

## Original Research

# Clinical Descriptive Study of Psoriasis in India: Triggers, Morbidities and Coincidences

Piyu P. Naik, MD\*

Department of Dermatology, Saudi-German Hospitals and Clinics, Dubai, UAE

\*Corresponding author

Piyu P. Naik, MD

Department of Dermatology, Saudi-German Hospitals and Clinics, Dubai, UAE; Tel. +971562173323; ORCID: 0000-0002-6499-4062; E-mail: [drpiyu85@gmail.com](mailto:drpiyu85@gmail.com)

### Article information

Received: April 25<sup>th</sup>, 2021; Revised: June 15<sup>th</sup>, 2021; Accepted: June 18<sup>th</sup>, 2021; Published: June 21<sup>st</sup>, 2021

### Cite this article

Naik PP. Clinical descriptive study of psoriasis in India: Triggers, morbidities and coincidences. *Dermatol Open J.* 2021; 6(1): 8-14. doi: [10.17140/DRMTOJ-6-144](https://doi.org/10.17140/DRMTOJ-6-144)

## ABSTRACT

### Background

Psoriasis is a T-cell mediated chronic inflammatory, a papulosquamous disease involving complex interactions between the innate and adaptive immune system and commonly manifested by skin lesions. It is characterized by hyperproliferation of keratinocytes and inflammatory infiltration in the epidermis and dermis. Chronic psoriasis can be a risk factor for developing comorbid diseases that share common immune pathophysiology and can be triggered by environmental factors in genetically susceptible individuals.

### Aim

To study the clinico-demographic profile, determine the most common triggering factors and determine comorbidities' coexistence in patients with psoriasis at a tertiary care centre.

### Study Design

A cross-sectional study.

### Methods

A teaching hospital-based cross-sectional study including 231 psoriasis patients visiting skin outpatient department (OPD) was conducted by the dermatology department at Sri Krishna hospital, Karamsad, India following acceptance of the study proposal by the human research ethics committee. This study was outcome of the dissertation topic of the author during dermatology residency. Total 5 qualified dermatologists working in the dermatology department and 3 resident doctors took part in the study as evaluators. After taking informed consent, detailed history regarding aggravating factors, progress and morbidities was taken with clinical examinations, and the diagnosis was purely clinical. Data were analysed using statistical package for the social sciences (SPSS).

### Result

Our study revealed a peak incidence of psoriasis in the fourth and fifth decade of life with male preponderance (1.9:1). The most commonly found psoriasis type was psoriasis vulgaris, and chronic plaque psoriasis and the most common site of involvement was extensors and trunk. Pruritus was the most disabling complaint (91.34%), and the disease course was progressive. Aggravating factors included stress, winter season, implant insertion, smoking, alcohol consumption, tobacco chewing and obesity. Koebner phenomenon was commonly found with implant insertion in psoriasis patients (76.2%). Family history was one of the well-established risk factors for developing psoriasis (14.2%). Our study's most commonly found nail changes were pitting (35.49%) and dystrophic changes (18.61%). Palmoplantar keratoderma (4.76%) and vitiligo (4.76%) were the most commonly found dermatological condition with psoriasis and have been associated with various comorbidities such as cardiovascular disorder, metabolic syndrome, psoriatic arthritis and psychiatric disorders. As it was a cross-sectional study, no controls were used.

### Conclusion

The study shows male preponderance and extensors, trunk as common sites of psoriatic lesion presentation. Aggravating factors included stress, winter season, implant insertion, smoking, alcohol consumption, tobacco chewing and obesity. Screening is encouraged for symptoms of psoriatic arthritis, cardiovascular diseases and metabolic syndromes in psoriasis patients due to its predilection with systemic comorbidities.

### Keywords

Psoriasis; Comorbidities; Cardiovascular disease; Metabolic syndrome; Risk factor; Triggers.

**INTRODUCTION**

Psoriasis is a non-contagious chronic inflammatory papulosquamous skin disease characterized by sharply demarcated erythematous plaques with a whitish scale.<sup>1,2</sup> Psoriasis can appear at any age, but two peaks of onset are the third and fifth decades of life.<sup>3-6</sup> It has affected 125 million people worldwide and significantly impacts the physical and emotional quality of life.<sup>7-9</sup> Psoriasis has a polygenic inheritance pattern with multiple alleles being encoded by a single gene resulting in multi-focal disease manifestations.<sup>10-14</sup>

Epidermal proliferation is the earliest pathogenic feature in psoriasis. There is an increase in deoxyribonucleic acid (DNA) synthesis and mitotic activity in the basal layer. The cells rapidly divide, move to the surface and shed as incomplete keratinized scales.<sup>15-17</sup> Migration of neutrophils into epidermis and collection in subcorneal spaces leads to micro-abscesses formation.<sup>18-20</sup> Cluster of differentiation 4 (CD4+) and CD8+ T-cells found in the epidermis and dermis play an essential role in developing cutaneous lesions by releasing T helper type 1 (Th1)/Th17 mediators like interferon-gamma (IFN-γ), interleukin 2 (IL2), IL17, IL23 and TNF-α, which acts on keratinocytes.<sup>21-23</sup>

Differences in the prevalence of psoriasis in various countries suggest genetic and environmental factors affecting the onset of this condition.<sup>24,25</sup> The primary susceptibility locus of psoriasis is psoriasis susceptibility gene 1 (PSORS 1) in the major histocompatibility complex on chromosome 6p21.<sup>26-28</sup> In genetically susceptible individuals, several exogenous factors such as skin aggression, infection, stress, alcohol and drugs and endogenous factors like allergies and hormonal changes can trigger the eruption.<sup>29-34</sup>

Classically associated comorbidities with psoriasis which are described in literature are psoriatic arthritis, Crohn's disease, uveitis and psychiatric disorders. Newly emerging comorbidities associated with psoriasis are metabolic syndrome, cardiovascular disease, erectile dysfunction, celiac disease, nonalcoholic fatty liver disease (NAFLD), osteoporosis and Parkinson's disease.<sup>35-38</sup> Association between psoriasis and diabetes was first reported by Strauss in 1897, followed by Reed et al in 1961, who observed an association between psoriasis and heart disease.<sup>39,40</sup>

**MATERIALS AND METHODS**

This was a teaching hospital-based cross-sectional study conducted for two-years. A total of 231 patients visiting dermatology outpatient department (OPD) in tertiary care centres of Charutar Arogya Mandal, India were recruited for this study. The study proposal was approved by the human research ethics committee, and informed written consent was taken from the study participants.

Detailed history regarding triggering factors, disease progression, and associated co-morbidities and socio-demographic information was recorded. Disease diagnosis was purely clinical. Data were analysed using statistical package for the social sciences (SPSS).

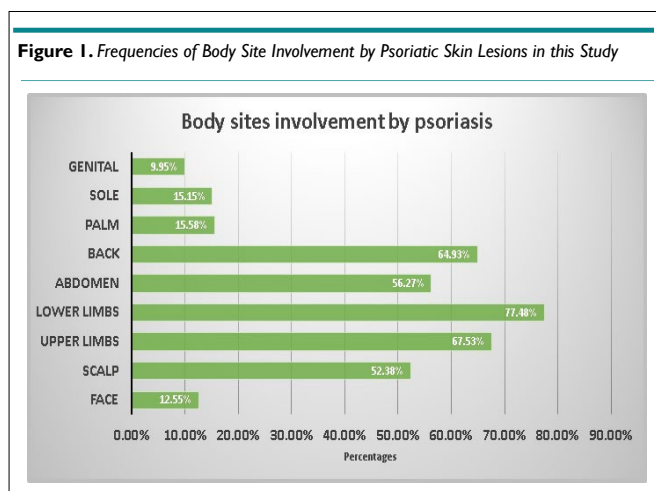
**RESULTS**

Based on the data analysis of 231 study participants, the majority of the affected patients were males (66.23%) with a peak incidence in the fourth (23.38%) and fifth (22.51%) decades of life (Table 1). Among the study subjects, 12.21% had psoriasis Vulgaris and 9.95% presented with chronic plaque psoriasis. Other types of psoriasis observed were guttate (1.73%), palmoplantar (1.73%), inverse (1.73%), psoriatic erythroderma (1.73%), scalp psoriasis (0.86%), elephantine psoriasis (1.29%), single plaque psoriasis (1.29%) and plantar psoriasis (0.43%).

**Table 1. Age Group and Gender Distribution of Study Subjects with Psoriasis**

Age Group (in Years)	Male	Female	Total
0-10	3 (1.96%)	5 (6.41%)	8 (3.46%)
11-20	10 (6.54%)	12 (15.38%)	22 (9.52%)
21-30	17 (11.11%)	19 (24.36%)	36 (15.58%)
31-40	41 (26.80%)	13 (16.67%)	54 (23.38%)
41-50	37 (24.18%)	15 (19.23%)	52 (22.51%)
51-60	28 (18.30%)	10 (12.82%)	38 (16.45%)
>61	17 (11.11%)	4 (5.13%)	21 (9.09%)
Total	153 (66.23%)	78 (33.77%)	231 (100%)

Collected data showed most common sites involved for psoriatic skin lesions were the lower limbs (77.48%) and upper limbs (67.53%), while genitals (9.95%) were least frequently involved. Frequencies of involvement of rest body sites were described in Figure 1.



88.74% reported the course of the disease as progressive. 48.05% affected individuals belonged to the middle class based on socioeconomic status and occupation were farmer (22.20%), labourer (22.87%), businessman (15.69%) and private sector (16.99%).

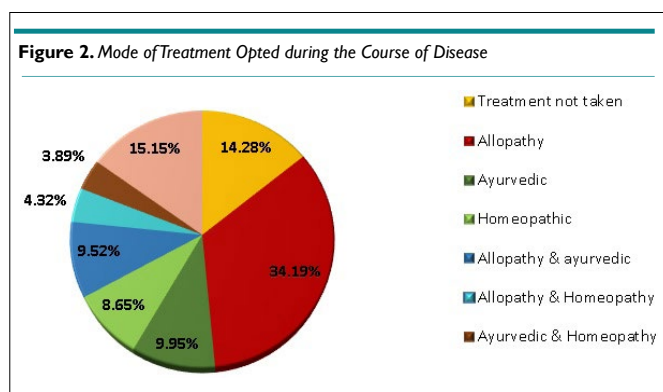
Disease triggering factors involved smoking (32.9%), alcohol (15.2%), tobacco (24.2%), itching (91.34%), winter season (70.12%) and stress (51.08%). Koebner phenomenon was ob-

served in patients with history of any implant insertion in 57.14% of subjects with significant correlation (Table 2).

**Table 2. History of Implant Insertion and its Correlation with Koebner Phenomenon**

Koebner phenomenon	History of implant insertion in body		Total
	No	Yes	
No	96 (44.04%)	3 (23.08%)	99 (42.86%)
Yes	122 (55.96%)	10 (76.92%)	132 (57.14%)
Total	218 (94.28%)	13 (5.62%)	231 (100%)

34.19% of the patients undertook allopathic treatment for psoriasis, while others underwent treatment using alternate medicine courses such as Ayurveda, homeopathy or combination (Figure 2).



Patient history revealed 28.44% were antihypertensive medications. Other drugs taken by subjects included non-steroidal anti-inflammatory drugs (NSAIDs) (18.34%), lipid-altering agent (13.76%), steroids (11.92%), anti-diabetics (8.25%), anti-psychotic (6.42%), anti-thyroid (5.5%) and drugs for chronic obstructive pulmonary disease (COPD) (7.3%).

85.28% of patients reported no family history of psoriasis, while 14.42% were genetically predisposed, and among them,

12.13% had first-degree relatives with psoriasis.

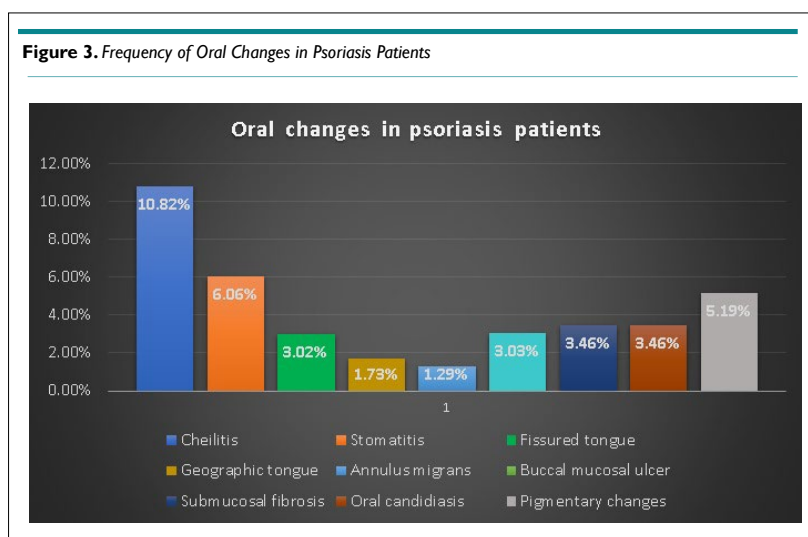
Types of joint affected in psoriatic patients were knee joint (20.34%), peripheral mono or asymmetrical oligoarticular (14.71%), distal interphalangeal (11.68%) and axial affection (2.16%).

Nail and oral changes in psoriasis patients are depicted in Table 3 and Figure 3.

**Table 3. Nail Changes in Psoriasis Patients**

Nail changes	Hand nail (Number %)	Foot nail (Number %)
Pitting	82 (35.49%)	41 (17.74%)
Onycholysis	13 (5.62%)	5 (2.16%)
Subungual hyperkeratosis	13 (5.62%)	18 (7.79%)
Salmon patch	3 (1.29%)	0
Onychomycosis	26 (11.25%)	15 (6.49%)
Transverse ridges	10 (4.32%)	9 (3.89%)
Vertical bands	22 (9.52%)	15 (6.49%)
Beaus line	7 (3.03%)	9 (3.89%)
Dystrophic changes	15 (6.49%)	43 (18.61%)
Leukonychia	2 (0.86%)	0
Pincer nail	1 (0.43%)	1 (0.43%)
Pterygium	2 (0.86%)	0
Twenty nail dystrophies	2 (0.86%)	2 (0.86%)
Koilonychia	4 (1.73%)	1 (0.43%)

The most common systemic comorbidity identified in these psoriasis patients is hypertension which was observed in 9.95% of subjects. Other notable comorbidities observed were COPD, hypercholesterolemia, diabetes mellitus (DM), thyroid disorder, depression, renal calculi, ischemic heart disease (IHD), pleural effusion and tuberculosis, glaucoma, vocal cord carcinoma, lower respiratory tract infection (LRTI), Vitamin A deficiency, anaemia, piles, hepatitis and multiple fractures with kyphoscoliosis (Table 4). The most common dermatological condition found in psoriasis patients is palmoplantar keratoderma (4.76%), followed by vitiligo (4.76%).

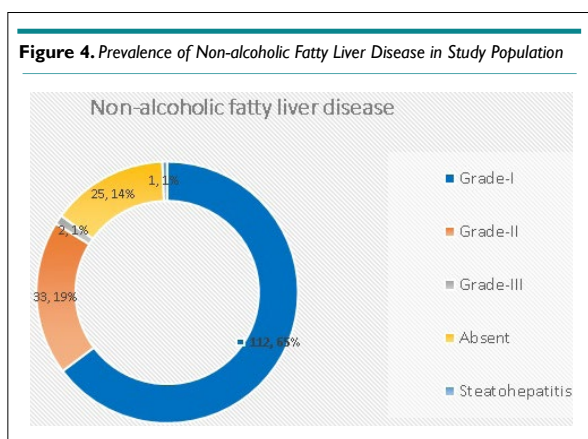


**Table 4.** The Systemic Conditions Identified with Psoriasis in the Study Population

Systemic Condition	Percentage
Hypertension	9.95%
Chronic obstructive pulmonary disease	3.03%
Hypercholesterolemia	3.03%
Diabetes mellitus	3.03%
Thyroid disorder	2.16%
Depression	1.29%
Renal calculi	1.29%
Ischemic heart diseases	0.86%
Pleural effusion and tuberculosis	0.43%
Glaucoma	0.43%
Vocal cord carcinoma	0.43%
Lower respiratory tract infection	0.43%
Vitamin A deficiency	0.43%
Anaemia	0.43%
Piles	0.43%
Hepatitis	0.43%
Multiple fractures with kyphoscoliosis	0.43%

Another dimension of this study was to see the prevalence of human leukocyte antigen (HLA)-B57 allele in these psoriasis patients. HLA-B57 was positive in 83 patients (35.93%) out of 231 total cases. In total, of all 113 cases of psoriasis with arthropathy, 69 patients (61.06 %) showed presence of HLA-B57.

Fetching the ultrasonography abdomen data also revealed interesting findings. One hundred ninety-six (196) patients were screened by ultrasound scanning of upper abdomen for presence of non-alcoholic fatty liver disease in all of these psoriasis cases after asking details of alcohol consumption. Rest 35 cases gave history of seldom alcohol intake, so they were excluded of this section of study. One hundred and seventy-three (173) cases out of 196 screened showed variable degrees of NAFLD (Figure 4).



## DISCUSSION

The prevalence of psoriasis varies across different countries, suggesting the influence of ethnicity, genetic background and environmental factors on the disease onset. As per the reports published across the world, prevalence varied from 0 to 11.8%. The prevalence of psoriasis in the USA and Canada were 4.6% and 4.7%, respectively.<sup>41</sup> Hospital-based studies done by Okhandiar et al<sup>41</sup> stated that psoriasis' incidence ranged between 0.44 and 2.2%. The report also speculated that extreme temperature, genetic background and dietary habits might be associated with the incidence of psoriasis. Our study showed male to female ratio as 1.9:1, male preponderance was noted in a study conducted by Okhandiar et al<sup>41</sup> and Bedi<sup>42</sup> who noted the male to female ratio as 2.46:1 and 2.5:1, respectively.

The peak incidence of disease onset is fourth and fifth decades of life in our study, and similar results were observed in studies conducted in the Faroe Islands and Denmark<sup>3,4</sup> and the studies done in North India by Okhandiar et al<sup>41</sup> and Bedi<sup>42</sup>. However, psoriasis with onset prior to the age of 40-years is linked with genetic susceptibility and recurrent as well as severe course.<sup>43</sup>

The role of genetic predisposition in psoriasis' etiopathogenesis has been observed in studies reported by Faber et al. 14.42% were genetically predisposed, and among them, 12.13% had first-degree relatives with psoriasis based on the reports collected in our study, and familial incidence was also seen in data published by Bedi and Kaur et al.<sup>42-44</sup>

Clinical classification of psoriasis can be broadly classified into non-pustular and pustular psoriasis with different clinical phenotypes of each, and the most common clinical type is classic chronic plaque-type psoriasis. Precise clinical recognition of subtype is essential to reflect upon disease activity and choice of treatment. In our study, most of the patients presented with chronic plaque-type psoriasis, and similarly, data analysed by Bedi<sup>42</sup> of 530 psoriasis patients revealed that 90% had chronic plaque phenotype. The most common sites of involvement were trunk and limbs in our study and the study conducted by Bedi<sup>42</sup>; however, Kaur et al<sup>45</sup> reported scalp as the most typical site of presentation followed by limbs.<sup>41,44</sup> While psoriasis causes cosmetic disability, it also leads to morbidity due to pruritus and burning sensation, as observed in our study in 91.34% subjects and 95% cases reported by Okhandiar et al.<sup>41</sup> The itching was significant in 81% of patients reported by Bedi<sup>42</sup> and 65% of patients in the study by Kaur et al.<sup>45</sup>

Nail involvement is common, initial, and at times the only site of involvement and the morphology depends on whether nail matrix, nail bed or hypochondrium is involved. Pitting was the most common nail change observed in our study. Similar results were observed by Kaur et al<sup>45</sup> in a study involving 167 patients, and the changes observed were onycholysis, discoloration and subungual hyperkeratosis.<sup>44</sup> Ghosal et al<sup>46</sup> observed nail involvement in 100 psoriatic patients with pitting and subungual hyperkeratosis as the most common presentation.

Jean Louis Alibert first recognized arthritis in association

with psoriasis in 1818; psoriatic arthritis was clearly defined in 1973 by Moll et al.<sup>47</sup> Polyarticular pattern simulating RA was observed by Rajendran et al.<sup>48</sup> and Ray et al.<sup>49</sup>

Palmoplantar keratoderma and vitiligo were the common dermatological conditions associated with psoriasis in our study. Similarly, Sandhu et al.<sup>50</sup> noted vitiligo in 38 out of 4700 psoriatic patients. Data from several studies in western literature revealed the association of psoriasis with systemic conditions such as metabolic syndrome. Our study also showed high prevalence of systemic disorders in the psoriasis patients (Table 4). The higher prevalence of metabolic syndrome in psoriatic patients than patients with other skin conditions was relatively higher in a study conducted by Gisoni et al.<sup>51</sup>

Many research studies in the past have been done to describe correlation between HLA-B57 and autoimmune diseases as well as psoriasis. Similar recent study done by Cassia et al.<sup>52</sup> showed 23.6% presence of HLA-B57 in psoriasis patients, while this study shows 35.93% prevalence of HLA-B57 in study population. Moreover, high (61.06%) positivity rate of HLA-B57 in psoriatic arthritis study group could be potentially studied in future research to monitor musculo-skeletal involvement of psoriasis.

Ultrasonography of upper abdomen is an excellent tool to evaluate solid organs including liver. Newer high resolution sonography probes can quantify fat level grading in liver easily. General diffuse increase in liver echogenicity with preserved periportal and diaphragmatic echogenicity is labelled as grade-I fatty changes. Similar findings with obscuration of periportal echogenicity is graded as II while rarefaction of periportal and diaphragmatic echogenicity is labelled as grade III. Associated coarsened echotexture of liver will lead to diagnosis of steatohepatitis which could lead to end-stage liver cirrhosis.<sup>53,54</sup> 88.26% positivity of NAFLD shows significant correlation between the diagnosis of psoriasis and NAFLD. Cheaper and easy nature of ultrasound could be handy in all psoriasis patients to identify “at-risk” cases as stages I to III always occur in serial progression and fatty changes without signs of coarse parenchyma are still reversible.

## CONCLUSION

This descriptive study shows male preponderance and extensors, trunk as a common sites of psoriatic lesion presentation. Aggravating factors included stress, winter season, implant insertion, smoking, alcohol consumption, tobacco chewing and obesity. Screening is encouraged for symptoms of psoriatic arthritis, cardiovascular diseases and metabolic syndromes in psoriasis patients due to its predilection with systemic comorbidities. HLA-B57 could be studied as potential disease severity identifier marker in psoriatic arthritic cases. High prevalence of NAFLD in psoriasis study population states the need of using ultrasonography in all psoriasis cases to pin down “at-risk” cases where cirrhosis could be imminent.

## DECLARATIONS

### Ethical Approval

The ethical approval was taken from ethical committee at Sri Krishna Hospital, Karamsad, India.

### Consent to Participate

The author have received written informed consent from the patient.

### Consent for Publication

Taken from all participants.

### Supporting Data

All data and references present.

### Funding Sources

This article was self-funded and no other source of funding present.

### Acknowledgments

The author thanks all resilient patients of psoriasis.

## REFERENCES

- Boehncke WH, Schon MP. Psoriasis. *Lancet*. 2015; 386: 983-994. doi: [10.1016/S0140-6736\(14\)61909-7](https://doi.org/10.1016/S0140-6736(14)61909-7)
- Nestle FO, Kaplan DH, Barker J. Psoriasis. *N Engl J Med*. 2009; 361: 496-509. doi: [10.1056/NEJMra0804595](https://doi.org/10.1056/NEJMra0804595)
- Lomholt G. Prevalence of skin diseases in a population: A census study from the Faroe Islands. *Dan Med Bull*. 1964; 11: 1-7.
- Brandrup F, Green A. The prevalence of psoriasis in Denmark. *Acta Derm Venereol*. 1981; 61(4): 344-346. doi: [10.1136/bmjopen-2018-028116](https://doi.org/10.1136/bmjopen-2018-028116)
- Christophers E. Psoriasis - epidemiology and clinical spectrum. *Clin Exp Dermatol*. 2001; 26(4): 314-320. doi: [10.1046/j.1365-2230.2001.00832.x](https://doi.org/10.1046/j.1365-2230.2001.00832.x)
- Gudjonsson JE, Elder JT. Psoriasis: Epidemiology. *Clin Dermatol*. 2007; 25(6): 535-546. doi: [10.1016/j.clindermatol.2007.08.007](https://doi.org/10.1016/j.clindermatol.2007.08.007)
- National Psoriasis Foundation®. Web site: [https://www.psoriasis.org/cure\\_known\\_statistics](https://www.psoriasis.org/cure_known_statistics). Accessed October 1, 2015.
- Kurd SK, Gelfand JM. The prevalence of previously diagnosed and undiagnosed psoriasis in US adults: Results from NHANES 2003-2004. *J Am Acad Dermatol*. 2009; 60: 218-224. doi: [10.1016/j.jaad.2008.09.022](https://doi.org/10.1016/j.jaad.2008.09.022)
- Rapp SR, Feldman SR, Exum ML, Fleischer AB, Reboussin DM. Psoriasis causes as much disability as other major medical diseases. *J Am Acad Dermatol*. 1999; 41(3 pt 1): 401-407. doi: [10.1016/s0190-9622\(99\)70112-x](https://doi.org/10.1016/s0190-9622(99)70112-x)

10. Watson W, Cann HM, Farber EM, Nall ML. The genetics of psoriasis. *Arch Dermatol.* 1972; 105(2): 197-207.
11. Elder JT, Nair RP, Guo SW, Henseler T, Christophers E, Voorhees JJ. The genetics of psoriasis. *Arch Dermatol.* 1994; 130(2): 216-224.
12. Henseler T. Genetics of psoriasis. *Arch Dermatol Res.* 1998; 290(9): 463-476.
13. Bhalerao J, Bowcock AM. The genetics of psoriasis: A complex disorder of the skin and immune system. *Hum Mol Genet.* 1998; 7(10): 1537-1545. doi: [10.1093/hmg/7.10.1537](https://doi.org/10.1093/hmg/7.10.1537)
14. Bowcock AM, Barker JN. Genetics of psoriasis: The potential impact on new therapies. *J Am Acad Dermatol.* 2003; 49(2 Suppl): S51-S56. doi: [10.1016/s0190-9622\(03\)01135-6](https://doi.org/10.1016/s0190-9622(03)01135-6)
15. Braun-Falco O, Christophers E. Structural aspects of initial psoriatic lesions. *Arch Dermatol Forsch.* 1974; 251(2): 95-110. doi: [10.1007/BF00560390](https://doi.org/10.1007/BF00560390)
16. Van de Kerkhof PC, Van Erp PE. The role of epidermal proliferation in the pathogenesis of psoriasis. *Skin Pharmacol.* 1996; 9(6): 343-354. doi: [10.1159/000211445](https://doi.org/10.1159/000211445)
17. Hatta N, Takata M, Kawara S, Hirose T, Takehara K. Tape stripping induces marked epidermal proliferation and altered TGF- $\alpha$  expression in non-lesional psoriatic skin. *J Dermatol Sci.* 1997; 14(2): 154-161. doi: [10.1016/s0923-1811\(96\)00567-1](https://doi.org/10.1016/s0923-1811(96)00567-1)
18. Burks JW, Montgomery H. Histopathologic study of psoriasis. *Arch Derm Syphilol.* 1943; 48(5): 479-493. doi: [10.1001/archderm.1943.01510050003001](https://doi.org/10.1001/archderm.1943.01510050003001)
19. Gordon M, Johnson WC. Histopathology and histochemistry of psoriasis. I. The active lesion and clinically normal skin. *Arch Dermatol.* 1967; 95(4): 402-407.
20. Murphy M, Kerr P, Grant-Kels JM. The histopathologic spectrum of psoriasis. *Clin Dermatol.* 2007; 25(6): 524-528. doi: [10.1016/j.clindermatol.2007.08.005](https://doi.org/10.1016/j.clindermatol.2007.08.005)
21. Austin LM, Ozawa M, Kikuchi T, Walters IB, Krueger JG. The majority of epidermal T cells in Psoriasis Vulgaris lesions can produce type 1 cytokines, interferon- $\gamma$ , interleukin-2, and tumor necrosis factor- $\alpha$ , defining TC1 (cytotoxic T lymphocyte) and TH1 effector populations: A type 1 differentiation bias is also measured in circulating blood T cells in psoriatic patients. *J Invest Dermatol.* 1999; 113(5): 752-759. doi: [10.1046/j.1523-1747.1999.00749.x](https://doi.org/10.1046/j.1523-1747.1999.00749.x)
22. Lima HC, Kimball AB. Targeting IL-23: Insights into the pathogenesis and the treatment of psoriasis. *Indian J Dermatol.* 2010; 55(2): 171-175. doi: [10.4103/0019-5154.62760](https://doi.org/10.4103/0019-5154.62760)
23. Krueger JG, Fretzin S, Suárez-Fariñas M, Haslett PA, Phipps KM, Cameron GS, et al. IL-17A is essential for cell activation and inflammatory gene circuits in subjects with psoriasis. *J Allergy Clin Immunol.* 2012; 130(1): 145-54.e9. doi: [10.1016/j.jaci.2012.04.024](https://doi.org/10.1016/j.jaci.2012.04.024)
24. Parisi R, Symmons DP, Griffiths, CE, Ashcroft DM, IMPACT project team. Global epidemiology of psoriasis: A systematic review of incidence and prevalence. *J Invest Dermatol.* 2013; 133: 377-385. doi: [10.1038/jid.2012.339](https://doi.org/10.1038/jid.2012.339)
25. Michalek IM, Loring B, John SM. A systematic review of worldwide epidemiology of psoriasis. *J Eur Acad Dermatol Venereol.* 2017; 31: 205-212. doi: [10.1111/jdv.13854](https://doi.org/10.1111/jdv.13854)
26. Trembath, R.C, Clough, R.L, Rosbotham, J.L, Jones, A.B, Camp, R.D, Frodsham, A, et al. Identification of a major susceptibility locus on chromosome 6p and evidence for further disease loci revealed by a two-stage genome-wide search in psoriasis. *Hum Mol Genet.* 1997; 6: 813-820. doi: [10.1093/hmg/6.5.813](https://doi.org/10.1093/hmg/6.5.813)
27. Burden AD, Javed S, Bailey M, Hodgins M, Connor M, Tillman D. Genetics of psoriasis: Paternal inheritance and a locus on chromosome 6p. *J Invest Dermatol.* 1998; 110: 958-960. doi: [10.1046/j.1523-1747.1998.00213.x](https://doi.org/10.1046/j.1523-1747.1998.00213.x)
28. Sagoo GS, Tazi-Ahnini R, Barker JW, Elder JT, Nair RP, Samuelsson L, Taupe, H, Trembath, R.C, Robinson, D.A, Iles, M.M. Meta-analysis of genome-wide studies of psoriasis susceptibility reveals linkage to chromosomes 6p21 and 4q28-q31 in Caucasian and Chinese Hans population. *J Invest Dermatol.* 2004; 122: 1401-1405. doi: [10.1111/j.0022-202X.2004.22607.x](https://doi.org/10.1111/j.0022-202X.2004.22607.x)
29. Camargo CM, Brotas AM, Ramos-e-Silva M, Carneiro S. Isomorphic phenomenon of Koebner: Facts and controversies. *Clin Dermatol.* 2013; 31(6): 741-749. doi: [10.1016/j.clindermatol.2013.05.012](https://doi.org/10.1016/j.clindermatol.2013.05.012)
30. Noah PW. The role of microorganisms in psoriasis. *Semin Dermatol.* 1990; 9(4): 269-276.
31. Poikolainen K, Reunala T, Karvonen J, Lauharanta, Kärkkäinen P. Alcohol intake: A risk factor for psoriasis in young and middle aged men? *BMJ.* 1990; 300(6727): 780-783. doi: [10.1136/bmj.300.6727.780](https://doi.org/10.1136/bmj.300.6727.780)
32. Jankovic S, Raznatovic M, Marinkovic J, Jankovic J, Maksimovic N. Risk factors for psoriasis: A case-control study. *J Dermatol.* 2009; 36(6): 328-334. doi: [10.1111/j.1346-8138.2009.00648.x](https://doi.org/10.1111/j.1346-8138.2009.00648.x)
33. Kanda N, Watanabe S. Regulatory roles of sex hormones in cutaneous biology and immunology. *J Dermatol Sci.* 2005; 38(1): 1-7. doi: [10.1016/j.jdermsci.2004.10.011](https://doi.org/10.1016/j.jdermsci.2004.10.011)
34. Lipozencić J, Milavec-Puretić V, Pasić A. Contact allergy and psoriasis. *Arh Hig Rada Toksikol.* 1992; 43(3): 249-254.
35. Birkenfeld S, Dreier J, Weitzman D, Cohen AD. Celiac disease

- associated with psoriasis. *Br J Dermatol.* 2009; 161: 1331-1334. doi: [10.1111/j.1365-2133.2009.09398.x](https://doi.org/10.1111/j.1365-2133.2009.09398.x)
36. Gisondi P, Targher G, Zoppini G, Girolomoni G. Non-alcoholic fatty liver disease in patients with chronic plaque psoriasis. *J Hepatol.* 2009; 51: 758-764. doi: [10.1016/j.jhep.2009.04.020](https://doi.org/10.1016/j.jhep.2009.04.020)
37. Miele L, Vallone S, Cefalo C, La Torre G, Di Stasi C, Vecchio FM, et al. Prevalence, characteristics and severity of non-alcoholic fatty liver disease in patients with chronic plaque psoriasis. *J Hepatol.* 2009; 51: 778-786. doi: [10.1016/j.jhep.2009.06.008](https://doi.org/10.1016/j.jhep.2009.06.008)
38. Goulding JM, Price CL, Defty CL, Hulangamuwa CS, Bader E, Ahmed I. Erectile dysfunction in patients with psoriasis: Increased prevalence, an unmet need, and a chance to intervene. *Br J Dermatol.* 2011; 164: 103-109. doi: [10.1111/j.1365-2133.2010.10077.x](https://doi.org/10.1111/j.1365-2133.2010.10077.x)
39. Strauss H. Zur Lehre von der neurogenen und der thyreogenen Glykosurie [German]. *Dtsch Med Wochenschr.* 1897; 20: 309-312. doi: [10.1055/s-0029-1204973](https://doi.org/10.1055/s-0029-1204973)
40. Reed WB, Becker SW, Rohde R, Heiskell CL. Psoriasis and arthritis. Clinicopathologic study. *Arch Dermatol.* 1961; 83: 541-548. doi: [10.1001/archderm.1961.01580100005001](https://doi.org/10.1001/archderm.1961.01580100005001)
41. Okhandiar RP, Banerjee BN. Psoriasis in the tropics: An epidemiological survey. *J Indian Med Assoc.* 1963; 41: 550-556.
42. Bedi TR. Psoriasis in north India. Geographical variations. *Dermatologica.* 1977; 155: 310-314. doi: [10.1159/000250983](https://doi.org/10.1159/000250983)
43. Smith AE, Kassab JY, Payne CMR, Beer WE. Bimodality in age of onset of psoriasis, in both patients and their relatives. *Dermatology.* 1993; 186: 181-186. doi: [10.1159/000247341](https://doi.org/10.1159/000247341)
44. Farber EM, Nall L. The Natural history of psoriasis in 5,600 patients. *Dermatologica.* 1974; 148: 1-18. doi: [10.1159/000251595](https://doi.org/10.1159/000251595)
45. Kaur I, Handa S, Kumar B. Natural history of psoriasis: A study from the Indian subcontinent. *J Dermatol.* 1997; 24: 230-234. doi: [10.1111/j.1346-8138.1997.tb02779.x](https://doi.org/10.1111/j.1346-8138.1997.tb02779.x)
46. Ghosal A, Gangopadhyay D, Chanda M, Das N. Study of nail changes in psoriasis. *Indian J Dermatol.* 2004; 49: 18-21.
47. Moll JM, Wright V. Psoriatic arthritis. *Semin Arthritis Rheum.* 1973; 3: 55-78. doi: [10.1016/0049-0172\(73\)90035-8](https://doi.org/10.1016/0049-0172(73)90035-8)
48. Rajendran CP, Ledge SG, Rani KP, Madhavan R. Psoriatic arthritis. *J Assoc Physicians India.* 2003; 51: 1065-1068.
49. Ray SPC, Singh T, Kaur I, Suri S, Sehgal S, Kaur S. Clinical profile of psoriatic arthropathy. *Indian J Dermatol Venereol Leprol.* 1990; 56: 200-2003.
50. Sandhu K, Kaur I, Kumar B. Psoriasis and vitiligo. *J Am Acad Dermatol.* 2004; 51: 149-150. doi: [10.1016/j.jaad.2003.12.014](https://doi.org/10.1016/j.jaad.2003.12.014)
51. Gisondi P, Tessari G, Conti A, Piaserico S, Schianchi S, Peserico A, et al. Prevalence of metabolic syndrome in patients with psoriasis: A hospital-based case-control study. *Br J Dermatol.* 2007; 157: 68-73. doi: [10.1111/j.1365-2133.2007.07986.x](https://doi.org/10.1111/j.1365-2133.2007.07986.x)
52. Cassia FF, Cardoso JF, Porto LC, Ramos-e-Silva M, Carneiro S. Association of HLA alleles and HLA haplotypes with psoriasis, psoriatic arthritis and disease severity in a miscegenated population. *Psoriasis (Auckl).* 2021; 11: 41-51. doi: [10.2147/PTT.S258050](https://doi.org/10.2147/PTT.S258050)
53. Singh D, Das CJ, Baruah MP. Imaging of non alcoholic fatty liver disease: A road less travelled. *Indian J Endocrinol Metab.* 2013; 17(6): 990-995. doi: [10.4103/2230-8210.122606](https://doi.org/10.4103/2230-8210.122606)
54. Gandha N, Wibawa LP, Jacob TNA, Sulaiman AS. Correlation between psoriasis severity and nonalcoholic fatty liver disease degree measured using controlled attenuation parameter. *Psoriasis (Auckl).* 2020; 10: 39-44. doi: [10.2147/PTT.S272286](https://doi.org/10.2147/PTT.S272286)