Immunology
The Journal of cells, molecules, systems and technologies

British Society for immunology

IMMUNOLOGY REVIEW ARTICLE

Autoimmunity and autoimmune co-morbidities in psoriasis

Kazuhisa Furue,[1] Takamichi Ito,[1] Gaku Tsuji,[1] Takafumi Kadono,[2] Takeshi Nakahara[1,3] and Masutaka Furue[1,3]

[1]*Department of Dermatology, Kyushu University, Fukuoka, Japan,* [2]*Department of Dermatology, St Marianna University School of Medicine, Kawasaki, Japan and* [3]*Division of Skin Surface Sensing, Department of Dermatology, Kyushu University, Fukuoka, Japan*

doi:10.1111/imm.12891
Received 31 October 2017; revised 18 December 2017; accepted 29 December 2017.
Correspondence: Masutaka Furue, Department of Dermatology, Kyushu University, Maidashi 3-1-1, Higashiku, Fukuoka 812-8582, Japan.
Email: furue@dermatol.med.kyushu-u.ac.jp
Senior author: Masutaka Furue

Summary

Psoriasis is characterized by widespread scaly erythematous plaques that cause significant physical and psychological burdens for the affected individuals. Accelerated inflammation driven by the tumour necrosis factor-α/interleukin-23/interleukin-17 axis is now known to be the major mechanism in the development of psoriasis. In addition, psoriasis has an autoimmune nature that manifests as autoreactive T cells and is co-morbid with other autoimmune diseases, such as autoimmune bullous diseases, vitiligo, alopecia and thyroiditis. In this article, we review the recent topics on autoimmunity and autoimmune co-morbidities in psoriasis.

Keywords: alopecia; autoimmunity; bullous pemphigoid; psoriasis; thyroiditis; vitiligo.

Introduction

Psoriasis is a chronic, immune-mediated, inflammatory skin disorder that affects approximately 0·1–3% of the general population worldwide.[1–3] Given the visible nature of skin lesions, including those on the face, scalp, hands and nails,[4–7] psoriasis is a significant physical and psychological burden to afflicted patients, decreases patient quality of life, and induces feelings of internalized stigma.[8–12]

The disease burden of psoriasis is further increased by its association with psoriatic arthritis, which is characterized by seronegative spondyloarthropathies, enthesitis and elevated C-reactive protein levels.[1,13–16] Approximately 15–30% of Caucasians with psoriasis eventually develop psoriatic arthritis.[17] Although the prevalence is lower in Asian populations (Chinese 5·3–7·3%, Japanese 10·5% and Korean 11·2%), psoriatic arthritis significantly hampers patients' daily lives.[16,17] Additionally, psoriasis has significant co-morbidity with systemic inflammatory diseases, such as cardiovascular diseases and metabolic syndrome, and these conditions are currently receiving increasing attention.[18–27] In parallel, a plethora of inflammatory biomarkers are elevated in psoriasis.[28–32]

In addition to these co-morbid diseases that have a systemic inflammatory nature,[33] psoriasis presents with autoimmune aspects and co-exists with a variety of autoimmune disorders.[34–39] In this article, we summarize recent topics on autoimmunity and autoimmune co-morbidities in psoriasis.

Current pathogenesis of psoriasis and its treatments

Pathogenesis of psoriasis is multifactorial, including environmental,[40–42] genetic[43,44] and immune-related factors.[45–49] Recent genome-wide association studies have identified numerous risk-associated variants within 44 susceptibility loci for psoriasis, including HLAC*06:02, LCE3D, IL23R and CARD14.[1,43,44,50–52] These susceptibility genes are predominantly related to the innate and adaptive immune system as well as to skin barrier function.[1,43,44]

Cutaneous trauma, such as incisional injury, induces the expression of functional cathelicidin at the injured site.[53] Cathelicidin (LL37) is one of the anti-microbial peptides that are produced by keratinocytes and neutrophils.[53] An initial trigger of psoriasis is believed to involve activation of plasmacytoid dendritic cells (DCs) upon stimulation by complexes of host DNA and LL37.[45–48] Activated plasmacytoid DCs and damaged

keratinocytes produce interferon-α (IFN-α), IFN-β and tumour necrosis factor-α (TNF-α), which result in further production of TNF-α and interleukin-23 (IL-23) by plasmacytoid and recruited inflammatory DCs [TNF/inducible nitric oxide synthase-producing DCs (TIP-DCs)].[45–48] Interleukin-23 is critically involved in the generation and activation of IL-17- and IL-22-producing effector T helper type 17 (Th17) and Th22 cells.[48,49]

Interleukin-17 up-regulates the proliferation of keratinocytes and down-regulates their differentiation.[54] Interleukin-22 also drives epidermal hyperplasia, primarily by down-regulation of genes involved in terminal differentiation.[55,56] Interleukin-17 acts on keratinocytes to induce chemokines that lead to neutrophil, TIP-DC and Th17 cell influx into the skin;[57] and up-regulates production of the neutrophil-attractive chemokines CXCL1, CXCL2 and CXCL8 by keratinocytes.[58–60]

The accelerated TNF-α/IL-23/IL-17 axis coincides with the characteristic histopathology of psoriasis, such as epidermal hyperproliferation, aberrant differentiation and neutrophilic microabscess. Regarding treatments, topical application of steroids and vitamin D3 analogues inhibits psoriatic inflammation and normalizes epidermal differentiation.[61–65] Systemic treatments, such as cyclosporine, methotrexate and the phosphodiesterase 4 inhibitor apremilast, are useful for patients with extensive lesions.[66–68] Clinical trials have demonstrated the efficacy of the oral janus kinase inhibitor tofacitinib for psoriasis.[69,70] Biological agents targeting TNF-α, IL-23 and IL-17 signalling exhibit high efficacy for patients with severe lesions, arthritis or disturbed quality of life.[71–79] However, development of the anti-IL-22 antibody fezakinumab (ILV-094) has been discontinued due to treatment failure, probably because of the redundancy of IL-22 in humans.[48] The biologicals are applicable in both paediatric and elderly patients, in patients undergoing haemodialysis and patients with perioperational circumstances.[80–83] They are also used in pregnant women, although their safety warrants further evaluation.[84,85] Switching of biologicals is recommended for patients with primary failure, secondary failure and infusion reactions.[86]

Genetic backgrounds are also related to the treatment response to biologicals.[87,88] The high cost of biologicals limits access to these medications; however, cost-saving biosimilars are rapidly advancing to the market worldwide.[89–91] Biologicals are effective not only for psoriasis but also for co-morbid cardiovascular and metabolic diseases.[92,93] In addition, monitoring of infection is mandatory to avoid serious consequences.[94–96] The emergence of antidrug antibodies is another important issue that reduces the treatment response and promotes treatment failure during biological therapy.[97,98]

Autoimmunity in psoriasis

In addition to the TNF-α/IL-23/IL-17-shifted immune deviation, psoriasis is likely to be associated with an autoimmune background.[34,35] Several studies have

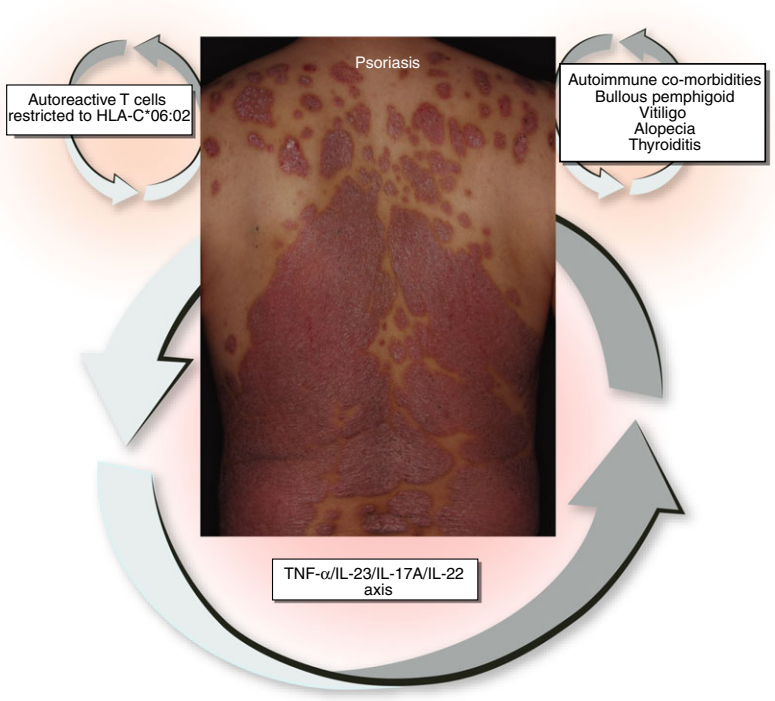

Figure 1. Based on clear-cut effectiveness of biologicals, the tumour necrosis factor-α (TNF-α)/ interleukin-23 (IL-23)/ IL-17A/ IL-22 axis plays a major role in the pathogenesis of psoriasis. In addition, psoriasis possesses an autoimmune nature manifesting autoreactive T cells that are frequently restricted to HLA-C*06:02. Psoriasis is also co-morbid with other autoimmune diseases, such as bullous pemphigoid, vitiligo, alopecia and thyroiditis.

revealed the presence of autoreactive T cells in psoriasis (Fig. 1).[99–101]

Exacerbation of chronic psoriasis can be associated with streptococcal throat infections.[99,100] Lesional skin and tonsils of patients with psoriasis harbour T cells co-reactive to streptococcal M protein and type I keratin.[99,100] Streptococcal M protein exhibits structural homology with type I keratins in epidermal keratinocytes.[100] These co-reactive T cells elicited by molecular mimicry are cutaneous lymphocyte antigen $(CLA)^+$, $CD8^+$ and IL-17 producers.[100] The co-reactive T cells may be pathogenic because tonsillectomy improves clinical symptoms in concert with a significant reduction of this T-cell population.[100] Type I keratin-specific T cells are also reported in psoriasis by another research group.[101]

The antimicrobial peptide LL37 is over-expressed in psoriatic epidermis.[46,101,102] LL37 not only triggers the TNF-α/IL-23/IL-17 axis by activating DCs but also works as an autoantigen to activate the T-cell adaptive immune system.[46,101,102] LL37 is recognized as an autoantigen by circulating T cells in 46% of psoriasis patients and more frequently (75%) in moderate-to-severe psoriasis.[101] In 54% of LL37-responder patients, LL37-specific T cells are exclusively $CD4^+$, 29% are both $CD4^+$ and $CD8^+$, and 17% are exclusively $CD8^+$. These LL37-specific T cells express CLA and produce variable amounts of IFN-γ, IL-17 and IL-22 but not IL-4. The frequency of LL37-specific T cells is significantly correlated with disease severity and is reduced by anti-TNF-α therapy.[101]

The pivotal role of intra-epidermal $CD8^+$ T cells has been documented in psoriasis.[103–106] These intra-epidermal $CD8^+$ T cells produce pathogenic IL-17A, and neutralization of $CD8^+$ T cells effectively prevents psoriasis development in xenotransplanted mice models *in vivo*.[105] Moreover, a recent study by Arakawa *et al.*[106] demonstrated that intraepidermal $CD8^+$ T cells recognize ADAMTS-like protein 5 (ADAMTSL5) on melanocytes in concert with HLA-C*06:02, which is the main psoriasis risk allele. Of note, LL37-specific $CD8^+$ T cells are also restricted to HLAC*06:02.[101]

In addition to the autoreactive T-cell response, several autoantibodies have been demonstrated in patients with psoriasis, including anti-stratum corneum antibody,[107] anti-squamous cell carcinoma antigen[108] and anti-heat-shock protein 65.[109] However, the clinical significance of the autoantibody production remains elusive.

HLA class I and class II polymorphisms are associated with immune-mediated diseases typically in a disease-specific manner.[110,111] Among susceptibility genes in psoriasis, HLAC*06:02 has a fundamental importance and defines familial clustering, early onset and a more severe course of psoriasis.[112,113] The fact that patients with psoriasis harbour autoreactive T cells that are restricted to HLAC*06:02 stress a potential role for these T cells in the development and perpetuation of psoriasis.

Intriguingly, the occurrence of psoriasis is commonly noted in human immunodeficiency virus (HIV) infection.[114–118] Psoriasis with HIV infection may have a more severe course with sudden exacerbations and may be refractory to treatment.[114–118] Paradoxical onset of psoriasis and arthritis is also demonstrated in patients treated with biologicals.[119,120] Anti-TNF-α antibodies frequently induce increased levels of KL-6, which is a serum marker for interstitial pneumonia.[121] In addition, the onset and exacerbation of psoriasis have been reported in melanoma patients treated with the anti-programmed cell death protein 1 antibody nivolumab.[122,123] These phenomena may be related to a disruption in cytokine balance following treatment with biologicals, resulting in the up-regulation of plasmacytoid DCs and the subsequent production of unopposed IFNs, leading to the bypassed IL-17 over-production.[124]

Autoimmune diseases co-morbid with psoriasis

Consistent with the autoimmune nature of psoriasis, numerous other autoimmune diseases, including autoimmune bullous diseases,[125] vitiligo,[126] alopecia[127] and thyroiditis,[128] are co-morbid with psoriasis. Among them, autoimmune bullous diseases, such as bullous pemphigoid and anti-laminin γ1 (p200) pemphigoid, are consistently documented.[37,125,129,130] In a nationwide population-based study of 3485 patients with bullous pemphigoid and 17425 matched controls, psoriasis is significantly associated with bullous pemphigoid [odds ratio (OR) 2·02; 95% CI 1·54–2·66].[130] In a case–control study of 287 patients with bullous pemphigoid and 1373 matched controls, the prevalence rate of psoriasis is greater in patients with bullous pemphigoid compared with control subjects (OR 4·4; 95% CI 2·2–8·9).[125] This association is also significant among both sexes.[125] In a case series of coexisting psoriasis and autoimmune bullous diseases, bullous pemphigoid is the most prevalent (63·4%) followed by anti-laminin γ1 pemphigoid (37·2%).[129]

Vitiligo is an autoimmune hypopigmentation disorder that is associated with psoriasis. Arunachalam *et al.*[126] reported that 27 of 463 (5·8%) patients with vitiligo have co-morbid psoriasis. A cross-sectional study of 1925 children and adolescents with psoriasis and 1 194 712 controls without psoriasis reveals a strong association of vitiligo with psoriasis (adjusted OR 4·76; 95% CI 1·71–13·20).[131] A significant association of vitiligo is evident in a nationwide study including 51 800 psoriasis patients with an OR of 5·94 (95% CI 3·79–9·31).[132] In parallel, it is interesting that vitiligo and psoriasis share a common genetic susceptible locus (rs9468925 in *HLA-C/HLA-B*).[133]

Psoriasis is also associated with autoimmune hair loss, namely alopecia areata (OR 4·71; 95% CI 2·98–7·45), in a national database in Taiwan.[132] In a cohort study

including 25 341 patients with psoriasis, a significant association of alopecia areata and psoriasis was also noted (OR 2·5; 95% CI 2·0–3·0).[127]

Hashimoto's thyroiditis and thyroid autoimmunity are frequently observed in the general population.[134] Free thyroxin levels are significantly increased in 105 psoriasis patients without arthritis compared with control 96 patients with tinea pedis.[135] In a hospital-based cross-sectional study of 856 615 patients, 9615 patients were diagnosed with psoriasis, and 1745 patients had Hashimoto's thyroiditis. A significant association exists for psoriasis and Hashimoto's thyroiditis even after adjusting for confounding variables, including gender, age, psoriatic arthropathy and the use of systemic anti-psoriatic agents (OR 2·49; 95% CI 1·79–3·48).[128] However, Vassilatou *et al.*[136] did not identify any tendency of association between psoriasis and autoimmune thyroiditis in their 114 patients with psoriasis.

Although the precise mechanisms underlying the autoimmune co-morbidities are not completely understood, damage to the basement membrane, melanocytes and hairs by chronic psoriatic inflammation may initiate and accelerate skin-related autoimmune diseases. An association of Crohn's disease and collagen diseases, such as systemic lupus erythematosus, rheumatoid arthritis, Sjögren's syndrome and systemic sclerosis, has been demonstrated in psoriasis patients.[127,137,138] Some patients with psoriasis develop collagen diseases upon treatment with biologicals.[139–141]

Similar to psoriasis, IFNs, DNA–LL-37 complex and plasmacytoid DCs play a pivotal role in triggering and perpetuating systemic lupus erythematosus.[142–144] Interferons accelerate the formation of DNA–LL37 complex that activates plasmacytoid DCs to produce high levels of IFNs and IL-23.[142,144] Interleukin-23 induces IL-17 production, which promotes autoimmunity.[144] Crohn's disease is also deeply linked to TNF-α/IL-23 signalling because both anti-TNF-α and anti-IL-23 biologicals are effective in this disease.[145,146] In vitiligo, IFN signature is also involved in the initiation of its immune response with the activation of plasmacytoid DCs.[147] Serum levels of IL-23 are also elevated in vitiligo.[148] These results supports a notion that common pathogenic pathways or shared autoantigens exist in psoriasis and other autoimmune diseases.

Conclusion

Psoriasis is a common chronic inflammatory skin disease that significantly decreases the physical and psychological quality of life of afflicted patients. The underlying inflammatory process results in increased co-morbidity with systemic inflammatory diseases, such as cardiovascular diseases and metabolic syndrome. In addition, numerous pathogenic traits are shared by psoriasis and other autoimmune diseases, such as autoimmune bullous diseases, vitiligo and alopecia areata. Future studies are warranted to explore the distinct pathways that underpin the inflammatory and autoimmune co-morbidity.

Disclosures

The authors declare that they have no conflict of interest.

References

1 Boehncke WH, Schön MP. Psoriasis. *Lancet* 2015; **386**:983–94.
2 Furue M, Kadono T. Psoriasis: behind the scenes. *J Dermatol* 2016; **43**:4–8.
3 Furue M, Kadono T. "Inflammatory skin march" in atopic dermatitis and psoriasis. *Inflamm Res* 2017; **66**:833–42.
4 Kwon HH, Kim MW, Park GH, Bae YI, Kuk SK, Suh DH *et al.* Facial Psoriasis Log-based Area and Severity Index: a valid and reliable severity measurement method detecting improvement of facial psoriasis in clinical practice settings. *J Dermatol* 2016; **43**:894–9.
5 Choi JW, Kim BR, Seo E, Youn SW. Identification of nail features associated with psoriasis severity. *J Dermatol* 2017; **44**:147–53.
6 Errichetti E, Zabotti A, Stinco G, Quartuccio L, Sacco S, De Marchi G *et al.* Dermoscopy of nail fold and elbow in the differential diagnosis of early psoriatic arthritis sine psoriasis and early rheumatoid arthritis. *J Dermatol* 2016; **43**:1217–20.
7 Errichetti E, Stinco G. Dermoscopy in differential diagnosis of palmar psoriasis and chronic hand eczema. *J Dermatol* 2016; **43**:423–5.
8 Atakan N, Yazici AC, Özarmağan G, İnalÖz HS, Gürer MA, Sabuncu İ *et al.* TURPSO: a cross-sectional, study investigating quality of life and treatment status of psoriasis patients in Turkey. *J Dermatol* 2016; **43**:298–304.
9 López-Estebaranz JL, Sánchez-Carazo JL, Sulleiro S. Effect of a family history of psoriasis and age on comorbidities and quality of life in patients with moderate to severe psoriasis: results from the ARIZONA study. *J Dermatol* 2016; **43**:395–401.
10 Alpsoy E, Polat M, FettahlıoGlu-Karaman B, Karadag AS, Kartal-Durmazlar P, YalCın B *et al.* Internalized stigma in psoriasis: a multicenter study. *J Dermatol* 2017; **44**:885–91.
11 Hawro M, Maurer M, Weller K, Maleszka R, Zalewska-Janowska A, Kaszuba A *et al.* Lesions on the back of hands and female gender predispose to stigmatization in patients with psoriasis. *J Am Acad Dermatol* 2017; **76**:648–54.
12 Masaki S, Tatsukawa R, Uryu M, Takahara M, Furue M, Ohata C *et al.* Treatment satisfaction, willingness to pay and quality of life in Japanese patients with psoriasis. *J Dermatol* 2017; **44**:143–6.
13 Asahina A, Umezawa Y, Yanaba K, Nakagawa H. Serum C-reactive protein levels in Japanese patients with psoriasis and psoriatic arthritis: long-term differential effects of biologics. *J Dermatol* 2016; **43**:779–84.
14 Takata T, Takahashi A, Taniguchi Y, Terada Y, Sano S. Detection of asymptomatic enthesitis in psoriasis patients: an onset of psoriatic arthritis? *J Dermatol* 2016; **43**:650–4.
15 Yamamoto T, Ohtsuki M, Sano S, Igarashi A, Morita A, Okuyama R *et al.* Epidemiological analysis of psoriatic arthritis patients in Japan. *J Dermatol* 2016; **43**:1193–6.
16 Yamamoto T, Ohtsuki M, Sano S, Igarashi A, Morita A, Okuyama R *et al.* Prevalence and current therapies of psoriatic arthritis in Japan: a survey by the Japanese Society of Psoriasis Research in 2016. *J Dermatol* 2017; **44**:e121.
17 Stuart PE, Nair RP, Tsoi LC, Tejasvi T, Das S, Kang HM *et al.* Genome-wide association analysis of psoriatic arthritis and cutaneous psoriasis reveals differences in their genetic architecture. *Am J Hum Genet* 2015; **97**:816–36.
18 Koch M, Baurecht H, Ried JS, Rodriguez E, Schlesinger S, Volks N *et al.* Psoriasis and cardiometabolic traits: modest association but distinct genetic architectures. *J Invest Dermatol* 2015; **135**:1283–93.
19 Parisi R, Rutter MK, Lunt M, Young HS, Symmons DPM, Griffiths CEM *et al.* Psoriasis and the risk of major cardiovascular events: cohort study using the Clinical Practice Research Datalink. *J Invest Dermatol* 2015; **135**:2189–97.
20 Lonnberg AS, Skov L, Skytthe A, Kyvik KO, Pedersen OB, Thomsen SF. Association of psoriasis with the risk for type 2 diabetes mellitus and obesity. *JAMA Dermatol* 2016; **152**:761–7.
21 Cohen AD, Dreiher J, Shapiro Y, Vidavsky L, Vardy DA, Davidovici B *et al.* Psoriasis and diabetes: a population-based cross-sectional study. *J Eur Acad Dermatol Venereol* 2008; **22**:585–9.
22 Naito R, Imafuku S. Distinguishing features of body mass index and psoriasis in men and women in Japan: a hospital-based case–control study. *J Dermatol* 2016; **43**:1406–11.

23 Liu JH, Chen Y, Zhen Z, Yeung CK, Chan J, Chan HH *et al.* Relation between endothelial progenitor cells and arterial stiffness in patients with psoriasis. *J Dermatol* 2016; **43**:888–93.

24 Honma M, Shibuya T, Iinuma S, Kishibe M, Takahashi H, Ishida-Yamamoto A. Close correlation of bone mineral density and body mass index in Japanese psoriasis patients. *J Dermatol* 2017; **44**:e1–2.

25 Chularojanamontri L, Wongpraparut C, Silpa-Archa N, Chaweekulrat P. Metabolic syndrome and psoriasis severity in South-East Asian patients: an investigation of potential association using current and chronological assessments. *J Dermatol* 2016; **43**:1424–8.

26 Furue M, Tsuji G, Chiba T, Kadono T. Cardiovascular and metabolic diseases comorbid with psoriasis: beyond the skin. *Intern Med* 2017; **56**:1613–9.

27 Oh EH, Ro YS, Kim JE. Epidemiology and cardiovascular comorbidities in patients with psoriasis: a Korean nationwide population-based cohort study. *J Dermatol* 2017; **44**:621–9.

28 Røpke M, Bulai Livideanu C, Kaldate R, Snel A, Paul C. Changes in IL-17A, macrophage-derived chemokine and adiponectin following treatment of psoriasis with calcipotriol plus betamethasone dipropionate aerosol foam: results from the PSO-ABLE study. *Br J Dermatol* 2017; https://doi.org/10.1111/bjd.15814.

29 Kim DS, Shin D, Lee MS, Kim HJ, Kim DY, Kim SM *et al.* Assessments of neutrophil to lymphocyte ratio and platelet to lymphocyte ratio in Korean patients with psoriasis vulgaris and psoriatic arthritis. *J Dermatol* 2016; **43**:305–10.

30 Pina T, Genre F, Lopez-Mejias R, Armesto S, Ubilla B, Mijares V *et al.* Asymmetric dimethylarginine but not osteoprotegerin correlates with disease severity in patients with moderate-to-severe psoriasis undergoing anti-tumor necrosis factor-α therapy. *J Dermatol* 2016; **43**:389–94.

31 Sato Y, Kajihara I, Yamada-Kanazawa S, Jinnin M, Ihn H. S100A7 expression levels in coordination with interleukin-8 indicate the clinical response to infliximab for psoriasis patients. *J Dermatol* 2017; **44**:838–9.

32 Takahashi T, Asano Y, Shibata S, Tada Y, Sato S. Serum angiopoietin-2 level as a potential biomarker in psoriasis vulgaris. *J Dermatol* 2017; **44**:205–6.

33 Davidovici BB, Sattar N, Prinz J, Puig L, Emery P, Barker JN *et al.* Psoriasis and systemic inflammatory diseases: potential mechanistic links between skin disease and comorbid conditions. *J Invest Dermatol* 2010; **130**:1785–96.

34 Sticherling M. Psoriasis and autoimmunity. *Autoimmun Rev* 2016; **15**:1167–70.

35 Prinz JC. Autoimmune aspects of psoriasis: heritability and autoantigens. *Autoimmun Rev* 2017; **16**:970–9.

36 Akiyama M, Ueno T, Kanzaki A, Kuwana M, Nagao M, Saeki H. Association of psoriasis with Hashimoto's thyroiditis, Sjögren's syndrome and dermatomyositis. *J Dermatol* 2016; **43**:711–2.

37 Maki N, Demitsu T, Umemoto N, Nagashima K, Nakamura T, Kakurai M *et al.* Possible paraneoplastic syndrome case of bullous pemphigoid with immunoglobulin G anti-BP180 C-terminal domain antibodies associated with psoriasis and primary macroglobulinemia. *J Dermatol* 2016; **43**:571–4.

38 Matsuo H, Asahina A, Fukuda T, Umezawa Y, Nakagawa H. Relapsing polychondritis associated with psoriasis vulgaris successfully treated with adalimumab: a case report with published work review. *J Dermatol* 2017; **44**:826–9.

39 Gál B, Dulic S, Kiss M, Groma G, Kovács L, Kemény L *et al.* Increased circulating anti-α6-integrin autoantibodies in psoriasis and psoriatic arthritis but not in rheumatoid arthritis. *J Dermatol* 2017; **44**:370–4.

40 Wolters M. Diet and psoriasis: experimental data and clinical evidence. *Br J Dermatol* 2005; **153**:706–14.

41 Mattozzi C, Paolino G, Richetta AG, Calvieri S. Psoriasis, vitamin D and the importance of the cutaneous barrier's integrity: an update. *J Dermatol* 2016; **43**:507–14.

42 Zeng J, Luo S, Huang Y, Lu Q. Critical role of environmental factors in the pathogenesis of psoriasis. *J Dermatol* 2017; **44**:863–72.

43 Tang H, Jin X, Li Y, Jiang H, Tang X, Yang X *et al.* A large-scale screen for coding variants predisposing to psoriasis. *Nat Genet* 2014; **46**:45–50.

44 Baurecht H, Hotze M, Brand S, Büning C, Cormican P, Corvin A *et al.* Genome-wide comparative analysis of atopic dermatitis and psoriasis gives insight into opposing genetic mechanisms. *Am J Hum Genet* 2015; **96**:104–20.

45 Nestle FO, Conrad C, Tun-Kyi A, Homey B, Gombert M, Boyman O *et al.* Plasmacytoid predendritic cells initiate psoriasis through interferon-α production. *J Exp Med* 2005 Jul 4; **202**:135–43.

46 Lande R, Gregorio J, Facchinetti V, Chatterjee B, Wang YH, Homey B *et al.* Plasmacytoid dendritic cells sense self-DNA coupled with antimicrobial peptide. *Nature* 2007; **449**:564–9.

47 Lowes MA, Suárez-Fariñas M, Krueger JG. Immunology of psoriasis. *Annu Rev Immunol* 2014; **32**:227–55.

48 Teng MW, Bowman EP, McElwee JJ, Smyth MJ, Casanova JL, Cooper AM *et al.* IL-12 and IL-23 cytokines: from discovery to targeted therapies for immune-mediated inflammatory diseases. *Nat Med* 2015; **21**:719–29.

49 Guttman-Yassky E, Krueger JG, Lebwohl MG. Systemic immune mechanisms in atopic dermatitis and psoriasis with implications for treatment. *Exp Dermatol* 2017; https://doi.org/10.1111/exd.13336.

50 Inoue N, Dainichi T, Fujisawa A, Nakano H, Sawamura D, Kabashima K. CARD14 Glu138 mutation in familial pityriasis rubra pilaris does not warrant differentiation from familial psoriasis. *J Dermatol* 2016; **43**:187–9.

51 Feng C, Wang T, Li SJ, Fan YM, Shi G, Zhu KJ. CARD14 gene polymorphism c.C2458T (p.Arg820Trp) is associated with clinical features of psoriasis vulgaris in a Chinese cohort. *J Dermatol* 2016; **43**:294–7.

52 Takeo N, Fujiwara S, Sakai T, Saito-Shono T, Ishikawa K, Hatano Y. Hereditary lactate dehydrogenase M-subunit deficiency with late-developing pustular psoriasis-like lesions. *J Dermatol* 2016; **43**:1429–32.

53 Dorschner RA, Pestonjamasp VK, Tamakuwala S, Ohtake T, Rudisill J, Nizet V *et al.* Cutaneous injury induces the release of cathelicidin anti-microbial peptides active against group A Streptococcus. *J Invest Dermatol* 2001; **117**:91–7.

54 Ha HL, Wang H, Pisitkun P, Kim JC, Tassi I, Tang W *et al.* IL-17 drives psoriatic inflammation via distinct, target cell-specific mechanisms. *Proc Natl Acad Sci USA* 2014; **111**:E3422–31.

55 Boniface K, Bernard FX, Garcia M, Gurney AL, Lecron JC, Morel F. IL-22 inhibits epidermal differentiation and induces proinflammatory gene expression and migration of human keratinocytes. *J Immunol* 2005; **174**:3695–702.

56 Sa SM, Valdez PA, Wu J, Jung K, Zhong F, Hall L *et al.* The effects of IL-20 subfamily cytokines on reconstituted human epidermis suggest potential roles in cutaneous innate defense and pathogenic adaptive immunity in psoriasis. *J Immunol* 2007; **178**:2229–40.

57 Nograles KE, Zaba LC, Guttman-Yassky E, Fuentes-Duculan J, Suárez-Fariñas M, Cardinale I *et al.* Th17 cytokines interleukin (IL)-17 and IL-22 modulate distinct inflammatory and keratinocyte-response pathways. *Br J Dermatol* 2008; **159**:1092–102.

58 Johnston A, Xing X, Wolterink L, Barnes DH, Yin Z, Reingold L *et al.* IL-1 and IL-36 are dominant cytokines in generalized pustular psoriasis. *J Allergy Clin Immunol* 2017; **140**:109–20.

59 Reich K, Papp KA, Matheson RT, Tu JH, Bissonnette R, Bourcier M *et al.* Evidence that a neutrophil-keratinocyte crosstalk is an early target of IL-17A inhibition in psoriasis. *Exp Dermatol* 2015; **24**:529–35.

60 Furue K, Yamamura K, Tsuji G, Mitoma C, Uchi H, Nakahara T *et al.* Highlighting interleukin-36 signalling in plaque psoriasis and pustular psoriasis. *Acta Derm Venereol* 2018; **98**:5–13.

61 Ito K, Koga M, Shibayama Y, Tatematsu S, Nakayama J, Imafuku S. Proactive treatment with calcipotriol reduces recurrence of plaque psoriasis. *J Dermatol* 2016; **43**:402–5.

62 Karakawa M, Komine M, Kishimoto M, Maki N, Matsumoto A, Sugai J *et al.* Effects of maxacalcitol ointment on skin lesions in patients with psoriasis receiving treatment with adalimumab. *J Dermatol* 2016; **43**:1354–7.

63 Usui K, Okubo Y, Hirano T, Tsuboi R. Vitamin D3 derivatives, alone or in combination with glucocorticoids, suppress streptococcal pyrogenic enterotoxin A-stimulated proliferation of peripheral blood mononuclear cells in patients with psoriasis. *J Dermatol* 2017; **44**:567–72.

64 Takahashi H, Ishida-Yamamoto A, Iizuka H. Linear psoriasis treated with topical glucocorticoid and active vitamin D3 ointments. *J Dermatol* 2016; **43**:1438–9.

65 Ergun T, Seckin Gencosmanoglu D, Alpsoy E, Bulbul-Baskan E, Saricam MH, Salman A *et al.* Efficacy, safety and drug survival of conventional agents in pediatric psoriasis: a multicenter, cohort study. *J Dermatol* 2017; **44**:630–4.

66 Nast A, Gisondi P, Ormerod AD, Saiag P, Smith C, Spuls PI *et al.* European S3-Guidelines on the systemic treatment of psoriasis vulgaris–Update 2015–Short version–EDF in cooperation with EADV and IPC. *J Eur Acad Dermatol Venereol* 2015; **29**:2277–94.

67 Bae SH, Yun SJ, Lee JB, Kim SJ, Won YH, Lee SC. Algorithm to select optimal systemic anti-psoriatic drugs in relation with patients' Psoriasis Area and Severity Index score for plaque psoriasis. *J Dermatol* 2016; **43**:643–9.

68 Ohtsuki M, Okubo Y, Komine M, Imafuku S, Day RM, Chen P *et al.* Apremilast, an oral phosphodiesterase 4 inhibitor, in the treatment of Japanese patients with moderate to severe plaque psoriasis: efficacy, safety and tolerability results from a phase 2b randomized controlled trial. *J Dermatol* 2017; **44**:873–84.

69 Abe M, Nishigori C, Torii H, Ihn H, Ito K, Nagaoka M *et al.* Tofacitinib for the treatment of moderate to severe chronic plaque psoriasis in Japanese patients: subgroup analyses from a randomized, placebo-controlled phase 3 trial. *J Dermatol* 2017; **44**:1228–37.

70 Asahina A, Etoh T, Igarashi A, Imafuku S, Saeki H, Shibasaki Y, *et al.* Oral tofacitinib efficacy, safety and tolerability in Japanese patients with moderate to severe plaque psoriasis and psoriatic arthritis: a randomized, double-blind, phase 3 study. *J Dermatol* 2016; **43**:869–80.

71 Menter A, Korman NJ, Elmets CA, Feldman SR, Gelfand JM, Gordon KB *et al.* Guidelines of care for the management of psoriasis and psoriatic arthritis: section 6.

Guidelines of care for the treatment of psoriasis and psoriatic arthritis: case-based presentations and evidence-based conclusions. *J Am Acad Dermatol* 2011; **65**:137–74.
72 Jabbar-Lopez ZK, Yiu ZZN, Ward V, Exton LS, Mohd Mustapa MF, Samarasekera E *et al.* Quantitative evaluation of biologic therapy options for psoriasis: a systematic review and network meta-analysis. *J Invest Dermatol* 2017; **137**:1646–54.
73 Torii H, Nakano M, Yano T, Kondo K, Nakagawa H. Efficacy and safety of dose escalation of infliximab therapy in Japanese patients with psoriasis: results of the SPREAD study. *J Dermatol* 2017; **44**:552–9.
74 Torii H, Terui T, Matsukawa M, Takesaki K, Ohtsuki M, Nakagawa H. Safety profiles and efficacy of infliximab therapy in Japanese patients with plaque psoriasis with or without psoriatic arthritis, pustular psoriasis or psoriatic erythroderma: results from the prospective post-marketing surveillance. *J Dermatol* 2016; **43**:767–78.
75 Imafuku S, Honma M, Okubo Y, Komine M, Ohtsuki M, Morita A *et al.* Efficacy and safety of secukinumab in patients with generalized pustular psoriasis: a 52-week analysis from phase III open-label multicenter Japanese study. *J Dermatol* 2016; **43**:1011–7.
76 Asahina A, Torii H, Ohtsuki M, Tokimoto T, Hase H, Tsuchiya T *et al.* Safety and efficacy of adalimumab treatment in Japanese patients with psoriasis: results of SALSA study. *J Dermatol* 2016; **43**:1257–66.
77 Saeki H, Nakagawa H, Nakajo K, Ishii T, Morisaki Y, Aoki T *et al.* Efficacy and safety of ixekizumab treatment for Japanese patients with moderate to severe plaque psoriasis, erythrodermic psoriasis and generalized pustular psoriasis: results from a 52-week, open-label, phase 3 study (UNCOVER-J). *J Dermatol* 2017; **44**:355–62.
78 Matsumoto A, Komine M, Karakawa M, Kishimoto M, Ohtsuki M. Adalimumab administration after infliximab therapy is a successful treatment strategy for generalized pustular psoriasis. *J Dermatol* 2017; **44**:202–4.
79 Hwang YJ, Youn SW, Kim BR, Yu DY, Kim Y, Pires A *et al.* Clinical factors predicting the therapeutic response to ustekinumab in patients with moderate to severe chronic plaque psoriasis. *J Dermatol* 2017; **44**:560–6.
80 Kusakari Y, Yamasaki K, Takahashi T, Tsuchiyama K, Shimada-Omori R, Nasu-Tamabuchi M *et al.* Successful adalimumab treatment of a psoriasis vulgaris patient with hemodialysis for renal failure: a case report and a review of the previous reports on biologic treatments for psoriasis patients with hemodialysis for renal failure. *J Dermatol* 2015; **42**:727–30.
81 Nimmannitya K, Tateishi C, Mizukami Y, Hamamoto K, Yamada S, Goto H *et al.* Successful treatment with ustekinumab of psoriasis vulgaris in a patient undergoing hemodialysis. *J Dermatol* 2016; **43**:92–4.
82 Kawakami H, Matsumoto Y, Abe N, Katori Y, Takahashi K, Tsuboi R *et al.* Perioperative management of tumor necrosis factor-α blocker-treated psoriatic patients: case reports and review. *J Dermatol* 2016; **43**:190–3.
83 Momose M, Asahina A, Hayashi M, Yanaba K, Umezawa Y, Nakagawa H. Biologic treatments for elderly patients with psoriasis. *J Dermatol* 2017; **44**:1020–3.
84 Rademaker M, Agnew K, Andrews M, Armour K, Baker C, Foley P *et al.* Psoriasis in those planning a family, pregnant or breast-feeding. The Australasian Psoriasis Collaboration. *Australas J Dermatol* 2017; https://doi.org/10.1111/ajd.12641.
85 Adachi A, Komine M, Hirano T, Tsuda H, Karakawa M, Murata S *et al.* Case of generalized pustular psoriasis exacerbated during pregnancy, successfully treated with infliximab. *J Dermatol* 2016; **43**:1439–40.
86 Honda H, Umezawa Y, Kikuchi S, Yanaba K, Fukuchi O, Ito T *et al.* Switching of biologics in psoriasis: reasons and results. *J Dermatol* 2017; **44**:1015–9.
87 Tejasvi T, Stuart PE, Chandran V, Voorhees JJ, Gladman DD, Rahman P *et al.* TNFAIP3 gene polymorphisms are associated with response to TNF blockade in psoriasis. *J Invest Dermatol* 2012; **132**:593–600.
88 Nishikawa R, Nagai H, Bito T, Ikeda T, Horikawa T, Adachi A *et al.* Genetic prediction of the effectiveness of biologics for psoriasis treatment. *J Dermatol* 2016; **43**:1273–7.
89 de la Cruz C, de Carvalho AV, Dorantes GL, Londoño Garcia AM, Gonzalez C, Maskin M *et al.* Biosimilars in psoriasis: clinical practice and regulatory perspectives in Latin America. *J Dermatol* 2017; **44**:3–12.
90 Cohen AD, Wu JJ, Puig L, Chimenti S, Vender R, Rajagopalan M *et al.* Biosimilars for psoriasis: worldwide overview of regulatory guidelines, uptake and implications for dermatology clinical practice. *Br J Dermatol* 2017; **117**:1495–502.
91 Takahashi H, Satoh K, Takagi A, Iizuka H. Economic burden of psoriatic patients in Japan: analysis from a single outpatient clinic. *J Dermatol* 2017; **44**:1024–6.
92 Gkalpakiotis S, Arenbergerova M, Gkalpakioti P, Potockova J, Arenberger P, Kraml P. Impact of adalimumab treatment on cardiovascular risk biomarkers in psoriasis: results of a pilot study. *J Dermatol* 2017; **44**:363–9.
93 Pina T, Corrales A, Lopez-Mejias R, Armesto S, Gonzalez-Lopez MA, Gómez-Acebo I *et al.* Anti-tumor necrosis factor-α therapy improves endothelial function and arterial stiffness in patients with moderate to severe psoriasis: a 6-month prospective study. *J Dermatol* 2016; **43**:1267–72.
94 Dávila-Seijo P, Dauden E, Descalzo MA, Carretero G, Carrascosa JM, Vanaclocha F *et al.* Infections in moderate to severe psoriasis patients treated with biological drugs compared to classic systemic drugs: findings from the BIOBADADERM Registry. *J Invest Dermatol* 2017; **137**:313–21.
95 Miyachi H, Nakamura Y, Wakabayashi S, Iwasawa MT, Oikawa A, Watanabe A *et al.* Case of recurrent severe cellulitis and cutaneous candidiasis during psoriasis treatment with ustekinumab. *J Dermatol* 2017; **44**:e206–7.
96 Stöllberger C, Finsterer J. Varicella zoster virus meningitis under ustekinumab because of plaque psoriasis. *J Dermatol* 2017; **44**:703–5.
97 Kui R, Gál B, Gaál M, Kiss M, Kemény L, Gyulai R. Presence of antidrug antibodies correlates inversely with the plasma tumor necrosis factor (TNF)-α level and the efficacy of TNF-inhibitor therapy in psoriasis. *J Dermatol* 2016; **43**:1018–23.
98 Gyldenløve M, Zachariae C, Jensen P, Griehsel H, Ståhle M, Skov L. Drug concentration and antidrug antibodies in patients with psoriasis treated with adalimumab or etanercept. *J Eur Acad Dermatol Venereol* 2017; **31**:e518–9.
99 Diluvio L, Vollmer S, Besgen P, Ellwart JW, Chimenti S, Prinz JC. Identical TCR β-chain rearrangements in streptococcal angina and skin lesions of patients with psoriasis vulgaris. *J Immunol* 2006; **176**:7104–11.
100 Thorleifsdottir RH, Sigurdardottir SL, Sigurgeirsson B, Olafsson JH, Sigurdsson MI, Petersen H *et al.* Improvement of psoriasis after tonsillectomy is associated with a decrease in the frequency of circulating T cells that recognize streptococcal determinants and homologous skin determinants. *J Immunol* 2012; **188**:5160–5.
101 Lande R, Botti E, Jandus C, Dojcinovic D, Fanelli G, Conrad C *et al.* The antimicrobial peptide LL37 is a T-cell autoantigen in psoriasis. *Nat Commun* 2014; **5**:5621.
102 Lande R, Chamilos G, Ganguly D, Demaria O, Frasca L, Durr S *et al.* Cationic antimicrobial peptides in psoriatic skin cooperate to break innate tolerance to self-DNA. *Eur J Immunol* 2015; **45**:203–13.
103 Chang JC, Smith LR, Froning KJ, Schwabe BJ, Laxer JA, Caralli LL *et al.* $CD8^+$ T cells in psoriatic lesions preferentially use T-cell receptor Vβ3 and/or Vβ13.1 genes. *Proc Natl Acad Sci USA* 1994; **91**:9282–6.
104 Conrad C, Boyman O, Tonel G, Tun-Kyi A, Laggner U, de Fougerolles A *et al.* α1β1 integrin is crucial for accumulation of epidermal T cells and the development of psoriasis. *Nat Med* 2007; **13**:836–42.
105 Di Meglio P, Villanova F, Navarini AA, Mylonas A, Tosi I, Nestle FO *et al.* Targeting $CD8^+$ T cells prevents psoriasis development. *J Allergy Clin Immunol* 2016; **138**:274–6.
106 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**:2203–12.
107 Jablonska S, Chorzelski TP, Beutner EH, Maciejowska EJarzabek-Chorzelska M, Rzesa G. Autoimmunity in psoriasis. Relation of disease activity and forms of psoriasis to immunofluorescence findings. *Arch Dermatol Res* 1978; **261**:135–46.
108 El-Rachkidy RG, Young HS, Griffiths CE, Camp RD. Humoral autoimmune responses to the squamous cell carcinoma antigen protein family in psoriasis. *J Invest Dermatol* 2008; **128**:2219–24.
109 Rambukkana A, Das PK, Witkamp L, Yong S, Meinardi MM, Bos JD. Antibodies to mycobacterial 65-kDa heat shock protein and other immunodominant antigens in patients with psoriasis. *J Invest Dermatol* 1993; **100**:87–92.
110 Stokkers PC, Reitsma PH, Tytgat GN, van Deventer SJ. HLA-DR and -DQ phenotypes in inflammatory bowel disease: a meta-analysis. *Gut* 1999; **45**:395–401.
111 Fernando MMA, Stevens CR, Walsh EC, De Jager PL, Goyette P, Plenge RM *et al.* Defining the role of the MHC in autoimmunity: a review and pooled analysis. *PLoS Genet* 2008; **4**:e1000024.
112 Gudjónsson JE, Kárason A, Antonsdóttir AA, Rúnarsdóttir EH, Gulcher JR, Stefánsson K *et al.* HLA-Cw6-positive and HLA-Cw6-negative patients with Psoriasis vulgaris have distinct clinical features. *J Invest Dermatol* 2002; **118**:362–5.
113 Gudjonsson JE, Karason A, Antonsdottir A, Runarsdottir EH, Hauksson VB, Upmanyu R *et al.* Psoriasis patients who are homozygous for the HLA-Cw*0602 allele have a 2.5-fold increased risk of developing psoriasis compared with Cw6 heterozygotes. *Br J Dermatol* 2003; **148**:233–5.
114 Duvic M, Johnson TM, Rapini RP, Freese T, Brewton G, Rios A. Acquired immunodeficiency syndrome-associated psoriasis and Reiter's syndrome. *Arch Dermatol* 1987; **123**:1622–32.
115 Obuch ML, Maurer TA, Becker B, Berger TG. Psoriasis and human immunodeficiency virus infection. *J Am Acad Dermatol* 1992; **27**:667–73.
116 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**:493–9.
117 Johnson TM, Duvic M, Rapini RP, Rios A. AIDS exacerbates psoriasis. *N Engl J Med* 1985; **313**:1415.
118 Itoi-Ochi S, Hayashi M, Yamaoka T, Kobayashi Y, Isei T, Shirasaka T *et al.* Occult HIV infection in Japanese rupioid psoriasis. *J Dermatol* 2017; **44**:e172–3.
119 Ruiz-Genao D, Perez-Zafrilla B, Lopez-Estebaranz JL, Belinchón-Romero I, Carrascosa JM, Ferrán M *et al.* Possible paradoxical occurrence of inflammatory arthritis in patients with psoriasis treated with biologics: findings in the Biobadaderm cohort. *Br J Dermatol* 2017; **176**:797–9.

120 Koizumi H, Tokuriki A, Oyama N, Ido H, Sugiura K, Akiyama M *et al.* Certolizumab pegol, a pegylated anti-TNF-α antagonist, caused *de novo*-onset palmoplantar pustulosis followed by generalized pustular psoriasis in a patient with rheumatoid arthritis. *J Dermatol* 2017; **44**:723–4.
121 Hayashi M, Yanaba K, Umezawa Y, Asahina A, Nakagawa H. Impact of anti-tumor necrosis factor-α agents on serum levels of KL-6 and surfactant protein-D in patients with psoriasis. *J Dermatol* 2017; **44**:1063–6.
122 Kato Y, Otsuka A, Miyachi Y, Kabashima K. Exacerbation of psoriasis vulgaris during nivolumab for oral mucosal melanoma. *J Eur Acad Dermatol Venereol* 2016; **30**:e89–91.
123 Murata S, Kaneko S, Harada Y, Aoi N, Morita E. Case of *de novo* psoriasis possibly triggered by nivolumab. *J Dermatol* 2017; **44**:99–100.
124 Collamer AN, Guerrero KT, Henning JS, Battafarano DF. Psoriatic skin lesions induced by tumor necrosis factor antagonist therapy: a literature review and potential mechanisms of action. *Arthritis Rheum* 2008; **59**:996–1001.
125 Kridin K, Bergman R. Association between bullous pemphigoid and psoriasis: a case–control study. *J Am Acad Dermatol* 2017; **77**:370–2.
126 Arunachalam M, Dragoni F, Colucci R, Berti S, Crocetti E, Galeone M *et al.* Non-segmental vitiligo and psoriasis comorbidity – a case–control study in Italian patients. *J Eur Acad Dermatol Venereol* 2014; **28**:433–7.
127 Wu JJ, Nguyen TU, Poon KY, Herrinton LJ. The association of psoriasis with autoimmune diseases. *J Am Acad Dermatol* 2012; **67**:924–30.
128 Kiguradze T, Bruins FM, Guido N, Bhattacharya T, Rademaker A, Florek AG *et al.* Evidence for the association of Hashimoto's thyroiditis with psoriasis: a cross-sectional retrospective study. *Int J Dermatol* 2017; **56**:553–6.
129 Ohata C, Ishii N, Koga H, Fukuda S, Tateishi C, Tsuruta D *et al.* Coexistence of autoimmune bullous diseases (AIBDs) and psoriasis: a series of 145 cases. *J Am Acad Dermatol* 2015; **73**:50–5.
130 Chen YJ, Wu CY, Lin MW, Chen TJ, Liao KK, Chen YC *et al.* Comorbidity profiles among patients with bullous pemphigoid: a nationwide population-based study. *Br J Dermatol* 2011; **165**:593–9.
131 Blegvad C, Egeberg A, Nielsen TE, Gislason GH, Zachariae C, Nybo Andersen AM *et al.* Autoimmune disease in children and adolescents with psoriasis: a cross-sectional study in Denmark. *Acta Derm Venereol* 2017; **97**:1225–29.
132 Tsai TF, Wang TS, Hung ST, Tsai PI, Schenkel B, Zhang M *et al.* Epidemiology and comorbidities of psoriasis patients in a national database in Taiwan. *J Dermatol Sci* 2011; **63**:40–6.
133 Zhu KJ, Lv YM, Yin XY, Wang ZX, Sun LD, He SM *et al.* Psoriasis regression analysis of MHC loci identifies shared genetic variants with vitiligo. *PLoS One* 2011; **6**: e23089.
134 Chistiakov DA. Immunogenetics of Hashimoto's thyroiditis. *J Autoimmune Dis* 2005; **2**:1.
135 Gul U, Gonul M, Kaya I, Aslan E. Autoimmune thyroid disorders in patients with psoriasis. *Eur J Dermatol* 2009; **19**:221–3.
136 Vassilatou E, Papadavid E, Papastamatakis P, Alexakos D, Koumaki D, Katsimbri P *et al.* No association of psoriasis with autoimmune thyroiditis. *J Eur Acad Dermatol Venereol* 2017; **31**:102–6.
137 Eppinga H, Poortinga S, Thio HB, Nijsten TEC, Nuij VJAA, van der Woude CJ *et al.* Prevalence and phenotype of concurrent psoriasis and inflammatory bowel disease. *Inflamm Bowel Dis* 2017; **23**:1783–9.
138 Tselios K, Yap KS, Pakchotanon R, Polachek A, Su J, Urowitz MB *et al.* Psoriasis in systemic lupus erythematosus: a single-center experience. *Clin Rheumatol* 2017; **36**:879–84.
139 Cemil BC, Atas H, Canpolat F, Akca Y, Sasmaz R. Infliximab-induced discoid lupus erythematosus. *Lupus* 2013; **22**:515–8.
140 Poulin Y, Thérien G. Drug-induced hepatitis and lupus during infliximab treatment for psoriasis: case report and literature review. *J Cutan Med Surg* 2010; **14**:100–4.
141 Kikuchi S, Umezawa Y, Hayashi M, Yanaba K, Fukuchi O, Ito T *et al.* Interstitial pneumonia in two patients with psoriasis during ustekinumab treatment. *J Dermatol* 2016; **43**:712–3.
142 Garcia-Romo GS, Caielli S, Vega B, Connolly J, Allantaz F, Xu Z *et al.* Netting neutrophils are major inducers of type I IFN production in pediatric systemic lupus erythematosus. *Sci Transl Med* 2011; **3**:73ra20.
143 Chasset F, Arnaud L. Targeting interferons and their pathways in systemic lupus erythematosus. *Autoimmun Rev* 2018; **17**:44–52.
144 Dai H, He F, Tsokos GC, Kyttaris VC. IL-23 limits the production of IL-2 and promotes autoimmunity in lupus. *J Immunol* 2017; **199**:903–10.
145 Hanauer SB, Feagan BG, Lichtenstein GR, Mayer LF, Schreiber S, Colombel JF *et al.* Maintenance infliximab for Crohn's disease: the ACCENT I randomised trial. *Lancet* 2002; **359**:1541–9.
146 Wils P, Bouhnik Y, Michetti P, Flourie B, Brixi H, Bourrier A *et al.* Subcutaneous ustekinumab provides clinical benefit for two-thirds of patients with Crohn's disease refractory to anti-tumor necrosis factor agents. *Clin Gastroenterol Hepatol* 2016; **14**:242–50.
147 Bertolotti A, Boniface K, Vergier B, Mossalayi D, Taieb A, Ezzedine K *et al.* Type I interferon signature in the initiation of the immune response in vitiligo. *Pigment Cell Melanoma Res* 2014; **27**:398–407.
148 Vaccaro M, Cannavò SP, Imbesi S, Cristani M, Barbuzza O, Tigano V *et al.* Increased serum levels of interleukin-23 circulating in patients with non-segmental generalized vitiligo. *Int J Dermatol* 2015; **54**:672–4.