkish population. Sci World J. 2019;2019:7848314. https://doi.org/10.1155/2019/7848314.
15. Nanda A, Al-Fouzan AS, El-Kashlan M, Al-Sweih N, Al-Muzairai I. Salient features and HLA markers of childhood psoriasis in Kuwait. Clin Exp Dermatol. 2000;25(2):147–51. https://doi.org/10.1046/j.1365-2230.2000.00598.x.
16. Shaiq PA, Stuart PE, Latif A, Schmotzer C, Kazmi AH, Khan MS, et al. Genetic associations of psoriasis in a Pakistani population. Br J Dermatol. 2013;169(2):406–11. https://doi.org/10.1111/bjd.12313.
17. Shen M, Lim SWD, Tan ES, Oon HH, Ren EC. HLA correlations with clinical phenotypes and risk of metabolic comorbidities in Singapore Chinese psoriasis patients. Mol Diagn Ther. 2019;23(6):751–60. https://doi.org/10.1007/s40291-019-00423-z.
18. Yan D, Afifi L, Jeon C, Cordoro KM, Liao W. A cross-sectional study of psoriasis triggers among different ethno-racial groups. J Am Acad Dermatol. 2017;77(4):756-8.e1. https://doi.org/10.1016/j.jaad.2017.04.1109.
19. Chan TC, Sanyal RD, Pavel AB, Glickman J, Zheng X, Xu H, et al. Atopic dermatitis in Chinese patients shows T(H)2/T(H)17 skewing with psoriasiform features. J Allergy Clin Immunol. 2018;142(3):1013–7. https://doi.org/10.1016/j.jaci.2018.06.016.
20. Chen J, Li C, Li H, Yu H, Zhang X, Yan M, et al. Identification of a T(H) 2-high psoriasis cluster based on skin biomarker analysis in a Chinese psoriasis population. J Eur Acad Dermatol Venereol. 2020. https://doi.org/10.1111/jdv.16563.
21. Lo Y, Chiu HY, Tsai TF. Clinical features and genetic polymorphism in Chinese patients with erythrodermic psoriasis in a single dermatologic clinic. Mol Diagn Ther. 2019;24(1):85–93. https://doi.org/10.1007/s40291-019-00441-x.
22. Ozawa A, Miyahara M, Sugai J, Iizuka M, Kawakubo Y, Matsuo I, et al. HLA class I and II alleles and susceptibility to generalized pustular psoriasis: significant associations with HLA-Cw1 and

HLA-DQB1*0303. J Dermatol. 1998;25(9):573–81. https://doi.org/10.1111/j.1346-8138.1998.tb02461.x.
23. Romaní de Gabriel J. Darwinian medicine and psoriasis. Actas Dermosifiliogr. 2015;106(3):189–94. https://doi.org/10.1016/j.ad.2014.06.009.
24. Méndez-Samperio P. The human cathelicidin hCAP18/LL-37: a multifunctional peptide involved in mycobacterial infections. Peptides. 2010;31(9):1791–8. https://doi.org/10.1016/j.peptides.2010.06.016.
25. Lande R, Gregorio J, Facchinetti V, Chatterjee B, Wang Y-H, Homey B, et al. Plasmacytoid dendritic cells sense self-DNA coupled with antimicrobial peptide. Nature. 2007;449(7162):564–9. https://doi.org/10.1038/nature06116.
26. Mabuchi T, Hirayama N. Binding affinity and interaction of LL-37 with HLA-C*06:02 in psoriasis. J Invest Dermatol. 2016;136(9):1901–3. https://doi.org/10.1016/j.jid.2016.04.033.
27. Thorleifsdottir RH, Sigurdardottir SL, Sigurgeirsson B, Olafsson JH, Petersen H, Sigurdsson MI, et al. HLA-Cw6 homozygosity in plaque psoriasis is associated with streptococcal throat infections and pronounced improvement after tonsillectomy: a prospective case series. J Am Acad Dermatol. 2016;75(5):889–96. https://doi.org/10.1016/j.jaad.2016.06.061.
28. Arakawa A, Siewert K, Stöhr J, Besgen P, Kim SM, Rühl G, et al. Melanocyte antigen triggers autoimmunity in human psoriasis. J Exp Med. 2015;212(13):2203–12. https://doi.org/10.1084/jem.20151093.
29. Yunusbaeva M, Valiev R, Bilalov F, Sultanova Z, Sharipova L, Yunusbayev B. Psoriasis patients demonstrate HLA-Cw*06:02 allele dosage-dependent T cell proliferation when treated with hair follicle-derived keratin 17 protein. Sci Rep. 2018;8(1):6098. https://doi.org/10.1038/s41598-018-24491-z.
30. Johnston A, Gudjonsson JE, Sigmundsdottir H, Love TJ, Valdimarsson H. Peripheral blood T cell responses to keratin peptides that share sequences with streptococcal M proteins are largely restricted to skin-homing CD8(+) T cells. Clin Exp Immunol. 2004;138(1):83–93. https://doi.org/10.1111/j.1365-2249.2004.00600.x.
31. Haapasalo K, Koskinen LLE, Suvilehto J, Jousilahti P, Wolin A, Suomela S, et al. The psoriasis risk allele HLA-C*06:02 shows evidence of association with chronic or recurrent streptococcal tonsillitis. Infect Immun. 2018;86(10):e00304-e318. https://doi.org/10.1128/iai.00304-18.
32. Mallbris L, Wolk K, Sánchez F, Ståhle M. HLA-Cw*0602 associates with a twofold higher prevalence of positive streptococcal throat swab at the onset of psoriasis: a case control study. BMC Dermatol. 2009;9(1):5. https://doi.org/10.1186/1471-5945-9-5.
33. Winter CC, Gumperz JE, Parham P, Long EO, Wagtmann N. Direct binding and functional transfer of NK cell inhibitory receptors reveal novel patterns of HLA-C allotype recognition. J Immunol. 1998;161(2):571.
34. Moretta A, Vitale M, Bottino C, Orengo AM, Morelli L, Augugliaro R, et al. P58 molecules as putative receptors for major histocompatibility complex (MHC) class I molecules in human natural killer (NK) cells: anti-p58 antibodies reconstitute lysis of MHC class I-protected cells in NK clones displaying different specificities. J Exp Med. 1993;178(2):597–604. https://doi.org/10.1084/jem.178.2.597.
35. Fadda L, Borhis G, Ahmed P, Cheent K, Pageon SV, Cazaly A, et al. Peptide antagonism as a mechanism for NK cell activation. Proc Natl Acad Sci U S A. 2010;107(22):10160–5. https://doi.org/10.1073/pnas.0913745107.
36. Enciso-Vargas M, Alvarado-Ruíz L, Suárez-Villanueva AS, Macías-Barragán J, Montoya-Buelna M, Oceguera-Contreras E, et al. Association study between psoriatic arthritis and killer immunoglobulin-like receptor (KIR) genes: a meta-analysis. Immunol Invest. 2020;2020:1–12. https://doi.org/10.1080/08820139.2020.1713145.
37. Yan D, Afifi L, Jeon C, Cordoro KM, Liao W. A cross-sectional study of the distribution of psoriasis subtypes in different ethnoracial groups. Dermatol Online J. 2018;24(7):13030.
38. Güssow D, Rein RS, Meijer I, de Hoog W, Seemann GH, Hochstenbach FM, et al. Isolation, expression, and the primary structure of HLA-Cw1 and HLA-Cw2 genes: evolutionary aspects. Immunogenetics. 1987;25(5):313–22. https://doi.org/10.1007/bf00404424.
39. Shen Y, Cao D, Li Y, Kulski JK, Shi L, Jiang H, et al. Distribution of HLA-A, -B, and -C alleles and HLA/KIR combinations in Han population in China. J Immunol Res. 2014;2014:565296. https://doi.org/10.1155/2014/565296.
40. Chen N, Wang W, Wang F, Dong L, Zhao S, Zhang W, et al. The distributions of HLA-A, HLA-B, HLA-C, HLA-DRB1 and HLA-DQB1 allele and haplotype at high-resolution level in Zhejiang Han population of China. Int J Immunogenet. 2019;46(1):7–16. https://doi.org/10.1111/iji.12411.
41. Pavlos R, McKinnon EJ, Ostrov DA, Peters B, Buus S, Koelle D, et al. Shared peptide binding of HLA Class I and II alleles associate with cutaneous nevirapine hypersensitivity and identify novel risk alleles. Sci Rep. 2017;7(1):8653. https://doi.org/10.1038/s41598-017-08876-0.
42. Andersen MH, Søndergaard I, Zeuthen J, Elliott T, Haurum JS. An assay for peptide binding to HLA-Cw*0102. Tissue Antigens. 1999;54(2):185–90. https://doi.org/10.1034/j.1399-0039.1999.540210.x.
43. Hirata J, Hirota T, Ozeki T, Kanai M, Sudo T, Tanaka T, et al. Variants at HLA-A, HLA-C, and HLA-DQB1 confer risk of psoriasis vulgaris in Japanese. J Invest Dermatol. 2018;138(3):542–8. https://doi.org/10.1016/j.jid.2017.10.001.
44. West J, Ogston S, Berg J, Palmer C, Fleming C, Kumar V, et al. HLA-Cw6-positive patients with psoriasis show improved response to methotrexate treatment. Clin Exp Dermatol. 2017;42(6):651–5. https://doi.org/10.1111/ced.13100.
45. Indhumathi S, Rajappa M, Chandrashekar L, Ananthanarayanan PH, Thappa DM, Negi VS. Pharmacogenetic markers to predict the clinical response to methotrexate in south Indian Tamil patients with psoriasis. Eur J Clin Pharmacol. 2017;73(8):965–71. https://doi.org/10.1007/s00228-017-2255-x.
46. Chiu HY, Wang TS, Chan CC, Cheng YP, Lin SJ, Tsai TF. Human leucocyte antigen-Cw6 as a predictor for clinical response to ustekinumab, an interleukin-12/23 blocker, in Chinese patients with psoriasis: a retrospective analysis. Br J Dermatol. 2014;171(5):1181–8. https://doi.org/10.1111/bjd.13056.
47. van Vugt LJ, van den Reek J, Hannink G, Coenen MJH, de Jong E. Association of HLA-C*06:02 status with differential response to ustekinumab in patients with psoriasis: a systematic review and meta-analysis. JAMA Dermatol. 2019;155(6):708–15. https://doi.org/10.1001/jamadermatol.2019.0098.
48. Burlando M, Russo R, Clapasson A, Carmisciano L, Stecca A, Cozzani E, et al. The HLA-Cw6 dilemma: is it really an outcome predictor in psoriasis patients under biologic therapy? A monocentric retrospective analysis. J Clin Med. 2020;9(10):3140. https://doi.org/10.3390/jcm9103140.
49. Morelli M, Galluzzo M, Madonna S, Scarponi C, Scaglione GL, Galluccio T, et al. HLA-Cw6 and other HLA-C alleles, as well as MICB-DT, DDX58 and TYK2 genetic variants associate with optimal response to anti-IL-17A treatment in patients with psoriasis. Expert Opin Biol Ther. 2020. https://doi.org/10.1080/14712598.2021.1862082.
50. Liu X, DePrimo S, Chen Y, Li S, Munoz-Elias E. Association between HLA-Cw6 status and response to guselkumab in patients with moderate to severe plaque psoriasis. J Invest

Dermatol. 2018;138(5 Suppl.):S76. https://doi.org/10.1016/j.jid.2018.03.458.
51. Costanzo A, Bianchi L, Flori ML, Malara G, Stingeni L, Bartezaghi M, et al. Secukinumab shows high efficacy irrespective of HLA-Cw6 status in patients with moderate-to-severe plaque-type psoriasis: SUPREME study. Br J Dermatol. 2018;179(5):1072–80. https://doi.org/10.1111/bjd.16705.
52. Abrouk M, Lee K, Brodsky M, Nakamura M, Singh R, Zhu TH, et al. Ethnicity affects the presenting severity of psoriasis. J Am Acad Dermatol. 2017;77(1):180–2. https://doi.org/10.1016/j.jaad.2017.02.042.
53. Romphruk AV, Romphruk A, Choonhakarn C, Puapairoj C, Inoko H, Leelayuwat C. Major histocompatibility complex class I chain-related gene A in Thai psoriasis patients: MICA association as a part of human leukocyte antigen-B-Cw haplotypes. Tissue Antigens. 2004;63(6):547–54. https://doi.org/10.1111/j.0001-2815.2004.00238.x.
54. Chiu HY, Huang PY, Jee SH, Hu CY, Chou CT, Chang YT, et al. HLA polymorphism among Chinese patients with chronic plaque psoriasis: subgroup analysis. Br J Dermatol. 2012;166(2):288–97. https://doi.org/10.1111/j.1365-2133.2011.10688.x.
55. Kozin F, Duquesnoy R, Rodey GE, Lightfoot RW, Ryan LM. High prevalence of HLA-Cw1 and Cw2 antigens in spondylarthritis. Arthritis Rheum. 1978;21(8):889–95. https://doi.org/10.1002/art.1780210804.
56. Duquesnoy RJ, Kozin F, Rodey GE. High prevalence of HLA-B27, Cw1 and Cw2 in patients with seronegative spondyloarthritis. Tissue Antigens. 1978;12(1):58–62.
57. Säfwenberg J, Domeij-Nyberg B, Kjällman M. HLA antigens in females with ankylosing spondylitis and other forms of seronegative rheumatic diseases. Scand J Rheumatol. 1978;7(3):177–82. https://doi.org/10.3109/03009747809095650.
58. Arnett FC, Hochberg MC, Bias WB. HLA-C locus antigens in HLA-B27 associated arthritis. Arthritis Rheum. 1978;21(8):885–8. https://doi.org/10.1002/art.1780210803.
59. Gerber LH, Murray CL, Perlman SG, Barth WF, Decker JL, Nigra TA, et al. Human lymphocyte antigens characterizing psoriatic arthritis and its subtypes. J Rheumatol. 1982;9(5):703–7.
60. López-Larrea C, Torre Alonso JC, Rodriguez Perez A, Coto E. HLA antigens in psoriatic arthritis subtypes of a Spanish population. Ann Rheum Dis. 1990;49(5):318–9. https://doi.org/10.1136/ard.49.5.318.
61. Sin CZ, Wang TS, Chiu HY, Tsai TF. Human leukocyte antigen and demographic characteristics in Chinese patients with active peripheral type psoriatic arthritis who had inadequate response to conventional disease-modifying antirheumatic drugs in a single dermatologic clinic. PLoS ONE. 2019;14(1):e0210076. https://doi.org/10.1371/journal.pone.0210076.
62. Shao L-N, Wang N, Zhou S-H, Wang Z. Associations between human leukocyte antigen C locus polymorphism and psoriatic arthritis in populations of European and Middle Eastern descent: a meta-analysis. Ann Saudi Med. 2020;40(4):338–46. https://doi.org/10.5144/0256-4947.2020.338.
63. Suo C, Xu H, Khor CC, Ong RT, Sim X, Chen J, et al. Natural positive selection and north-south genetic diversity in East Asia. Eur J Hum Genet. 2012;20(1):102–10. https://doi.org/10.1038/ejhg.2011.139.
64. Siegel RJ, Bridges SL Jr, Ahmed S. HLA-C: an accomplice in rheumatic diseases. ACR Open Rheumatol. 2019;1(9):571–9. https://doi.org/10.1002/acr2.11065.
65. Correale P, Mutti L, Pentimalli F, Baglio G, Saladino RE, Sileri P, et al. HLA-B*44 and C*01 prevalence correlates with Covid19 spreading across Italy. Int J Mol Sci. 2020;21(15):5205. https://doi.org/10.3390/ijms21155205.
66. Balamurugan A, Sharma SK, Mehra NK. Human leukocyte antigen class I supertypes influence susceptibility and severity of tuberculosis. J Infect Dis. 2004;189(5):805–11. https://doi.org/10.1086/381689.
67. Jakubowiak WM, Bogorodskaya EM, Borisov SE, Danilova ID, Kourbatova EV. Risk factors associated with default among new pulmonary TB patients and social support in six Russian regions. Int J Tuberc Lung Dis. 2007;11(1):46–53.
68. Liu Y, Zhao Z, Li T, Liao Q, Kushner N, Touzjian NY, et al. High resolution human leukocyte antigen class I allele frequencies and HIV-1 infection associations in Chinese Han and Uyghur cohorts. PLoS ONE. 2012;7(12):e50656. https://doi.org/10.1371/journal.pone.0050656.
69. Fadda L, Körner C, Kumar S, van Teijlingen NH, Piechocka-Trocha A, Carrington M, et al. HLA-Cw*0102-restricted HIV-1 p24 epitope variants can modulate the binding of the inhibitory KIR2DL2 receptor and primary NK cell function. PLoS Pathog. 2012;8(7):e1002805. https://doi.org/10.1371/journal.ppat.1002805.
70. de Sá NBR, Ribeiro-Alves M, da Silva TP, Pilotto JH, Rolla VC, Giacoia-Gripp CBW, et al. Clinical and genetic markers associated with tuberculosis, HIV-1 infection, and TB/HIV-immune reconstitution inflammatory syndrome outcomes. BMC Infect Dis. 2020;20(1):59. https://doi.org/10.1186/s12879-020-4786-5.
71. Pegram HJ, Andrews DM, Smyth MJ, Darcy PK, Kershaw MH. Activating and inhibitory receptors of natural killer cells. Immunol Cell Biol. 2011;89(2):216–24. https://doi.org/10.1038/icb.2010.78.
72. Ceccarelli M, Venanzi Rullo E, Vaccaro M, Facciolà A, d'Aleo F, Paolucci IA, et al. HIV-associated psoriasis: epidemiology, pathogenesis, and management. Dermatol Ther. 2019;32(2):e12806. https://doi.org/10.1111/dth.12806.
73. Morar N, Willis-Owen SA, Maurer T, Bunker CB. HIV-associated psoriasis: pathogenesis, clinical features, and management. Lancet Infect Dis. 2010;10(7):470–8. https://doi.org/10.1016/s1473-3099(10)70101-8.
74. Yen YF, Jen IA, Chen M, Lan YC, Lee CY, Chuang PH, et al. HIV infection increases the risk of incident psoriasis: a nationwide population-based cohort study in Taiwan. J Acquir Immune Defic Syndr. 2017;75(5):493–9. https://doi.org/10.1097/qai.0000000000001431.
75. Gambardella A, Licata G, Calabrese G, De Rosa A, Pagliuca F, Alfano R, et al. CMV infection: a clinical challenge in biological therapy? The case of asymptomatic patients with persistent positive immunoglobulin M anti-CMV treated with secukinumab. Psoriasis (Auckl). 2020;10:57–60. https://doi.org/10.2147/ptt.S284701.
76. Weitz M, Kiessling C, Friedrich M, Prösch S, Höflich C, Kern F, et al. Persistent CMV infection correlates with disease activity and dominates the phenotype of peripheral CD8+ T cells in psoriasis. Exp Dermatol. 2011;20(7):561–7. https://doi.org/10.1111/j.1600-0625.2011.01250.x.
77. Kondo E, Akatsuka Y, Kuzushima K, Tsujimura K, Asakura S, Tajima K, et al. Identification of novel CTL epitopes of CMV-pp65 presented by a variety of HLA alleles. Blood. 2004;103(2):630–8. https://doi.org/10.1182/blood-2003-03-0824.
78. Hyun SJ, Sohn HJ, Lee HJ, Lee SD, Kim S, Sohn DH, et al. Comprehensive analysis of cytomegalovirus pp65 antigen-specific CD8(+) T cell responses according to human leukocyte antigen class I allotypes and intraindividual dominance. Front Immunol. 2017;8:1591. https://doi.org/10.3389/fimmu.2017.01591.
79. Chiu H-Y, Chan C-C, Tsai T-F. The impact of long-term secukinumab treatment on Epstein-Barr virus and cytomegalovirus loads in patients with psoriasis. Int J Dermatol. 2016;55(11):e600–2. https://doi.org/10.1111/ijd.13346.
80. Horibe H, Ueyama C, Fujimaki T, Oguri M, Kato K, Ichihara S, et al. Association of a polymorphism of BTN2A1 with

dyslipidemia in community-dwelling individuals. Mol Med Rep. 2014;9(3):808–12. https://doi.org/10.3892/mmr.2014.1902.
81. Fu Y, Lee CH, Chi CC. Association of psoriasis with inflammatory bowel disease: a systematic review and meta-analysis. JAMA Dermatol. 2018;154(12):1417–23. https://doi.org/10.1001/jamadermatol.2018.3631.
82. Ellinghaus D, Ellinghaus E, Nair RP, Stuart PE, Esko T, Metspalu A, et al. Combined analysis of genome-wide association studies for Crohn disease and psoriasis identifies seven shared susceptibility loci. Am J Hum Genet. 2012;90(4):636–47. https://doi.org/10.1016/j.ajhg.2012.02.020.
83. Jung ES, Cheon JH, Lee JH, Park SJ, Jang HW, Chung SH, et al. HLA-C*01 is a risk factor for Crohn's disease. Inflamm Bowel Dis. 2016;22(4):796–806. https://doi.org/10.1097/mib.0000000000000693.
84. Ungprasert P, Wijarnpreecha K, Cheungpasitporn W. Patients with psoriasis have a higher risk of schizophrenia: a systematic review and meta-analysis of observational studies. J Postgrad Med. 2019;65(3):141–5. https://doi.org/10.4103/jpgm.JPGM_253_18.
85. Ungprasert P, Wijarnpreecha K, Cheungpasitporn W. Patients with schizophrenia have a higher risk of psoriasis: a systematic review and meta-analysis. Psychiatry Res. 2018;259:422–6. https://doi.org/10.1016/j.psychres.2017.11.021.
86. Strange A, Riley BP, Spencer CCA, Morris DW, Pirinen M, O'Dushlaine CT, et al. Genome-wide association study implicates HLA-C*01:02 as a risk factor at the major histocompatibility complex locus in schizophrenia. Biol Psychiatry. 2012;72(8):620–8. https://doi.org/10.1016/j.biopsych.2012.05.035.
87. Pouget JG, Han B, Wu Y, Mignot E, Ollila HM, Barker J, et al. Cross-disorder analysis of schizophrenia and 19 immune-mediated diseases identifies shared genetic risk. Hum Mol Genet. 2019;28(20):3498–513. https://doi.org/10.1093/hmg/ddz145.
88. Cardili RN, Deghaide NS, Mendes-Junior CT, Donadi EA, Souza CS. HLA-C and TNF gene polymorphisms are associated with psoriasis in Brazilian patients. Int J Dermatol. 2016;55(1):e16-22. https://doi.org/10.1111/ijd.12894.
89. Cibulova A, Zajacova M, Fojtikova M, Stolfa J, Sedova L, Cejkova P, et al. The HLA-Cw*06 allele and −1149 G/T polymorphism of extrapituitary promoter of PRL gene as a possible common genetic predisposing factors to psoriasis vulgaris and psoriatic arthritis in Czech population. Rheumatol Int. 2013;33(4):913–9. https://doi.org/10.1007/s00296-012-2472-7.
90. Gonzalez S, Martinez-Borra J, Torre-Alonso JC, Gonzalez-Roces S, del Río SJ, Rodriguez-Pérez A, et al. The MICA-A9 triplet repeat polymorphism in the transmembrane region confers additional susceptibility to the development of psoriatic arthritis and is independent of the association of Cw*0602 in psoriasis. Arthritis Rheum. 1999;42(5):1010–6. doi: https://doi.org/10.1002/1529-0131(199905)42:5<1010::Aid-anr21>3.0.Co;2-h.
91. Kim T-G, Han H, Lee HJ, Youn JI, Kim TY. The association of psoriasis with human leukocyte antigens in Korean population and the influence of age of onset and sex. J Invest Dermatol. 2000;114(2):309–13. https://doi.org/10.1046/j.1523-1747.2000.00863.x.
92. Nakagawa H, Asahina A, Akazaki S, Tokunaga K, Matsuki K, Ishibashi Y, et al. Association of Cw11 in Japanese patients with psoriasis vulgaris. Tissue Antigens. 1990;36(5):241–2. https://doi.org/10.1111/j.1399-0039.1990.tb01835.x.
93. Shawkatová I, Javor J, Párnická Z, Kozub P, Zilínková M, Frey P, et al. HLA-C, DRB1 and DQB1 alleles involved in genetic predisposition to psoriasis vulgaris in the Slovak population. Folia Microbiol (Praha). 2013;58(4):319–24. https://doi.org/10.1007/s12223-012-0213-7.
94. Szczerkowska Dobosz A, Rebała K, Szczerkowska Z, Nedoszytko B. HLA-C locus alleles distribution in patients from northern Poland with psoriatic arthritis: preliminary report. Int J Immunogenet. 2005;32(6):389–91. https://doi.org/10.1111/j.1744-313X.2005.00543.x.
95. Zhang XJ, Zhang AP, Yang S, Gao M, Wei SC, He PP, et al. Association of HLA class I alleles with psoriasis vulgaris in southeastern Chinese Hans. J Dermatol Sci. 2003;33(1):1–6. https://doi.org/10.1016/s0923-1811(03)00157-9.