International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2024: 7.101

# Psoriasis as a Systemic Disease: Immune, Microbiota, and Mental Health Intersections

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**Abstract:** ***Psoriasis is a chronic, immune-mediated skin disease characterized by erythematous, scaly plaques and systemic inflammation. Affecting approximately 2-4% of the global population, it significantly impairs quality of life and is associated with numerous comorbid conditions, including psoriatic arthritis, cardiovascular disease, and metabolic syndrome. This paper explores the multifactorial etiology of psoriasis, including genetic predisposition, immune dysregulation, and environmental triggers such as stress and microbiota imbalance. Emphasis is placed on the systemic implications of the disease, its psychological impact, and evolving treatment strategies. Current therapies range from topical agents and phototherapy to targeted immunomodulators. The integration of microbiome-based research and psychosocial support into treatment plans reflects a growing understanding of psoriasis as more than a dermatological disorder. Effective management requires a personalized, multidisciplinary approach that addresses both the physical and emotional burdens of the disease.***

**Keywords:** psoriasis management, immune system disorder, genetic and environmental factors, microbiome research, psychosocial impact

## 1. Introduction

Psoriasis is a chronic inflammatory skin disorder characterized by sharply demarcated, erythematous plaques with silvery scaling [1]. It arises from a complex interplay of genetic predisposition, immune system dysregulation, and environmental influences [3]. The disease typically follows a bimodal distribution, with onset most frequently occurring between the ages of 20–30 and 50–60 years [1]. Family history is a strong risk factor; about 30% of patients report having a first-degree relative with psoriasis, and genetic studies have identified over 40 susceptibility loci involved in immune regulation and skin barrier integrity [1, 3, 10]. The burden of psoriasis extends well beyond cutaneous symptoms. Although skin plaques are its hallmark, psoriasis is now widely regarded as a systemic inflammatory condition [4, 6]. The underlying immune response involves abnormal activation of dendritic cells and effector T cells, particularly the Th1 and Th17 subsets, which produce pro-inflammatory cytokines such as IL-17, IL-23, and TNF-α [3, 6]. These cytokines not only sustain local inflammation but also contribute to systemic involvement, explaining the disease's association with a wide range of comorbidities including cardiovascular disease, psoriatic arthritis, metabolic syndrome, and inflammatory bowel disease [5, 6]. Emerging research also points to the role of the skin and gut microbiota in modulating psoriatic inflammation. Dysbiosis in these microbial communities has been linked to both the onset and exacerbation of the disease [9–11]. Altered gut microbiota composition, particularly reduced butyrate-producing bacteria, has been associated with increased systemic inflammation and changes in cytokine profiles [10, 11]. Environmental and lifestyle factors further influence disease activity. Common triggers include infections, trauma to the skin, obesity, smoking, and notably, psychological stress [8]. Stress has been shown to affect immune homeostasis via the hypothalamic–pituitary–adrenal axis, potentially exacerbating disease flares in susceptible individuals [8]. This highlights the importance of holistic approaches in disease management. Globally, psoriasis affects an estimated 2–4% of the population and has a substantial impact on quality of life [2, 7]. Its visible nature, chronicity, and associated symptoms often lead to psychosocial distress, stigmatization, and impaired daily functioning—factors that reinforce the need for multidisciplinary care strategies [7].

In summary, psoriasis is a multifactorial, immune-mediated disease that extends beyond dermatologic symptoms to affect systemic health and psychosocial well-being. A deeper understanding of its immunologic and environmental drivers, including the role of microbiota and stress, continues to shape evolving approaches to treatment and patient care [1–11].

This study aims to explore the multifactorial nature of psoriasis by examining its underlying causes, associated comorbidities, and the various treatment options currently available. The scope includes an in-depth analysis of the immunopathogenesis of psoriasis, the role of microbiota in disease modulation, and the psychological as well as systemic comorbid conditions often linked to psoriasis, such as cardiovascular disease, metabolic syndrome, and depression. In terms of treatment, the study will focus primarily on topical therapies, phototherapy, and emerging microbiota-targeted interventions, with special emphasis on clinically relevant and evidence-based approaches. Only treatments with support from peer-reviewed research and clinical studies will be included.

**Inclusions:**
- Causes and immunological basis of psoriasis
- Comorbid conditions associated with psoriasis
- Overview on Quality of life of individuals
- Treatment Approaches

**Exclusions:**
- Surgical or invasive treatments for psoriasis
- Cost analysis or pharmacoeconomic studies
- Pediatric-specific treatment protocols (unless part of general findings)
- Non-peer-reviewed or anecdotal treatment claims

This research follows a qualitative, narrative review methodology based on secondary data collected from peer-reviewed journal articles, clinical reviews, and authoritative web resources.

## 2. Causes of Psoriasis

Psoriasis is an immune-mediated inflammatory skin disease marked by scaly, red, and well-demarcated plaques. These arise due to a combination of keratinocyte hyperproliferation, abnormal differentiation, inflammatory cell infiltration, and neovascularisation [2]. Clinical and experimental evidence highlights the central role of the immune system in the pathogenesis of psoriasis, particularly through dysregulation involving both the innate and adaptive immune responses [1, 3]. In the early, or initiation phase, plasmacytoid dendritic cells (pDCs) become activated and trigger dermal dendritic cells (DCs), which subsequently stimulate skin-resident and newly recruited helper and cytotoxic T cells. These T cells, especially Th1, Th17, and IL-22-producing Th22 cells, infiltrate psoriatic plaques and sustain chronic inflammation by secreting cytokines [1]. Although psoriasis was long viewed as a Th1-driven disease, the discovery of Th17 cells' involvement has redefined this view, with IL-17 and IL-23 emerging as dominant cytokines orchestrating the aberrant immune activity in psoriasis [1, 3].

Keratinocytes- key mediators in this immune dysregulation- not only exhibit hyperproliferation, reducing the normal epidermal turnover from 21–28 days to just 3–4 days, but also initiate immune signalling cascades by recruiting inflammatory dendritic cells that produce IL-12 and IL-23. These cytokines further activate T cells, which release inflammatory mediators such as IL-17, IL-22, interferon-gamma (IFN-γ), and tumor necrosis factor-alpha (TNF-α), sustaining both epidermal damage and chronic inflammation [3]. In addition to classical antigen-driven mechanisms, T-cell activation in psoriasis may also proceed through antigen-independent pathways, further amplifying the immune response. A novel mechanism involves the T-cell surface molecule UM4D4, found predominantly on lesional skin T cells. This molecule enables activation of psoriatic T-cell clones even in the absence of antigen presentation, as shown by their proliferation upon exposure to anti-UM4D4 monoclonal antibodies. At the same time, antigen-presenting cells are increased in number and function in psoriatic lesions compared to uninvolved skin, contributing further to immune activation [6].

The lymphokines released by activated T cells directly influence keratinocyte behavior by inducing the expression of molecules such as intercellular adhesion molecule-1 (ICAM-1) and HLA-DR on their surfaces, while also enhancing their proliferation. This tightly interwoven interaction between immune cells and keratinocytes establishes a self-sustaining inflammatory cycle, driving the persistence and severity of the disease [6]. Genetic studies reinforce this model, identifying over 40 susceptibility loci associated with psoriasis, many of which encode proteins related to skin barrier integrity and immune regulation [3]. These findings collectively underscore the fundamental role of immune dysfunction—through both innate and adaptive mechanisms—in the initiation and maintenance of psoriatic disease [3, 6].

Up to 60% of the patients describe stress as being a key trigger of their disease. It is known that psychological stress has the potential to regulate the immune response, and there is emerging evidence that abnormal neuroendocrine responses to stress may contribute to the pathogenesis of chronic autoimmune diseases, as has been described for rheumatoid arthritis (RA). It is likely that, in some patients with psoriasis, there is an abnormal hypothalamic-adrenal axis response to acute stress. This area is poorly understood and thus deserves further investigation. [8]

### 2.1 Associated conditions and comorbid conditions

Recent studies have shown that psoriasis is not limited to the skin but is linked to a range of systemic health conditions. Around 75% of individuals with psoriasis experience at least one comorbid disorder, with many affected by multiple coexisting diseases. This connection is believed to stem from the persistent inflammation and increased levels of pro-inflammatory cytokines characteristic of psoriasis, which contribute to a broader systemic inflammatory response. [1] These comorbidities are thought to arise from the systemic nature of chronic inflammation seen in psoriasis, driven by immune dysregulation and elevated levels of pro-inflammatory cytokines such as IL-17, IL-23, and TNF-α. This persistent inflammatory state not only affects the skin but also contributes to the dysfunction of other organ systems, explaining the broad spectrum of associated conditions.

Some examples are: psoriatic arthritis, cardiovascular disease, metabolic syndrome (such as obesity, diabetes, and dyslipidemia), inflammatory bowel disease etc.

**1) Psoriasis arthritis**

Psoriatic arthritis (PsA) is an inflammatory joint condition that occurs in 20–30% of individuals with psoriasis [1]. It often develops in patients with a long-standing history of cutaneous psoriasis, typically 5 to 12 years after the onset of skin symptoms; however, around 20% of patients may present with joint involvement before any skin manifestations appear [1]. In approximately 70% of cases, psoriasis precedes the development of PsA, underscoring the critical role of dermatologists in identifying early musculoskeletal signs and referring patients for rheumatologic evaluation [2]. While PsA shares some clinical features with rheumatoid arthritis (RA), such as joint inflammation, it is usually seronegative for rheumatoid factor and differs by typically affecting fewer joints, often asymmetrically, and following a less aggressive clinical course [2]. The disease is marked by recurring episodes of enthesitis (inflammation where tendons or ligaments insert into bone) and dactylitis (diffuse swelling of an entire digit) [2]. Importantly, PsA can vary greatly in severity from one person to another, and its joint symptoms do not always correlate with the extent of skin involvement [1]. Joint pain is also frequently reported by patients with psoriasis who have not been diagnosed with PsA. One study found that 51.8% of such patients experienced joint discomfort, with nearly half of them (48.1%) having pain in more than four joints [3]. The pathogenesis of PsA, like psoriasis itself, is multifactorial and involves complex immunological, inflammatory, and vascular processes in genetically predisposed individuals [2]. Notably, many of the immune pathways and cellular infiltrates that drive psoriatic skin lesions are similarly implicated in joint inflammation seen in PsA [2].

**2) Cardiovascular risk**

Psoriasis is increasingly recognized not only as a skin condition but also as a systemic disease with significant implications for cardiovascular health. Patients with psoriasis are more likely to exhibit cardiovascular risk factors such as type 2 diabetes mellitus, obesity, hyperlipidemia, hypertension, and substance use, including tobacco and alcohol. Notably, even after accounting for these traditional risk factors, psoriasis itself has been shown to independently increase the risk of myocardial infarction, particularly in individuals with early-onset or severe disease [1]. One proposed explanation for this connection lies in shared genetic underpinnings. Genes that encode pro-inflammatory cytokines have been implicated in both psoriasis susceptibility and cardiovascular disease (CVD) risk, suggesting a potential genetic link between the two conditions. Supporting this, animal models—specifically studies in transgenic mice—have demonstrated overlapping pathways of inflammation and genetic expression relevant to both disorders [4]. Beyond genetic factors and lifestyle-related risks, psoriasis and cardiovascular disease are also united by common immunopathogenic mechanisms. Atherosclerosis, a hallmark of many cardiovascular diseases, is now considered a chronic immune-mediated inflammatory process. It shares features with psoriasis such as endothelial dysfunction, lipid accumulation in blood vessels, and immune cell infiltration, particularly involving monocyte-derived macrophages. Both conditions are driven by Th17-mediated inflammation and reduced T-regulatory cell activity, underscoring the systemic nature of psoriasis and its contribution to cardiovascular morbidity [5].

## 3. Quality of Life and Psychological Impact

Psoriasis profoundly affects patients' health-related quality of life (HRQoL), often to a degree comparable to or worse than other chronic conditions such as ischemic heart disease and diabetes [8]. While the physical symptoms—visible lesions, discomfort, and pruritus—are distressing, it is the emotional and psychosocial burden that many patients cite as the most debilitating. In a survey by the National Psoriasis Foundation, nearly 75% of respondents reported that psoriasis had a moderate to large negative effect on their daily lives [7], and at least 20% had seriously contemplated suicide [7], highlighting the mental health crisis underlying this condition. These psychological effects can be seen across all age groups, with younger individuals particularly vulnerable to suicidal ideation and body image issues [3]. Importantly, the psychological burden of psoriasis is not necessarily proportional to lesion severity or anatomical location. Patients with minimal visible disease can still experience profound distress, suggesting that perceived "severity" is shaped by both physical and emotional components [8]. Many patients engage in avoidance behaviors, such as hiding their skin or withdrawing from social settings, driven by the fear of being judged or misunderstood due to their appearance [8]. This pattern of maladaptive coping often leads to a persistent state of low-grade stress, which not only diminishes overall quality of life but also impairs treatment response—for example, those identified as "high worriers" undergoing PUVA therapy show delayed or incomplete clearance of lesions [8]. In the workplace, the impact of psoriasis is equally profound. Validated assessment tools like the Work Productivity Assessment Index (WPAI), SF-8, and HADS have shown significantly reduced productivity and increased emotional distress in psoriasis patients [7]. Nearly 60% of individuals report absenteeism directly due to their condition, with an average of 26 missed workdays annually [7]. This absenteeism, combined with the cost of long-term treatment, adds to the financial strain of living with psoriasis [7].

Across the lifespan, patients report challenges that evolve over time—from body image issues and social stigma in adolescence, to career and relationship stress in adulthood, and mental health difficulties throughout [3]. While many patients attempt to manage social discomfort by discussing the non-contagious nature of their condition or concealing lesions [7], such strategies rarely improve QoL, and in some cases, reinforce the cycle of shame and isolation. Encouragingly, studies suggest that open communication about the condition can help reduce stigma and improve social interactions, offering a path toward improved emotional outcomes [7]. Ultimately, the interplay between physical symptoms and psychological distress defines the true burden of psoriasis. Pathological worry and interpersonal difficulties infiltrate every aspect of daily life, yet they are often underrecognized in routine clinical care [8]. Effective psoriasis management must therefore go beyond pharmacologic interventions, integrating psychological support, stress management strategies, and patient-centered care to address the full spectrum of disease impact [3, 8]. Psoriasis profoundly affects patients' health-related quality of life (HRQoL), often to a degree comparable to or worse than other chronic conditions such as ischemic heart disease and diabetes [8].

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## 4. Treatment Approaches

### 1) Microbiota

The intricate relationship between skin, microbiota, and the immune system plays a crucial role in the pathogenesis and progression of psoriasis. Microscopic bacteria, fungi, and viruses colonize the skin's acidic environment, forming a dynamic ecosystem that varies by location (dry, moist, or apocrine-rich areas) and time, with both resident and transient species [9]. This skin microbiota, established at birth through maternal transmission during parturition and breastfeeding, interacts closely with the developing immune system. In neonates, this interaction is modulated by an attenuated inflammatory response of innate immune cells, facilitating symbiosis and immune tolerance [9]. Historically, the connection between microorganisms and psoriasis was first investigated in the early 20th century, particularly with regard to commensal nasopharyngeal streptococci [9]. While early studies lacked definitive evidence, later work by Norrlind et al. established a correlation between streptococcal pharyngitis and guttate psoriasis, as well as with flare-ups of chronic plaque psoriasis [9]. It is now understood that M proteins found on Group A, C, and G β-haemolytic streptococci may mimic keratin determinants, thereby triggering the activation of psoriatic T cells [9]. This is further supported by the interaction between type IV collagen and α1β1 integrin, a receptor found exclusively on psoriatic epidermal T cells, which promotes their expansion and leads to lesion formation [9]. Additional streptococcal antigens such as pyogenic toxins A and B, along with peptidoglycan, are also believed to contribute to T-cell activation in guttate psoriasis [9].

Beyond the skin, the gut microbiota has emerged as another critical player in psoriasis, particularly through its influence on systemic immune regulation. Dysbiosis—or microbial imbalance—within the intestines is commonly observed in psoriasis patients and has been shown to fluctuate with disease severity and activity [10]. Studies reveal inconsistent levels of Prevotella spp. in psoriasis subjects compared to healthy individuals, but consistently point toward microbiome disruption [10]. Specific microbial patterns have been linked to inflammatory markers: Phascolarctobacterium levels show a positive correlation with the IL-2 receptor, while Dialister levels correlate negatively, suggesting that their relative abundance may serve as potential indicators of disease activity [10]. Similarly, higher levels of Escherichia in psoriasis patients are negatively associated with complement 3, a component of innate immunity [10]. Further research has highlighted a decrease in beneficial bacteria such as Lachnospira and. Akkermansia muciniphila in the gut microbiota of psoriasis patients [11]. These findings have been replicated using advanced 16S rDNA sequencing methods, reinforcing the observation that gut microbial alterations are a common feature of the disease [11]. These changes are associated with impaired butanoate metabolism, particularly a reduction in butyrate—a short-chain fatty acid known to regulate inflammatory mediators such as TNF-α, IL-10, and IL-1β [11]. Taken together, these findings support a growing understanding that psoriasis is not merely a localized skin condition but a systemic inflammatory disorder influenced by microbial environments of both the skin and the gut. The dynamic interplay between microbiota and immune responses underscores the potential for targeted microbial therapies in managing psoriasis [9–11].

### 2) Topical Treatments (for mild to moderate psoriasis)

#### a) Corticosteroids:

Topical corticosteroids remain the cornerstone of treatment for psoriasis and are the most commonly prescribed agents due to their rapid anti-inflammatory and immunosuppressive actions [9]. According to [9], “they are classified based on potency, and their selection is influenced by the location and thickness of the lesions.” Potent corticosteroids are often “required for thick, chronic plaques, particularly on the elbows, knees, and scalp” [13]. However, long-term use must be monitored carefully, as it can lead to adverse effects such as “skin atrophy, telangiectasia, tachyphylaxis, and hypothalamic-pituitary-adrenal axis suppression” [13]. The review in [12] adds that corticosteroids “exhibit a rapid onset of action and are useful during acute exacerbations.”

#### b) Vitamin D analogs:

Vitamin D analogs are considered a safe and effective alternative to corticosteroids and function by regulating keratinocyte proliferation and differentiation [9]. As noted in [9], “calcipotriene is effective both as monotherapy and in combination with topical corticosteroids.” The combination of calcipotriene and betamethasone dipropionate “is more effective and better tolerated than monotherapy” [9]. According to [13], “Vitamin D analogues do not induce skin atrophy and are useful in both the induction and maintenance phases of therapy.” However, irritation may occur, especially on sensitive areas or when used in excessive amounts.

#### c) Coal Tar:

Coal tar has been used for more than a century in the treatment of psoriasis and is known for its antiproliferative and anti-inflammatory effects [9]. “Although less commonly used today, coal tar remains an effective and inexpensive topical therapy,” as stated in [12]. According to [13], “Coal tar preparations are widely used in many countries, especially in

combination with UV therapy." However, cosmetic issues such as "strong odor, messiness, and staining of clothes and bedding" reduce its acceptability among patients [12, 13].

**d) Salicylic Acid:**

Salicylic acid acts as a keratolytic agent that helps in softening and removing scales, thus enhancing the penetration of other topical agents like corticosteroids and vitamin D analogs [13]. "It is commonly used as an adjunct in the treatment of chronic plaque psoriasis, " especially in scalp formulations [13]. However, it should be used with caution in children and in large body areas due to the risk of systemic absorption and salicylism [13]. It is generally more effective when combined with corticosteroids in ointment bases for thick plaque removal [13].

e) **Calcineurin Inhibitors:**

These non-steroidal immunomodulators inhibit T-cell activation and are particularly useful in areas where corticosteroids are less appropriate, such as the face, intertriginous zones, and genital regions [12]. As noted in [12], "they are well tolerated and provide a suitable alternative for sensitive skin areas. " While not FDA-approved for psoriasis, tacrolimus and pimecrolimus have shown efficacy in treating facial and inverse psoriasis [12]. The agents are associated with less risk of skin atrophy and can be used for prolonged durations in these delicate sites [13].

f) **Tazarotene:**

Tazarotene is a topical retinoid derived from vitamin A that exerts both anti-inflammatory and anti-proliferative effects on psoriatic lesions [12]. "It is particularly effective in treating localized plaque psoriasis, especially when combined with corticosteroids to mitigate irritation" [12]. According to [13], "it normalizes keratinocyte differentiation and reduces scaling, thickness, and erythema of plaques." However, due to its potential for irritation, especially on sensitive skin, it is generally reserved for limited body surface area use and often applied at night [13].

**3) Phototherapy (for moderate to severe psoriasis)**

Phototherapy continues to be a central treatment option for moderate to severe psoriasis, particularly in patients who have not responded adequately to topical agents or are unsuitable for systemic therapies. The approach works by using ultraviolet (UV) light to slow down keratinocyte proliferation and reduce inflammation, which helps control psoriatic lesions [9]. Several types of phototherapy are available, including broadband UVB (BB-UVB), narrowband UVB (NB-UVB), psoralen plus UVA (PUVA), and targeted devices like the excimer laser. Among these, narrowband UVB (NB-UVB) is currently the most widely used and preferred modality. Compared to BB-UVB, NB-UVB offers improved efficacy and fewer side effects, making it a safer and more effective option overall [9]. It emits light in a specific range (around 311–313 nm) that has been shown to effectively reduce psoriatic inflammation and normalize skin cell turnover. In clinical practice, NB-UVB has demonstrated substantial success, with clearance of lesions in approximately 70% of patients within 20 to 30 sessions [9]. One major advantage of this modality is that it does not require the use of photosensitizing drugs, which enhances its tolerability and long-term safety.

NB-UVB is often the first-line phototherapy for plaque psoriasis and is considered by many experts to be the gold standard for phototherapeutic management of psoriasis vulgaris [14]. It is suitable for both adults and children, and it can be used long-term under medical supervision. Treatment typically involves two to three sessions per week, gradually increasing the UV dose until visible improvement is achieved. For more extensive or resistant forms of the disease, PUVA therapy may be considered. This treatment combines UVA light with psoralen, a photosensitizing compound administered orally or topically before the light exposure. PUVA can offer superior efficacy in some patients, particularly those who have not responded to NB-UVB or who have thicker plaques or involvement of areas like the palms and soles [14]. However, its use is limited by a greater risk of side effects, including nausea, phototoxic skin reactions, and long-term concerns such as photoaging and increased skin cancer risk with cumulative exposure [9, 14]. As a result, PUVA is typically reserved for select cases where the benefits outweigh the risks.

Another valuable phototherapeutic option, particularly for localized disease, is the 308 nm excimer laser. This modality delivers a focused beam of monochromatic UVB light, allowing for precise treatment of individual psoriatic plaques while sparing the surrounding healthy skin [9]. It is especially useful in treating small, stubborn areas such as the scalp, elbows, knees, and genital regions. Evidence suggests that the excimer laser can induce rapid clearance of lesions, often with fewer sessions compared to full-body phototherapy [15]. Patients with limited but treatment-resistant psoriasis may benefit greatly from this targeted approach. According to clinical findings, significant improvement can be observed after as few as 6 to 10 sessions, which adds to its convenience and efficiency [15]. Choosing the appropriate phototherapy depends on several factors, including disease severity, lesion location, patient preferences, and prior treatment responses. Phototherapy in general is most appropriate for patients with widespread lesions who have failed topical treatments or for whom systemic agents may not be appropriate due to contraindications or safety concerns [9]. Moreover, phototherapy can be safely combined with other treatments, including topical corticosteroids or vitamin D analogs, to enhance efficacy and reduce recurrence.

In summary, phototherapy remains a vital and adaptable tool in the management of psoriasis. Narrowband UVB is the preferred first-line option for many patients due to its safety and effectiveness. PUVA, while more potent in certain scenarios, is used selectively due to its side-effect profile. The excimer laser offers a modern, precise alternative for localized disease, particularly when rapid results are desired. When carefully tailored to the individual patient, phototherapy can provide significant improvement in quality of life and long-term disease control [9, 14, 15].

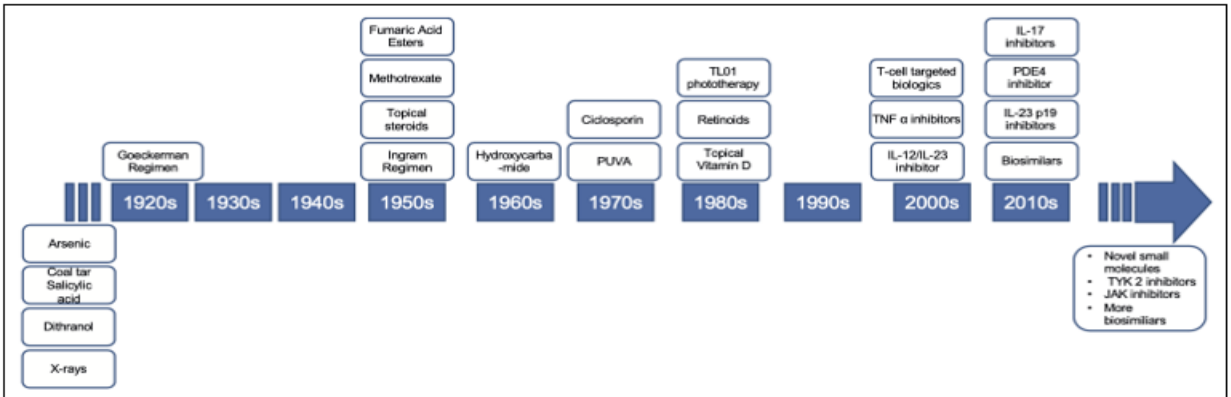


**Fig. 1. Advances in the treatment of psoriasis over the past century.** This timeline illustrates the major pharmacological advances which have occurred in the management of psoriasis over the past 100 years. It also speculates as to what may be the important therapies in the near future. IL: interleukin; PDE: phosphodiesterase; PUVA: psoralen plus ultraviolet (UV)-A; TNFα: tumour necrosis factor alpha; TYK: tyrosine kinase; JAK: janus kinase.

## 5. Conclusion

Psoriasis is a complex, systemic inflammatory condition that extends beyond the skin, affecting joints, cardiovascular health, and mental well-being. The interplay between genetic factors, immune system dysfunction, microbiota alterations, and environmental stressors contributes to its varied clinical presentation and chronic course. While traditional treatments like topical corticosteroids and phototherapy remain important, advancements in our understanding of immune pathways and the microbiome have expanded therapeutic options. Addressing the psychological and social dimensions of psoriasis is equally critical for improving patient outcomes. Ultimately, an individualized, holistic approach that combines medical, lifestyle, and psychosocial interventions offers the most promise for effective, long-term disease control and improved quality of life.

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