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Article

Pathophysiology of Psoriasis: A Review

Ruqaya M. Hassan[1], Yousif Sh. Raheem[2], Mawj N. Modher[3], Sarmed M. Hussein*[4]

1. Department of Biotechnology, College of Science, University of Baghdad, Baghdad, Iraq
2. Department of Biology, college of science, University of Baghdad, Baghdad, Iraq
3. Biotechnology Research Center, Al-Nahrain University, Baghdad, Iraq
4. Tropical-biological Research unit, College of Science, University of Baghdad, Baghdad, Iraq

*Correspondence: sarmed.m@sc.uobaghdad.edu.iq

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Abstract: A persistent immune-related reactive body disorder, psoriasis, with keratinocyte hyperproliferation and immune system dysfunction. Genetics, environment, and cytokine-mediated inflammation cause illness. The IL-23/Th17 axis is crucial, since Th17 cells release cytokines such as IL-17, IL-22, and TNF-α, causing epidermal hyperplasia, impaired barrier function, and persistent inflammation. Genetic studies have connected HLA-Cw6 to susceptibility loci such as PSORS1 on chromosome 6p21. As activated dendritic cells, T cells, and keratinocytes perpetuate inflammation, innate and adaptive immune responses prolong illness. Psoriatic arthritis, heart disease, and insulin resistance are immunopathological outcomes of the condition. Biological treatments targeting TNF-α, IL-17, and IL-23 have changed therapy, significantly reducing illness. These developments do not eliminate therapeutic difficulties such as medication response variability and return of disease after treatment. The microbiome, epigenetic alterations, and new immunological targets are being studied to provide more effective, tailored treatments. Psoriasis therapy and patient outcomes depend on understanding the molecular processes of the disease.

Keywords: Psoriasis Pathophysiology; Immune Dysregulation; IL-23/Th17 Axis; Genetic Susceptibility; Inflammatory Cytokines

Introduction

A chronic skin inflammation condition known as psoriasis causes rapid keratinocyte growth and systemic immune problems. Scaly, erythematous lesions result. Environmental, genetic, and immunological factors cause psoriasis. About 2-3% of individuals worldwide have psoriasis, which causes erythematous, indurated, scaly skin patches and occasionally nails and joints. It is characterized by excessive and disordered keratinization and epidermal cell proliferation [1]. Even while our understanding of the issue has grown, the causes of aberrant keratinization remain unknown. In psoriasis, increased cAMP, protein kinase C, epidermal growth factor receptor binding, and TGF-α levels suggest T cell dysfunction. An agreement exists that psoriasis is an immune-related inflammatory skin condition that develops in naturally predisposed persons exposed to environmental chemicals or triggers. Numerous immunomodulatory psoriasis treatments back this notion [2]. Recently, a barrier deficiency in psoriasis and the pro-inflammatory involvement of the NLR/CATERPILLAR (nucleotide-binding domain) group of DNA and bacteria have changed the spotlight from T lymphocytes to skin cells as the principal pathogens. Figure 1 demonstrates the

evidence for lymphocyte- or keratinocyte-centric psoriasis pathogenesis. Psoriasis may be caused by genetic predisposition, skin barrier abnormalities, and innate and adaptive immune dysregulation. The pathophysiology of psoriasis involves several immunologic, environmental, and genetic factors [3].

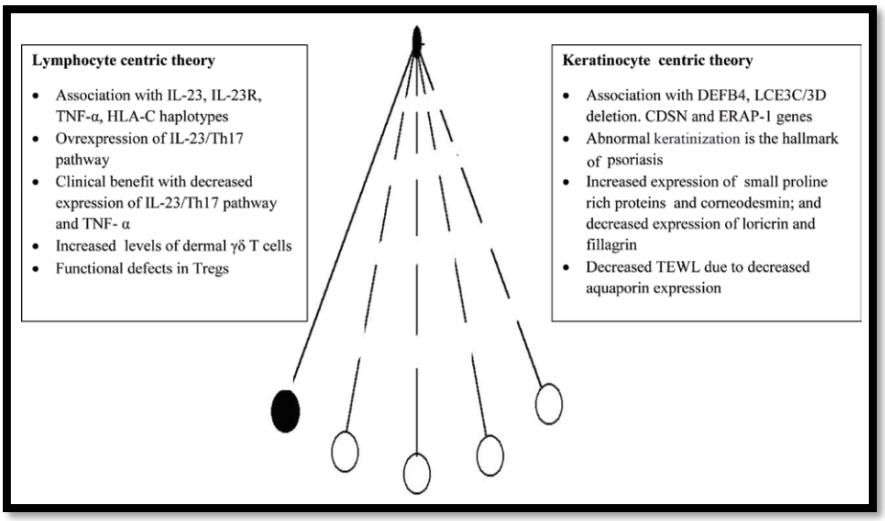

Figure 1. Evidence supporting lymphocytes or keratinocytes in psoriasis etiology [4].

A new research emphasizes the significance of T cells, specifically Th17 cells, in the disease's pathophysiology, together with pro-inflammatory cytokines including IL-17 and TNF-α. Epigenetics, microbiome, and innate and adaptive immune systems complicate psoriasis. In psoriasis, a chronic immune-mediated inflammatory skin disease, keratinocyte hyperproliferation, epidermal differentiation abnormalities, and immune cell infiltration cause erythematous, scaly plaques, external stresses and dysregulated immune responses, including the IL-23/Th17 axis, produce this complex disorder. The MHC class I allele HLA-Cw6 is strongly connected to PSORS1 on chromosome 6p21, one of several susceptibility loci [5]. In especially susceptible persons, infections, trauma (Koebner phenomenon), stress, and beta-blockers and lithium may cause or aggravate psoriasis. A dysfunctional immune response causes dendritic cells, T cells, and keratinocytes to cycle in an inflammatory cycle (psoriasis). Activated dermal cells produce TNF-α, IL-12, and IL-23, promoting Th1 and Th17 cell proliferation in naïve T cells [6].

Th17 cells release IL-17A, IL-17F, IL-22, and other proinflammatory cytokines that cause keratinocyte hyperproliferation, inflammation, and epidermal barrier dysfunction. IL-17 and TNF-α work together to produce antimicrobial peptides, chemokines, and cytokines that attract neutrophils and intensify inflammation. Psoriasis has extended rete ridges, parakeratosis, Munro micro abscesses, and a reduced granular layer [7]. Increased VEGF expression in psoriatic plaques contributes to an aberrant vascular network; angiogenesis is also important. Epidermal keratinocytes also react to inflammatory cytokines and enhance the immune response by producing CCL20, which draws additional Th17 cells to the lesion. Epigenetic alterations, metabolic dysregulation, and gut microbiome interactions sustain psoriasis' chronicity, indicating a systemic cause. Psoriasis is now considered a systemic inflammatory illness linked to metabolic syndrome, cardiovascular disease, and psoriatic arthritis. Non-coding RNAs, dysbiosis, and neuroimmune interaction affect illness severity and progression, according to a new study [8]. Biologic therapies targeting TNF-α, IL-17, IL-23, and JAK-STAT signaling pathways have revolutionized psoriasis treatment, resulting in significant clinical improvements for moderate-to-severe disease patients. Therapeutic problems include medication response variability, illness return after withdrawal, and the need for genetic, immunologic, and metabolic profile-based treatments. Despite advances in psoriasis pathophysiology, more research is needed to understand the molecular mechanisms of disease initiation and persistence to develop novel therapeutic strategies for long-term disease remission and improved quality of life [9]. This review consolidates current knowledge about the pathophysiological mechanisms of psoriasis, identifies key players, and discusses potential therapeutic targets to improve management strategies.

1. Understanding Psoriasis: An Overview of Pathophysiology

Dysplastic keratinocyte development, inflammation, and immune cell infiltration characterize chronic, immunological-mediated psoriasis. Erythematous plaques with silvery scales affect the scalp, elbows, knees, and lower back. Psoriasis is caused by a complex interplay of genetics, environment, and immunological dysregulation. The most strongly associated susceptibility locus is the PSORS1 locus on chromosome 6p21, especially the HLA-Cw6 allele. Environmental variables, including infections, stress, trauma, and drugs, might worsen the condition in genetically predisposed people [10]. Innate and adaptive immune response disorders cause psoriasis. TNF-α, IL-12, and IL-23 released by activated cutaneous dendritic cells promote naïve T cell differentiation into Th1 and Th17 subsets. Th17 cells produce IL-17A, IL-17F, and IL-22, which increase keratinocyte development and chronic inflammation. IL-17 boosts inflammation, attracts neutrophils, and alters epidermal barrier function. An inflammatory cycle caused by cytokine overproduction causes epidermal hyperplasia, parakeratosis, and aberrant keratinocyte differentiation [11] Psoriasis has thickened epidermis (acanthosis), extended rete ridges, loss of the granular layer, and Munro micro abscesses neutrophil clusters in the stratum corneum. Psoriatic lesions have erythema due to enhanced angiogenesis and vascular remodeling. Psoriasis is now considered a systemic inflammatory illness linked to metabolic syndrome, cardiovascular disease, and psoriatic arthritis. Comprehensive disease treatment is needed due to systemic inflammation [12]. Targeted medicines such as biologics inhibiting important cytokines, including TNF-α, IL-17, and IL-23, have been developed to treat psoriasis (e.g., adalimumab, infliximab, secukinumab, ixekizumab, guselkumab, risankizumab). These medicines have transformed moderate-to-severe psoriasis therapy, improving outcomes. Despite these advances, therapeutic response prediction and long-term remission remain difficult. Genetic, epigenetic, and environmental research uncovers new illness processes, enabling more effective, individualized treatments [13].

2. Psoriasis Pathogenesis

Psoriasis's pathogenesis is unclear. Overactivation of adaptive immune system components may contribute to psoriasis. During early psoriasis pathogenesis, macrophages, keratinocytes, natural killer T cells, and plasmacytoid dendritic cells produce cytokines that excite myeloid dendritic cells. Complexes like DNA-LL37 stimulate myeloid dendritic cells. The T H17 pathway is activated by IL-23 most often. Tyk2-Jak2 and STAT3 intracellularly mediate IL-23 signaling, producing key inflammatory compounds. They also boost angiogenic mediators, endothelial adhesion proteins, keratinocyte growth, and immune system penetration into lesional tissue [2], as shown in Figure 2.

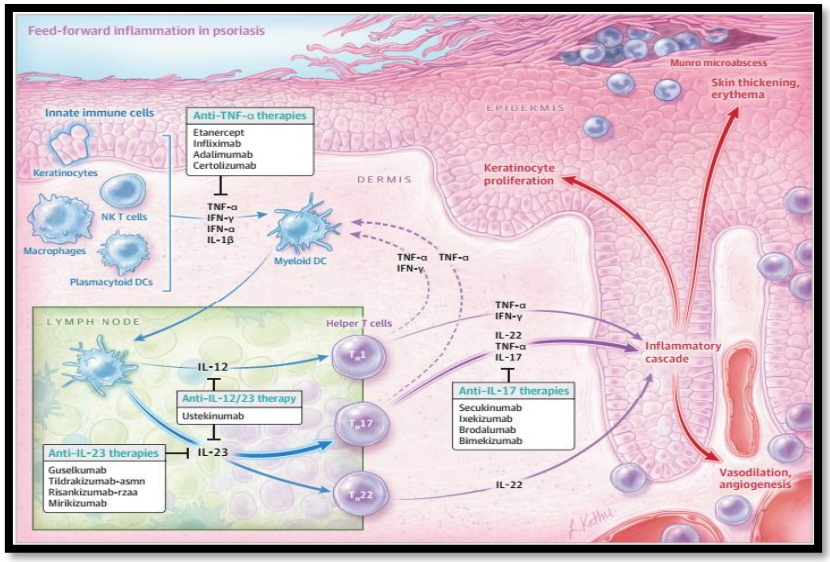

Figure 2. Pathophysiology of Psoriasis.

3. Genetic Factors' Role

There is strong evidence that hereditary factors mostly influence psoriasis. 9.8% of children in research conducted in northern India had a family history of psoriasis; in another study conducted in Kuwait, the percentage reached 28 %. A kid has a sixteen percent chance of getting psoriasis if just one parent has it. If both parents suffer from psoriasis, the likelihood rises to 50%. Twin pair study shows

72% concordance for monozygotic twins and 22% for dizygotic twins. Molecular patterning makes males more likely to carry on psoriasis [14]. A few non-major histocompatibility complex (MHC) susceptibility loci have been discovered, although they have a low disease risk and may not be useful for prediction. Chen et al. constructed a psoriasis global genetic risk score (GRS) using 10 SNPs that have been identified as susceptibility loci. Of the ten SNPs they analysed, the HLA-C gene at rs10484554 exhibited the greatest signal, increasing psoriasis risk by 206%. WGRS, which weights each risk allele by the logarithm of odds ratio, was slightly associated with guttate psoriasis but not psoriatic arthritis. In western India, Umapathy et al. discovered a strong connection between psoriasis and HLA-A2, B8, and B17 antigens [15].

4. Impaired Skin Barrier Role in Psoriasis

Psoriasis is characterized by skin keratinocyte hyperproliferation and abnormal differentiation, T lymphocyte penetration, and skin layer capillary vascular changes such as angiogenesis, dilatation, and HEVs. Water and electrolytes cannot travel through the skin because it functions as a two-way barrier. The majority of the barrier is found in the epidermis, with the remaining dermis being almost entirely permeable if the epidermis is removed. The separated epidermis is impenetrable, like the covering of skin. Stratocum corneum contains the epidermal barrier [16]. Both the intercellular material, especially lipids, and the cornified substance of the keratinocytes are necessary for the barrier to function. It is widely believed that the stratum corneum functions as a barrier in two compartments, with corneocytes, protein-rich cells embedded inside a continuous matrix rich in lipids. Normal skin flora and harmful microbes cannot infiltrate the skin while the stratum corneum is intact. Skin conditions and minor wounds may serve as entrance points for bacteria, especially Staphylococci or Streptococci. The initial line of immunological defense is provided by AMPs, which are peptides found on the epidermis and its appendages [17]. Six of the seven β-defensin genes, aside from DEFB1, are spread throughout a sizable repeat unit on chromosome 8p23.1, with copy numbers that may fluctuate. Increased CNV of the β-defensin gene cluster has been associated to psoriasis in studies comparing cases to controls. High levels of hBD-2 β-defensin are linked to psoriatic lesions. High defensin levels could suggest why psoriatic aggregates have fewer skin infections despite their proinflammatory characteristics. Large β-defensin copy numbers may increase the immune system's reaction to minimal stimuli, explaining the KP 17]. Cathelicidin LL-37, which is increased in psoriasis-related inflammation skin, binds to deceased cells' exposed self-DNA and stimulates plasmacytoid cells, called dendritic cells. Next, pDCs release type I interferons, starting an auto-inflammatory cascade [18].

5. Immune System's Role in Psoriasis Development

Psoriasis is a caused by the immune system inflammatory skin disorder involving keratinocyte hyperproliferation, immune cell infiltration, and inflammation. Psoriasis develops due to both adaptive and innate immune dysregulation. Psoriasis pathogenesis and focused treatment are explained by immune cells, cytokines, and keratinocytes' complex interactions [19].

A. Psoriasis and Innate Immunity

Psoriasis is caused by the innate immune system. Infections, trauma, and stress activate dendritic cells, macrophages, and neutrophils, increasing inflammation. AMPs, particularly LL-37, activate plasmacytoid dendritic cells (pDCs) by complexing with self-DNA, leading to type I interferon production (IFN-α and IFN-β). Activated by interferons, myeloid dendritic cells (mDCs) release pro-inflammatory cytokines such TNF-α, IL-12, and IL-23. Neutrophils induce inflammation in psoriatic lesions by creating neutrophil extracellular traps. NETs stimulate dendritic cells and sustain inflammation using DNA, histones, and antimicrobial proteins. Additionally, psoriatic skin macrophages emit pro-inflammatory cytokines including TNF-α, promoting immunological activation and keratinocyte growth [20].

B. Psoriasis and Adaptive Immunity

Chronic inflammation in psoriasis is maintained by the adaptive immune system, notably T cells. Activated myeloid dendritic cells lead naïve T cells to grow into Th1 and Th17 subsets, causing psoriatic inflammation. The Th1 pathway includes IL-12 leading to the differentiation of naïve T cells into Th1 cells that produce IFN-γ and TNF-α. Cytokines activate dendritic and keratinocytes, causing inflammation. IFN-γ enhances endothelial cell adhesion, allowing immune cells to penetrate psoriatic skin. [21] In instance, IL-17A boosts CCL20 production, which attracts additional immune cells to the

skin and sustains inflammation. Psoriasis plaques thicken owing to IL-22-induced keratinocyte hyperproliferation and epidermal acanthosis. Psoriasis may include dysfunctional regulatory T cells (Tregs), which limit excessive immune activation and inflammation. Treg dysfunction favours Th1 and Th17 cells by counterbalancing pro- and anti-inflammatory responses [21].

C. Cytokine Network and Psoriasis Development

Psoriasis progression depends on the cytokine network. The inflammatory trio of TNF-α, IL-17, and IL-23 drives psoriatic pathology. The activation of dendritic cells, T cells, and keratinocytes by TNF-α leads to inflammation. IL-17A and TNF-α collaborate to produce pro-inflammatory mediators, whereas IL-23 promotes pathogenic Th17 cell growth. Additional cytokines, such as IL-6, IL-1β, and IL-36, further enhance the inflammatory process [22].

D. Treating Psoriasis Using Immune System Targets

Targeted biologic treatments that block major inflammatory cytokines have been developed to treat psoriasis due to the immune system's essential involvement. Initially approved for psoriasis, TNF-α inhibitors including infliximab, etanercept, and adalimumab have shown efficacy. The IL-23/Th17 axis is unique to psoriasis, thus recent advances have targeted it. IL-17 and IL-23 inhibitors like secukinumab, ixekizumab, brodalumab, guselkumab, risankizumab, and tildrakizumab are more efficacious and safe [23].

Although breakthroughs have been made, understanding individual therapy responses and illness severity remains difficult. The immunological systems of psoriasis will be studied to generate new treatments with fewer adverse effects. Psoriasis is a complicated immune-mediated illness caused by innate and adaptive immune responses. The distinctive skin lesions are caused by a self-sustaining inflammatory cycle involving dendritic cells, Th1 and Th17 cells, and pro-inflammatory cytokines. Immunology has transformed psoriasis therapy with tailored medications. We need more research to create tailored treatments and find more molecular targets for better long-term illness management [24].

6. Genetic Factors Contributing to Psoriasis Pathophysiology

A complicated, multifaceted illness, psoriasis' pathophysiology is heavily influenced by genetics. Family and twin studies indicate substantial heritability, with genetic predisposition determining illness risk and severity. This area contains HLA-Cw6, an MHC class I allele highly associated to early-onset psoriasis and disease severity. HLA-Cw6 may offer self-antigens to T lymphocytes to activate them, causing an overreaction to keratinocytes. Other immune-related genes contribute to psoriasis' dysregulated inflammatory response than MHC-related genes [25]. Psoriasis genetic control is confounded by non-coding RNAs and epigenetic alterations that impact gene expression. Despite genetic results, psoriasis is polygenic and worsened by infections, trauma, and stress. The identification of genetic pathways has led to the creation of IL-17, IL-23, and TNF-α inhibitors, improving therapy outcomes. Genetic variability influences therapeutic response, emphasizing the need for personalized medicine. Discovering new genetic risk factors and their links to immune signaling pathways helps explain illness etiology and therapy targets. Understanding psoriasis genetics helps diagnosis, prognosis, and precision medicine, improving patient outcomes and disease management [26].

7. Cytokines and Inflammatory Pathways in Psoriasis

Chronic skin inflammation A complex network of cytokines and immunological pathways promotes keratinocyte hyperproliferation and immune cell infiltration in psoriasis. The IL-23/Th17 axis promotes inflammation and illness. IL-23 from dendritic cells promotes Th17 cell growth and survival by producing IL-17A, IL-17F, and IL-22 [25]. Cytokines cause keratinocyte proliferation, poor differentiation, and pro-inflammatory mediators so TNF-α, IL-6, and CCL20, attracting antibodies to psoriatic lesions. IL-17A and TNF-α, a major cytokine in psoriasis, work together to increase inflammation by promoting chemokine and antimicrobial peptide synthesis, promoting immune activation. Another important Th17 cytokine, IL-22, causes epidermal hyperplasia and impairs skin barrier function, causing thick, scaly plaques in psoriasis [26].

Furthermore, IL-12 enhances Th1 cell development, resulting in increased IFN-γ production, activating macrophages and dendritic cells, sustaining the inflammatory process. By activating keratinocytes and recruiting neutrophils, IL-36, an IL-1 family member, amplifies psoriatic inflammation. These cytokines create a self-sustaining inflammatory cycle that causes persistent skin

inflammation. Targeted biological therapies like TNF inhibitors (adalimumab, infliximab), IL-17 inhibitors (secukinumab, ixekizumab), and IL-23 inhibitors (guselkumab, risankizumab) have revolutionized psoriasis treatment by disrupting key inflammatory pathways. Novel cytokine interactions and their effects on illness persistence are being studied to provide more effective and tailored long-term disease management therapies [26].

8. Psoriasis and Comorbidities: A Pathophysiological Perspective

Increasing evidence links psoriasis to a variety of comorbidities caused by common immunopathogenic pathways, making it a systemic inflammatory condition. One of the most well-known comorbidities is psoriatic arthritis (PsA), an inflammatory joint disease that affects 30% of psoriasis patients and is driven by the same IL-23/Th17 axis as skin pathology. It causes synovial inflammation, enthesitis, and bone erosion [27]. Beyond musculoskeletal involvement, as systemic inflammation accelerates endothelial dysfunction, increases oxidative stress, and promotes thrombosis (28).

Increased CRP and TNF-α levels correlate with vascular inflammation, making psoriasis a risk factor for cardiovascular disease. Metabolic syndrome (MetS) is common in psoriasis patients, including obesity, insulin resistance, dyslipidaemia, and hypertension. IL-6 and TNF-α contribute to dysfunction in adipose tissue and impaired glucose metabolism, raising the risk of type 2 diabetes. Since common genetic predispositions such IL23R polymorphisms and overlapping immunological dysregulation including Th17-mediated pathways, psoriasis has been related to inflammatory bowel illnesses (IBD) like Crohn's disease and ulcerative colitis [28].

9. Current Research Trends in Psoriasis Pathophysiology

Psoriasis research has recently discovered new molecular pathways, therapeutic targets, and systemic aspects of the disease. As the main cause of psoriatic inflammation, the IL-23/Th17 immunological axis is under study. Studies continue to examine how IL-17A, IL-17F, IL-22, and IL-36 promote keratinocyte hyperproliferation and immune cell recruitment, causing persistent skin inflammation. Single-cell RNA sequencing (scRNA-seq) is also being used to identify novel dendritic, T, and macrophage subsets that contribute to disease pathogenesis in psoriatic lesions. Other trends include studying the skin microbiota and its involvement in immune responses. Microbial dysbiosis may affect disease severity since psoriatic flares have been linked to a drop in Cutibacterium species and an increase in Streptococcus [29]. In addition, genetic and epigenetic research are finding novel psoriasis susceptibility loci and regulatory mechanisms such as non-coding RNAs and DNA methylation that regulate inflammatory pathways. Beyond the skin, psoriasis is being studied for its systemic effects on cardiovascular disease, metabolic syndrome, and inflammatory bowel disease. Recent research indicates that psoriasis and atherosclerosis share inflammatory pathways, with TNF-α, IL-17, and IL-6 contributing to endothelial dysfunction and cardiovascular risk. Chronic systemic inflammation affects the central nervous system, which may contribute to psoriasis-related sadness and anxiety. Next-generation biologics and small-molecule inhibitors targeting IL-23, IL-17, and JAK-STAT signaling are being developed as treatments, combined with gene-editing technologies like CRISPR to tune immune responses genetically. AI and machine learning are also transforming psoriasis research by providing precision medicine techniques that predict disease progression and therapy responses based on genetic and immunological characteristics. These research trends might improve targeted medicines, disease management, and psoriasis sufferers' quality of life as we learn more about pathogenesis [30].

10. Therapeutic Implications of Psoriasis Pathophysiology

Targeted treatments for immune dysregulation have emerged from psoriasis pathophysiology research. Topical corticosteroids, vitamin D analogs, and phototherapy cure symptoms but not the cause of psoriasis. However, understanding the IL-23/Th17 axis has enabled the creation of biological treatments that specifically inhibit inflammatory cytokines, revolutionizing therapeutic tactics. The first biologics licensed for psoriasis, such as TNF inhibitors like adalimumab, infliximab, and etanercept, target TNF-α, a key mediator of inflammation. Effective, but prone to infections and subsequent treatment failure owing to anti-drug antibodies [10]. Secukinumab, ixekizumab, and brodalumab, which directly block IL-17A or its receptor, have become extremely successful treatments for keratinocyte hyperproliferation and immune cell recruitment. IL-23 inhibitors, such as guselkumab,

risankizumab, and tildrakizumab, preferentially target IL-23 to limit Th17 cell development and cytokine production, resulting in longer-lasting remission. JAK and TYK2 inhibitors like tofacitinib and deucravacitinib are also being studied for their capacity to alter intracellular inflammatory signaling. Beyond biologics, personalized medicine research optimizes therapeutic responses by tailoring therapies to genetic, immunologic, and microbial profiles. Psoriasis is now recognized as a systemic illness. Therefore, multidisciplinary treatments target comorbidities, including cardiovascular disease, metabolic syndrome, and depression. Predicting illness severity and therapeutic results using AI and machine learning is improving treatment techniques. With current research into CRISPR, next-generation biologics, and microbiome-targeted medicines, psoriasis therapy may become more effective and long-lasting [31].

Conclusion

Genetic vulnerability, immunological dysregulation, and environmental variables cause psoriasis, a complicated inflammatory condition. The IL-23/Th17 axis is crucial for disease development, since inflammatory cytokines, including IL-17, IL-22, and TNF-α, promote keratinocyte hyperproliferation and immune cell infiltration. Stress and infections worsen symptoms, but genetic predisposition, notably the PSORS1 locus, affects illness susceptibility. Psoriasis may cause systemic comorbidities such as psoriatic arthritis, cardiovascular disease, and metabolic syndrome, requiring a multifaceted therapy. Key cytokine biologic treatments have improved illness management by controlling symptoms, although therapy resistance and recurrence persist. Research into the microbiota, epigenetic factors, and immunological signalling pathways is enabling more focused and personalized treatments. Precision medical advances like AI-driven diagnostics and gene-based therapies may lead to long-term illness remission. Psoriasis patients will have better therapy, results, and quality of life if they understand pathogenesis.

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