# Psoriasis and Periodontitis – Shared Inflammatory Mechanisms and Clinical Implications

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**SUMMARY**

Psoriasis is a chronic, autoimmune, genetically determined and frequently recurring skin disease characterized by specific clinical features in the form of erythematous squamous plaques. The development of psoriasis is influenced by genetic and environmental factors, as well as a complex inflammatory cascade. To this day, there is no precise information identifying the main trigger of the inflammation and formation of psoriatic plaques. In patients suffering from psoriasis, an increased prevalence of periodontitis has been observed. Severe forms of periodontitis are the 6$^{th}$ most common chronic disease, with a prevalence of 11.2% and affecting more than 800 million people worldwide. The chronic inflammation and immune mechanisms involved in the development of this disease remain a subject of ongoing research and the search for key therapeutic options. In this review paper, we explored the relationship between the chronic inflammatory immune processes underlying psoriasis and periodontitis, as well as the potential frequency and possibility of triggering the onset of a multisystem disease, such as psoriasis, in patients with periodontal disease.

**KEYWORDS**

*Psoriasis*; *Periodontitis*; *Systemic inflammation*; *Chronic infections*

## Introduction

Psoriasis is a chronic, inflammatory, multifactorial systemic disease that primarily affects the skin and joints, but it can be related to other organs, such as those of the cardiovascular system, etc. It has been a very well-known disease for centuries and the first one to mention and describe it was Hippocrates[1].

Psoriasis is a disease that occurs worldwide, affecting both sexes equally with a global prevalence of approximately 2%. It is more common among Europeans and Caucasians compared to people of African ancestry, in whom the prevalence is approximately half as frequent[2]. According to research, less than 20% of countries have epidemiological data on the number of patients with psoriasis, and a consistent increase in the number of affected individuals has been recorded. In the Chinese population, an increased BMI has been observed in as many as 63% of psoriasis patients compared to the control population and it is associated with at least one comorbidity. This indicates that psoriasis is not just a disease related to skin changes, but rather a systemic disease[3].

With the advancement and development of science and medicine, it has become clear that psoriasis is not merely a disease limited to the skin, but rather a multisystem autoimmune disease caused by chronic inflammation throughout the body, and one that may be mediated by genetic mutations[2,3]. In 1985, Hensel and Christopher theoretically classified psoriasis into two types. Type 1 occurs earlier in life, presents with a more severe clinical course and is more frequently associated with the HLA-Cw06 genetic mutation. Type 2, on the other hand, refers to "adult-onset" psoriasis, which typically appears later in life (around the ages of 60 to 65) and is characterized by a milder clinical presentation without the presence of the mentioned HLA gene mutation.

Psoriasis presents in several clinical forms. The most common is plaque psoriasis, characterized by erythematous, scaly plaques with superficial white-silver scales, typically affecting the elbows, knees, lower trunk, intergluteal region and scalp (Fig. 1). Guttate psoriasis appears as droplet-shaped lesions, is most common in children and can be triggered by bacterial throat infections. The lesions usually begin on the trunk and spread centrifugally. Inverse psoriasis affects intertriginous areas, such as the axillae, popliteal fossae, the groin and other skin folds. Erythrodermic psoriasis is a severe form that may develop in untreated cases, often in patients with limited awareness or resources for disease management. Psoriasis can also involve the nails. Nail psoriasis is characterized by changes in the fingernails and toenails, which may be localized to a single nail and present as punctate depressions, spots or onycholysis[2,3].

The typical histological characteristics of psoriasis can be observed in both the epidermis and dermis. The epidermis shows acanthosis (a thickening of its outermost layer) with parakeratosis (the presence of nucleated corneocytes) and elongated rete ridges, giving it a "comb-like" appearance as the enlarged dermal papillae extend upward. The granular layer of the epidermis is either very thin or absent, and there is an increased number of mitotic figures above the basal layer. In the parakeratotic superficial layer, clusters of neutrophils known as Munro's microabscesses can be seen and they sometimes

**FIG. 1.** Psoriatic plaque

form larger pustules referred to as Kogoj's pustules. The dermal papillae contain dilated capillaries, which are responsible for the Auspitz phenomenon; when psoriatic plaque is scraped, pinpoint bleeding occurs from these vessels. The dermis also shows an inflammatory infiltrate composed mainly of mononuclear cells, along with neutrophils, mast cells and lymphocytes[4]. Psoriasis is a clinical diagnosis, and a biopsy of skin changes is not necessary, but can be used to confirm the diagnosis[2-4].

The severity and extent of the disease are assessed using standardized tools currently available and applied during the patient's physical examination. The Body Surface Area (BSA) scale is used to estimate the percentage of the body surface affected by skin lesions, where 1% corresponds to the area of the patient's palm[5]. The Psoriasis Area and Severity Index (PASI) scale incorporates the previously mentioned BSA, as well as the presence of erythema, plaque induration and scaling on the plaque surface. Each of these parameters is scored from 0 to 4, and combined with the affected body surface area, they yield a total score indicating the severity of the clinical presentation as determined by physical examination[5,6]. In addition to these two tools, it is also necessary to include the patient's subjective perception, which reflects their quality of life, i.e. how much the disease affects their daily functioning. Another useful tool is the Dermatology Life Quality Index (DLQI), a questionnaire that covers various aspects of everyday life from emotional well-being and forming emotional relationships to daily activities, such as having a good night's sleep, leaving the house, or hiding from social events because of the visibility of this condition. Each answer is counted as a specific number of points and the total score reflects the overall impact of the disease on the patient's quality of life. The higher the score, the greater the impact of the disease on the patient's ability to function normally on a daily basis[7,8,9].

There are various therapeutic options for the treatment of psoriasis, which are divided into topical and systemic therapy, depending on the clinical stage of the disease and, of course, any potential comorbidities. Topical therapy is used in the milder forms of the disease, employing preparations that combine corticosteroids with calcipotriol (a vitamin D derivative) or topical corticosteroid formulations with the addition of salicylic acid. In cases where a larger body surface area is affected, which is determined using the previously mentioned tools — BSA and PASI — systemic therapeutic options can be used, such as methotrexate, acitretin, cyclosporine or phototherapy. Other systemic options include biologic drugs such as TNF-alpha inhibitors, IL-17 inhibitors and IL-23 inhibitors, as well as small molecules with immunomodulatory effects, such as apremilast and JAK-TYK inhibitors.

Periodontitis is a chronic inflammatory disease affecting the periodontium that, if left untreated, can lead to tooth loss. The cause of periodontitis is untreated gingivitis, during which bacterial plaque triggers an aberrant immune response[10]. The creation of periodontal pockets, bleeding of the gums and progressive bone loss are typical clinical signs of this disease[11]. Recent classifications divide periodontitis into 4 stages, going from mild to advanced forms of the disease. Poor oral hygiene, smoking, diabetes, stress and genetic predisposition are major risk factors for disease onset and progression. Periodontitis is also linked to numerous systemic diseases, such as rheumatoid arthritis, psoriasis, cardiovascular disease, diabetes and other systemic disorders[12]. Management requires better oral hygiene, a control of the risk factors and non-surgical periodontal treatment. In certain situations, adjuvant therapies such as systemic antibiotics or local antimicrobials may be used. To avoid recurrence, long-term maintenance therapy is necessary. It is possible to effectively control the progression of periodontitis with early detection and consistent care[13].

The co-occurrence of psoriasis and periodontitis has become a major clinical consideration. Patients with psoriasis have significantly higher rates of periodontal disease compared to healthy control populations, according to several epidemiological studies[14]. This connection is particularly relevant, as periodontitis shares mechanistic pathways with several immune-mediated inflammatory disorders; for dermatologists, the most notable one is psoriasis. Understanding the exact immunological pathways of periodontitis, a known aggravator of many systemic inflammatory disorders including psoriasis, is key for better control and treatment of both diseases.

## Methods

We conducted a narrative literature review to identify publications reporting on the epidemiology, pathogenesis, inflammatory mechanisms, therapeutic options, mutual influences and potential triggers for the development of psoriasis and periodontitis, as well as their bidirectional interactions. A comprehensive search of PubMed/MEDLINE, Embase, Web of Science, Scopus and Google Scholar was performed from database inception to November 2025. Search terms included combinations of the following keywords and MeSH terms: “psoriasis”, “periodontitis”, “systemic therapy”, “treatment”, “biologic therapy”, “tumor necrosis factor inhibitors”, “interleukin-17”, “interleukin-23”, “inflammatory loop”, “cytokine network”, “chronic inflammation”, “interleukin-6”, “interleukin-1”, “gingiva” and “periodontal ligament.” Reference lists of relevant review articles and case series were additionally screened to identify further reports. We included case reports, case series, retrospective cohorts, clinical trials and systematic reviews that described clinical features, therapeutic interventions or outcomes, as well as the incidence of each condition in patients with psoriasis and periodontitis. Articles not available in English and publications lacking primary clinical data (e.g., conference abstracts without full text) were excluded.

## Psoriasis – Immunological basis and systemic manifestations

### Basis and systemic inflammatory nature of psoriasis

#### Overview and pathophysiological foundation

Psoriasis is a chronic, immune-mediated inflammatory disease that primarily affects the skin, but can also affect other parts of the human body and act as a systemic inflammation. Its pathophysiological mechanism is still unknown, but the disease itself is characterized by a hyperproliferation of keratinocytes induced by T cells. Other subsequent mechanisms include an altered differentiation of the cells, vascular growth and remodeling, and cell infiltration in the dermis and epidermis[15,16]. Plaques, which are specific clinical signs of psoriasis, are the result of complex pathophysiological mechanisms that include inflammation due to a cytokine and chemokine loop along with specific T cells and other parts of both the adaptive and innate immunity[17]. Psoriatic skin and other parts of the human body are filled with many different cell types, such as keratinocytes, macrophages, dendritic cells and T cells that form self-perpetuating inflammatory loops. Activated immune cells produce cytokines, such as tumor necrosis factor-α (TNF-α), interleukin (IL)-23 and IL-17, which stimulate keratinocyte proliferation and chemokine release and result in recruiting additional immune cells. This feedback loop results in chronic epidermal activation, angiogenesis and tissue remodeling[16,17].

Importantly, this local inflammatory process spills into systemic circulation, contributing to comorbidities such as arthritis, cardiovascular disease and metabolic syndrome[18-21].

## Genetic susceptibility and molecular determinants

The genetic inheritance of psoriasis is associated with loci that have an impact on the immune aspect of the disease. The *HLA-C*06:02* allele remains the strongest risk factor, particularly for early-onset and chronic plaque psoriasis[15,22,23]. Beyond the major histocompatibility complex (MHC), genome-wide association studies (GWAS) have identified over 80 susceptibility loci involving immune-modulatory genes such as *IL23A*, *IL12B*, *IL23R*, *TNFAIP3* and *CARD14*. These converge upon the IL-23/IL-17 signaling axis, highlighting the T helper 17 (Th17) pathway as a central mediator of psoriatic inflammation[23].

## Triggers and lesion initiation

In genetically predisposed individuals, environmental and physiological triggers can initiate lesion formation. Physical trauma, like the Koebner phenomenon, which presents as an initiating skin lesion on previously damaged skin, infections (particularly streptococcal), stress, smoking and certain medications (e.g., β-blockers, lithium) activate innate immune responses[15-17,24].

Keratinocytes play an active role in this initiation phase. Upon injury or infection, they release antimicrobial peptides such as LL-37 and β-defensins. These peptides form complexes with self-DNA and RNA, which activate plasmacytoid dendritic cells (pDCs) through toll-like receptors 7 and 9, leading to type I interferon (α/β) production. The resulting inflammatory group activates myeloid dendritic cells (mDCs), which produce IL-12 and IL-23: important cytokines that drive Th1 and Th17 polarization. When released, IL-23 binds to its receptor, which results in the activation of JAK kinase. This causes the phosphorylation of STAT3 and STAT4 genes, which are then activated and transferred to nuclei, upon which they activate gene replication resulting in the production of other cytokines, such as IL-17, IL-22 and GM-CSF. These cytokines are proinflammatory; their production causes the maintenance of Th17 cells and the activation of other immune cells, such as natural killer cells and innate lymphoid cells, which further contribute to the development of inflammation[15-19,25]. This early immune activation is the main part of the disease because it transforms transient inflammation into a chronic immune process. Recruited neutrophils, macrophages and T cells sustain cytokine release, further enhancing keratinocyte activation and epidermal hyperplasia[15-17,26,27].

## The cytokine network and chronic inflammatory loop

The IL-23/Th17 complex forms the cornerstone of psoriatic inflammation (Fig. 2). IL-23 promotes the differentiation and maintenance of Th17 cells, which secrete IL-17A, IL-17F and IL-22. These cytokines act synergistically to amplify keratinocyte proliferation and suppress terminal differentiation, generating the characteristic psoriatic phenotype of a thickened epidermis and parakeratosis[28,29]. Keratinocytes respond to IL-17 and TNF-α by producing additional inflammatory mediators, including IL-1β, IL-6, IL-36, CXCL1 and CCL20. These attract neutrophils, dendritic cells and T-cells, thereby sustaining inflammation. IL-36 in particular has emerged as an important amplifier of local inflammation, particularly in

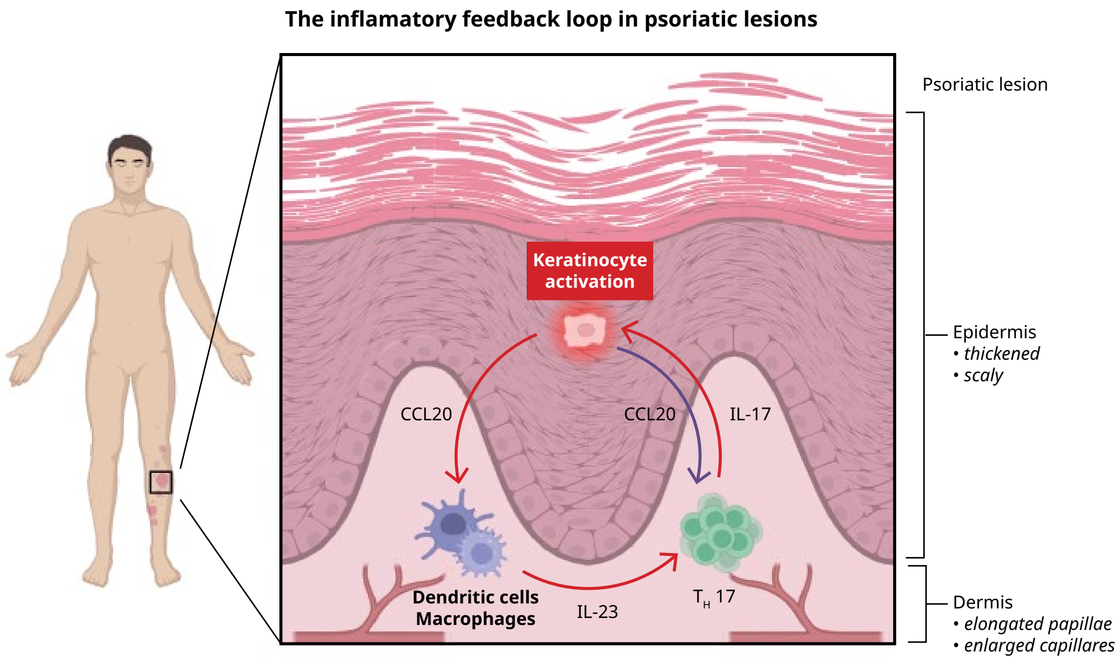


**FIG. 2.** The IL-23/IL-17 Axis in Psoriasis, created in BioRender.

pustular and severe plaque psoriasis[29,30]. TNF-α acts as a master regulator, bridging innate and adaptive immunity and potentiating IL-17–mediated effects[29]. In addition to the Th17 pathway, the Th1 axis also contributes through IFN-γ and IL-12 signaling, promoting macrophage activation and vascular changes. Together, these cytokine networks create a redundant, self-reinforcing system that makes psoriasis a prototypical example of chronic, cytokine-driven inflammation[15-17,19,25,28].

## Cellular crosstalk: From keratinocytes to the endothelium

Keratinocytes are not only passive targets; they also contribute to inflammation mechanisms. They express pattern-recognition receptors (TLRs, NODs), which detect microbial or damage-associated molecular patterns and result in the production of cytokines and chemokines that shape immune cell recruitment[31]. Endothelial cells also undergo inflammatory activation. Vascular endothelial growth factor (VEGF) and angiopoietin signaling promote neovascularization within lesions, while adhesion molecules, such as ICAM-1 and VCAM-1, facilitate leukocyte trafficking. This endothelial activation reflects systemic vascular inflammation, which parallels the increased cardiovascular risk in psoriasis[29,31,32]. Recent studies revealed metabolic reprogramming in both the immune system and skin in favor of cell glycolysis and lipid metabolism that maintain inflammation. This metabolic-immune interface links local inflammation to systemic metabolic alterations[29,32].

## Histopathological and structural changes

Histologically, psoriatic plaques show acanthosis (epidermal thickening), an elongation of the rete ridges, a thinning of the suprapapillary plates, dilated dermal capillaries and the formation of Munro microabscesses or aggregates of neutrophils within the stratum corneum. The combination of keratinocyte hyperproliferation, immune infiltration and vascular remodeling accounts for the erythematous, scaly plaques typical of the disease[15-17]. These structural alterations are not confined to the skin. Chronic low-grade systemic inflammation has been observed in psoriatic patients with elevated circulating levels of CRP, IL-6, TNF-α and IL-17, correlating with disease severity and comorbidities[15-18].

### Systemic inflammatory manifestations and clinical implications

#### Psoriatic arthritis and musculoskeletal inflammation

Approximately 30% of individuals with psoriasis develop psoriatic arthritis (PsA), characterized by synovial inflammation, enthesitis and bone remodeling. The IL-23/IL-17 axis underlies both skin and joint inflammation, with a tissue-specific expression of IL-17A and IL-17F by T cells and innate lymphoid cells[15-17,19,25]. Targeting this pathway has proven effective clinically, reinforcing its central pathogenic role.

#### Cardiometabolic comorbidities and endothelial dysfunction

Systemic inflammation extends beyond the skin, influencing the cardiovascular and metabolic systems. Chronic cytokine exposure promotes endothelial dysfunction, insulin resistance and atherosclerotic plaque formation[21,33]. Psoriasis patients exhibit higher incidences of myocardial infarction, stroke, obesity and metabolic syndrome compared to the general population. IL-17 and TNF-α drive vascular inflammation, while adipokines such as leptin and resistin further exacerbate the metabolic imbalance[18,20,21,33].

#### Neuroimmune and psychosocial dimensions

Psoriasis is connected to a higher prevalence of depression and anxiety due to neuroimmune dysregulation. Higher levels of IL-6 and TNF-α in cerebrospinal fluid and altered hypothalamic–pituitary–adrenal axis function can progress mood disorders. Overall, chronic inflammatory load and visible skin changes on typical locations also exacerbate psychological stress and create another loop of systemic inflammation, which contributes to the condition[34].

## Periodontitis — pathogenesis and systemic associations

### Overview and pathophysiological foundation in periodontitis

Periodontitis is a chronic multifactorial inflammatory disease that results in the progressive destruction of tooth-supporting structures, including the gingiva, periodontal ligament and alveolar bone (Fig. 3)[35].

The prevalence of periodontitis in the population increases with age; around 40% of Europeans between ages 65 and 74 are affected by this condition. The frequency of the disease itself is demonstrated by the fact that it was the 6$^{th}$ most common disease in the world in 2016[36]. Inflammation typically begins as reversible gingivitis. The process begins with the buildup of a complex bacterial biofilm (dental plaque) on the tooth surface, containing pathogens like *Porphyromonas gingivalis, Tannerella forsythia* and

**FIG. 3.** Periodontitis

*Aggregatibacter actinomycetemcomitans*. When the body's inflammatory response fails to remain controlled, this can lead to the development of destructive periodontitis[37].

The diagnosis and staging of periodontitis rely on identifying irreversible clinical and radiographic signs that reflect tissue destruction caused by the host's inflammatory response to the bacterial infection. As the disease progresses, the epithelial attachment migrates down the tooth's root, forming periodontal pockets that harbor pathogenic bacteria and sustain chronic inflammation. Clinical attachment loss (CAL) serves as the primary diagnostic indicator, measuring the extent of irreversible connective tissue damage. The most advanced stage involves severe alveolar bone loss, which leads to tooth mobility and eventual tooth loss[38].

Periodontitis is in fact a complex interplay between a dysbiotic microbiome, particularly one including pathogens such as the Gram-negative anaerobic bacterium *Porphyromonas gingivalis* and aberrant immune responses occurring around gingival and periodontal tissues. *Porphyromonas gingivalis* is a so-called keystone pathogen in the development of periodontitis. Although it exists in small amounts within the subgingival biofilm, it has a strong impact both on other bacteria and on the host's immune system[39].

*P. gingivalis* disrupts the balance of healthy oral microbes, leading to dysbiosis and chronic inflammation by interfering with Toll-like receptors (TLRs) and complement interactions[40]. By weakening immune defenses and promoting tissue breakdown, it allows the inflammation to persist and progress. This explains why PD can continue and cause bone destruction even without large amounts of bacteria; the progression of the disease is fueled by the strong influence of this keystone pathogen on the host's immune system[41]. Although microorganisms act as initiators, the pathogenesis itself is primarily mediated by an uncontrolled and prolonged host immune response. This leads to an overproduction of inflammatory mediators, such as IL-1β, IL-6, TNF-α and IL-17, which stimulate osteoclastic activity and cause irreversible tissue and bone destruction[37].

It is undeniable that periodontitis is not just a localized oral disease, but a key risk factor for the development and progression of numerous chronic systemic diseases. Epidemiological studies have strongly linked PD to cardiovascular disease (cerebrovascular disease, atherosclerosis and hypertension), diabetes, rheumatoid arthritis, pulmonary diseases such as chronic obstructive pulmonary disease, neurodegenerative conditions (Alzheimer's disease) and adverse pregnancy outcomes[42,43]. Chronic inflammation from periodontal disease can influence overall systemic health, while existing systemic disorders may, in turn, exacerbate the progression and severity of periodontal disease[44].

## The cytokine network and chronic inflammatory loop in periodontitis

Macrophages are important immune cells that play a crucial role in the development and progression of periodontitis. Because of their exceptional plasticity, they can change their functional state between inflammatory and proinflammatory phenotypes, based on the conditions inside periodontal tissue. In a healthy periodontium, two main phenotypes — the pro-inflammatory (M1) phenotype and the anti-inflammatory (M2) phenotype — are in balance. However, in periodontitis, this balance is disturbed and M1 macrophages become more prevalent. When bacterial products and inflammatory mediators activate M1 macrophages, cytokines such as TNF-α, IL-6, and iNOS (nitric oxide synthase) are produced. These mediators intensify inflammation and play a key role in tissue destruction. In contrast, M2 macrophages are crucial for resolving inflammation and promoting tissue healing because they express markers like CD206, arginase-1 and IL-10[45].

When periodontal infection becomes chronic, the local environment sustains the ongoing M1 activation while inhibiting M2 development. This imbalance intensifies inflammation, leading to the breakdown of the periodontal ligament and the loss of alveolar bone. Studies have demonstrated that in comparison to healthy sites, the M1/M2 macrophage ratio is markedly elevated in diseased periodontal tissue. Furthermore, there is a strong correlation between the extent of this imbalance and the degree of periodontal destruction[46,47,48].

The imbalance between two important cell populations — Th17 cells and regulatory T cells (Tregs) — plays a significant role in the development and severity of periodontitis. Th12 cells encourage inflammation, whereas Tregs inhibit it to preserve immunological homeostasis. These two subsets play opposing roles. By generating anti-inflammatory cytokines like TGF-β and IL-10, Tregs try to maintain immune homeostasis and control inflammation. Nevertheless, the body's capacity to control inflammation is weakened in periodontitis due to a marked decrease in Treg activity and quantity. At the same time, the gingival tissues of those who are affected have a noticeable increase in Th17 cells[49].

Alveolar bone loss results from cytokine IL-17, which is secreted by Th17 cells and increased inflammation and bone-resorbing osteoclasts. Many factors contribute to the overactivation of Th17 cells. Complement component C3a, for instance, can stimulate dendritic cells to release important cytokines that support Th17 differentiation, such as IL-6 and IL-23. Gingipains and other virulence factors from *Porphyromonas gingivalis* also promote Th17 polarization via the IL-6-dependent mechanism[50].

Th17 cells and macrophages work together in a feedback loop that exacerbates periodontal inflammation. Cytokines like IL-1, IL-6 and IL-23 are released by activated macrophages and aid in the differentiation of Th17 cells from naive CD4+ T cells. Th17 cells then release IL-17A, which in turn triggers the production of proinflammatory mediators like TNF-α, IL-β and IL-6 by macrophages. Within periodontal tissues, this reciprocal activation maintains a persistent inflammatory state. Alveolar bone loss and chronic tissue damage result from the constant interaction between these two cell types. In the end, the macrophage Th17 axis is a major mechanism that determines the severity and chronicity of periodontitis[51,52].

## Shared pathophysiological mechanisms

Both psoriasis and periodontal disease are chronic inflammatory diseases triggered by dysregulated immune responses and dysbiotic microbiota. Although the cellular sources and tissue targets are different, the pathophysiological mechanisms of both conditions involve similar immune signaling pathways, including pro-inflammatory mediators that include IL-1β, TNF-α, IL-6, IL-17 and IL-23[51].

In both periodontal and cutaneous inflammation, IL-17 is a prime example of the delicate interaction between pathological and protective immunity. This cytokine has two functions: by recruiting neutrophils, producing antimicrobial peptides and maintaining the integrity of the mucosal barrier, it improves host defense; however, excessive IL-17 expression causes chronic inflammation, accelerates tissue destruction and leads to alveolar bone loss in periodontitis[48].

While increased IL-17 in psoriatic skin encourages epidermal acanthosis and angiogenesis, IL-17-producing cells in periodontal tissues trigger osteoclast activation and periodontal ligament degradation. Given the overlap of the IL-17-mediated pathology in both tissues, it is possible that a fundamental mechanism underlying both conditions is a dysregulation of IL-17 signaling, which might be responsible for the higher susceptibility of people with periodontitis to systemic inflammatory diseases that share this immunological signature[49,53-55].

A crucial molecular link between systemic autoimmune reactions and periodontal inflammation is provided by neutrophil extracellular traps (NETs). Activated neutrophils release NETs, which are fibrous structures that act as self-antigens and can cause inflammatory cascades by triggering the complement and inflammasome systems[53,56].

This can disrupt self-tolerance mechanisms and speed up autoimmune responses. Exaggerated inflammatory periodontal lesions are the pathological changes caused by excessive neutrophil activation and NET release in periodontitis.

Through the circulation of self-antigens derived from NETs, as well as modifications in systemic immune tolerance, the dysregulation of neutrophil responses and NET formation in periodontal tissues may theoretically spread autoimmune responses that affect distant sites, including the skin. This mechanism offers a plausible explanation for why people with severe periodontitis are more susceptible to autoimmune diseases like psoriasis, which are characterized by corresponding immune dysregulation patterns[57,58].

By altering gut microbiota and compromising the integrity of the intestinal barrier (the "oral-gut axis"), pathogenic periodontal bacteria and associated byproducts can increase intestinal permeability and facilitate bacterial translocation. When periodontal pathogens and their metabolic byproducts interfere with the function of the intestinal barrier, more bacteria may circulate throughout, which increases the stimulation of pattern recognition receptors on intestinal dendritic cells and encourages the differentiation of proinflammatory Th17 cells: the same cell population implicated in the pathophysiology of psoriasis. In susceptible populations, periodontitis may be a risk factor for the onset or flare-up of psoriatic disease due to this pathophysiological pathway, which demonstrates how localized periodontal disease produces systemic immunological effects that can cause or exacerbate distant inflammatory diseases[59,60].

## Clinical and epidemiological correlations

Emerging evidence suggests a bidirectional relationship between psoriasis and periodontitis, both of which are inflammatory conditions, with epidemiological studies consistently indicating a higher prevalence of periodontitis among individuals with psoriasis and vice versa. A number of overlapping elements, such as similar immunological pathways involving the IL-17/Th17 axis and shared lifestyle and metabolic risk variables (smoking, obesity, diabetes), are probably responsible for the link between the two diseases. Additionally, these circumstances may be reinforced by reciprocal biological interactions. From a clinical perspective, this emphasizes the significance of periodontal evaluation in people with psoriasis, and dentists should also be aware of the elevated risk of psoriasis among individuals with periodontitis[61].

Epidemiological evidence indicates that patients with psoriasis have a significantly higher prevalence of chronic periodontitis compared to individuals without psoriasis. For example, a case-control study of 397 psoriasis patients and 359 controls found that 46.1% of the psoriasis group had periodontitis versus 33.1% of the controls[6].

A recent meta-analysis (16 studies) concluded that psoriasis patients showed more than a twofold increase in the odds for periodontal disease compared to non-psoriatic individuals[14].

In a different study with 152 psoriasis vulgaris patients, the prevalence of apical periodontitis was significantly higher in those with moderate or severe psoriasis than in those with mild psoriasis ($P = 0.002$). The presence of apical periodontitis and the severity of psoriasis were substantially correlated, indicating that psoriasis may be a key factor in the pathophysiology of this periodontal disease[63].

The adjusted hazard ratios for periodontal disease were significantly higher (1.108; 95% CI: 1.088–1.129) in a large nationwide population-based cohort study conducted in Korea that included 15,165 psoriasis patients and 75,825 age- and sex-matched controls. Similar trends were noted for dental caries and pulp and periapical disease. Significantly, the elevated risk of dental comorbidities was reduced in patients who received systemic anti-psoriatic treatment, indicating that good psoriasis management may reduce the related oral complications[64].

Growing evidence suggests a significant correlation between the severity of psoriasis as measured by PASI and the severity of periodontal disease, indicating a potential systemic inflammatory link between these two chronic conditions. Psoriasis patients frequently exhibit greater periodontal attachment loss, probing depth and inflammation compared to healthy controls. Moreover, the severity of periodontitis correlates positively with psoriasis activity, as measured by PASI[65].

Randomized clinical trials have shown that nonsurgical periodontal therapy results in statistically significant reductions in PASI scores when compared to control groups, indicating that treating periodontitis has produced tangible reductions in the severity of psoriasis. This therapeutic effect highlights how the relationship is reciprocal: lowering the oral inflammatory burden directly improves the symptoms of skin diseases. On the other hand, patients with severe psoriasis exhibit noticeably higher rates and severity of periodontal disease, indicating that systemic psoriatic inflammation promotes an immune environment that supports the development of periodontal disease[66].

## The impact of psoriasis therapy on periodontal health

Several recent reviews and scoping studies showed that systemic biologic agents used for immune-mediated diseases, including psoriasis, are combined with a modulation and, in many cases, reduction of local periodontal inflammation, probably due to down-regulation of systemic and mucosal pro-inflammatory cytokines. Clinical and observational reports included in a recent scoping review found that biologics are able to lower clinical periodontal indices and inflammatory markers, but effects vary between agents and studies, and may depend on baseline periodontal status and concurrent risk factors. These papers therefore suggest a potential beneficial effect of IL-17, IL-23 and anti-TNF therapies on periodontal inflammation, while also cautioning that high-quality randomized trials, specifically in psoriasis patients, are still limited[67].

Psoriatic patients are very often treated with immunosuppression. Methotrexate, as one such drug, has various effects on other organs as well as on oral tissue. It reduces inflammation, which is a wanted effect due to periodontitis. The negative aspect is lowering the ability to restore oral mucosa or wound healing. Systemic corticosteroids are known as strong medications due to their profound anti-inflammatory effects. The risks of taking systemic corticosteroids are very well known, as they cause a number of side effects, especially in the oral cavity area. Candidiasis, or wound healing, can postpone periodontal treatment and therapy. Including all of the reasons mentioned above, the literature emphasizes the importance of an individualized approach. Both dermatologists and dentists — and rheumatologists, who are sometimes included, — should be coordinated while patients are receiving long-term immunosuppressive medications[68].

Local therapy and daily oral hygiene have a profound influence on the regulation of periodontitis. Procedures that include local therapy, such as plaque removal, root planning and the removal of local irritants, simultaneously lead to a reduction in local inflammation. Clinical periodontal parameters have been shown to improve and some studies have shown improvements in inflammatory values and psoriasis status assessment indices. A systematic review mapping the evidence called for a minimum dental care protocol to soothe the impact of periodontal disease on chronic psoriasis plaques, stressing that good oral hygiene remains a low-risk, high-value strategy for patients with psoriasis[69].

Studies have shown that biologic treatment used in other immune-mediated diseases caused changes in oral cavity immunity measured by salivary immune parameters. Biologic agents are known to change not only systemic immune responses but also mucosal immune responses. Check-ups of already existing inflammatory points (periodontitis, dental infections) before and during treatment with biologic agents are recommended. Finally, large observational and case-control studies report that there is a greater possibility of severe periodontitis among psoriasis patients (for example, an increased possibility ratio of severe periodontal disease reported in some series) and that considerable smoking amplifies that risk, underlining the need for integrated periodontal care and smoking discontinuation interventions in this population[70].

## Multidisciplinary approach and clinical recommendations

A timely recognition and confirmation of the diagnosis are key. Dermatologists should also consider looking for different signs of oral cavity disease in psoriatic patients. Also, dentists, especially periodontists, have to be aware of their patients' anamneses and take more care to notice whether some of their patients have psoriasis[14,71]. Shared care (including dermatologists and dentists) makes it possible to recognize early signs of systemic inflammation, such as rising cytokine levels of TNF-α, IL-17 and IL-6, which are included in both psoriasis and periodontitis[72].

Coordinating the treatments of these conditions and an integrated follow-up, which recommends screening for periodontal diseases with every psoriatic patient, is needed to get good therapy results and prevent possible side effects or complications[73,74].

Platforms that connect dental teams and specialists — including both dermatologists and rheumatologists — along with primary care doctors, are crucial to get the best out of following and monitoring patients on specific medications and also to minimize potential side effects, or to recognize early signs of progression of both diseases. This approach includes medical reports, physical status information, the status of inflammation, responses to medication and potential detection of any side effects[65,75].

Therefore, it is crucial to include regular dental checkups while patients are receiving biologic or immunosuppressive therapy, because it raises the odds of an earlier detection of certain conditions and their earlier treatment to reduce complications due to shared inflammatory pathways[76]. A multidisciplinary approach is the key to getting good therapy results and bringing these diseases under control. Patients should be educated about the connections between psoriasis and periodontitis and made aware of the importance of good oral hygiene and regular dental checkups[65,72,73].

## Conclusion

Seemingly two completely unrelated diseases — psoriasis, which is a multisystem disease primarily affecting the skin, and periodontitis, which represents a distinct group of clinical oral-health conditions — clearly demonstrate the presence of shared inflammatory mechanisms that alternately exert their effects by interacting with one another across both conditions[53,71,74]. Although an association between these conditions has been established, its causality has not yet been confirmed[53,77]. Despite similarities in immunological and pathophysiological mechanisms (such as elevated levels of inflammatory cytokines IL-17 and TNF-alpha), further research is needed to identify potential predictors in the form of oral clinical conditions for skin diseases and vice versa. It is important to emphasize that this may only be the beginning of new dimensions in immunology, encompassing not only systemic inflammation and skin changes but also inflammatory conditions of the oral mucosa[53,72,74,77]. ■

## References

1. Bechet PE. Psoriasis: a brief historical review. Arch Dermatol Syphilol. 1936 Feb;33(2):327–334. doi:10.1001/archderm.1936.01470080123009.
2. Greb JE, Goldminz AM, Elder JT, Lebwohl MG, Gladman DD, Wu JJ, Mehta NN, Finlay AY, Gottlieb AB. Psoriasis. Nat Rev Dis Primers. 2016 Nov 24;2:16082. doi: 10.1038/nrdp.2016.82. PMID: 27883001.
3. Kimmel GW, Lebwohl M. Psoriasis: Overview and Diagnosis. Evidence-Based Psoriasis. 2018 Jul 1:1–16. doi: 10.1007/978-3-319-90107-7_1. PMCID: PMC7122924.

4. Ferreli C, Pinna AL, Pilloni L, Tomasini CF, Rongioletti F. Histopathological aspects of psoriasis and its uncommon variants. G Ital Dermatol Venereol. 2018 Apr;153(2):173-184. doi: 10.23736/S0392-0488.17.05839-4. Epub 2017 Dec 15. PMID: 29249124.
5. Merola JF, Amato DA, See K, Burge R, Mallinckrodt C, Ojeh CK, Gottlieb A. Evaluation of sPGA × BSA as an Outcome Measure and Treatment Target for Clinical Practice. J Invest Dermatol. 2018 Sep;138(9):1955-1961. doi: 10.1016/j.jid.2018.01.041. Epub 2018 Mar 22. PMID: 29577919.
6. Goff KL, Karimkhani C, Boyers LN, Weinstock MA, Lott JP, Hay RJ, Coffeng LE, Norton SA, Naldi L, Dunnick C, Armstrong AW, Dellavalle RP. The global burden of psoriatic skin disease. Br J Dermatol. 2015 Jun;172(6):1665-1668. doi: 10.1111/bjd.13715. Epub 2015 May 6. PMID: 25645671.
7. Liang Y, Epipode N, Deng L, Zhang Z, Wang F, Dang L, Li Z, Zhang J, Yi H, Zhang H, Yang S, Yan J, Gan W, Duan D, Deng B. Epidemiological Profile of Psoriasis and Linked Comorbidities in the Chinese Population at Shenzhen: A Cross-Sectional Study. Health Sci Rep. 2025 Sep 16;8(9):e71159. doi: 10.1002/hsr2.71159. PMID: 40970104; PMCID: PMC12441203.
8. Laskowski M, Schiöler L, Wennberg AM, Torén K, Gustafsson H. Translation and Validation of the Self-Assessment Psoriasis Area Severity Index. Dermatology. 2023;239(5):794-801. doi: 10.1159/000530045. Epub 2023 Mar 10. PMID: 36907178; PMCID: PMC10614249.
9. Finlay AY, Basra MKA, Piguet V, Salek MS. Dermatology life quality index (DLQI): a paradigm shift to patient-centered outcomes. J Invest Dermatol. 2012 Oct;132(10):2464-2465. doi: 10.1038/jid.2012.147. Epub 2012 May 17. PMID: 22592153.
10. Kwon T, Lamster IB, Levin L. Current Concepts in the Management of Periodontitis. Int Dent J. 2021 Dec;71(6):462-476. doi: 10.1111/idj.12630. Epub 2021 Feb 19. PMID: 34839889; PMCID: PMC9275292.
11. Page RC. The etiology and pathogenesis of periodontitis. Compend Contin Educ Dent. 2002 May;23(5 Suppl):11-4. PMID: 12789963.
12. Albandar JM. Epidemiology and risk factors of periodontal diseases. Dent Clin North Am. 2005 Jul;49(3):517-32, v-vi. doi: 10.1016/j.cden.2005.03.003. PMID: 15978239.
13. Kumar S. Evidence-Based Update on Diagnosis and Management of Gingivitis and Periodontitis. Dent Clin North Am. 2019 Jan;63(1):69-81. doi: 10.1016/j.cden.2018.08.005. Epub 2018 Oct 29. PMID: 30447793.
14. Nijakowski K, Gruszczyński D, Kolasińska J, Kopała D, Surdacka A. Periodontal Disease in Patients with Psoriasis: A Systematic Review. Int J Environ Res Public Health. 2022 Sep 8;19(18):11302. doi: 10.3390/ijerph191811302. PMID: 36141573; PMCID: PMC9516998.
15. Armstrong AW, Blauvelt A, Callis Duffin K, et al. Psoriasis. Nat Rev Dis Primers. 2025;11:45. doi: 10.1038/s41572-025-00630-5.
16. Man AM, Orăsan MS, Hoteiuc OA, Olănescu-Vaida-Voevod MC, Mocan T. Inflammation and psoriasis: a comprehensive review. Int J Mol Sci. 2023 Nov 16;24(22):16095. doi: 10.3390/ijms242216095.
17. Johnson-Huang LM, Lowes MA, Krueger JG. Putting together the psoriasis puzzle: an update on developing targeted therapies. Dis Model Mech. 2012 Jul;5(4):423-33. doi: 10.1242/dmm.009092. PMID: 22730473; PMCID: PMC3380706.
18. Kovitwanichkanont T, Chong AH, Foley P. Beyond skin deep: addressing comorbidities in psoriasis. Med J Aust. 2020 Jun;212(11):528-534. doi: 10.5694/mja2.50591. Epub 2020 May 10. PMID: 32388913.
19. Azuaga AB, Ramírez J, Cañete JD. Psoriatic Arthritis: Pathogenesis and Targeted Therapies. Int J Mol Sci. 2023 Mar 3;24(5):4901. doi: 10.3390/ijms24054901. PMID: 36902329; PMCID: PMC10003101.
20. Kunz M, Simon JC, Saalbach A. Psoriasis: Obesity and Fatty Acids. Front Immunol. 2019 Jul 31; 10:1807. doi: 10.3389/fimmu.2019.01807. PMID: 31417571; PMCID: PMC6684944.
21. Manolis AA, Manolis TA, Melita H, Manolis AS. Psoriasis and cardiovascular disease: the elusive link. Int Rev Immunol. 2019;38(1):33-54. doi: 10.1080/08830185.2018.1539084. Epub 2018 Nov 20. PMID: 30457023.
22. Mateu-Arrom L, Puig L. Genetic and Epigenetic Mechanisms of Psoriasis. Genes (Basel). 2023 Aug 13;14(8):1619. doi: 10.3390/genes14081619. PMID: 37628670; PMCID: PMC10454222.
23. Dand N, Stuart PE, Bowes J, Ellinghaus D, Nititham J, Saklatvala JR, Teder-Laving M, Thomas LF, Traks T, Uebe S, Assmann G, Baudry D, Behrens F, Billi AC, Brown MA, Burkhardt H, Capon F, Chung R, Curtis CJ, Duckworth M, Ellinghaus E, FitzGerald

O, Gerdes S, Griffiths CEM, Gulliver S, Helliwell P, Ho P, Hoffmann P, Holmen OL, Huang ZM, Hveem K, Jadon D, Köhm M, Kraus C, Lamacchia C, Lee SH, Ma F, Mahil SK, McHugh N, McManus R, Modalsli EH, Nissen MJ, Nöthen M, Oji V, Oksenberg JR, Patrick MT, Perez-White BE, Ramming A, Rech J, Rosen C, Sarkar MK, Schett G, Schmidt B, Tejasvi T, Traupe H, Voorhees JJ, Wacker EM, Warren RB, Wasikowski R, Weidinger S, Wen X, Zhang Z; BSTOP study group; Estonian Biobank research team; Barton A, Chandran V, Esko T, Foerster J, Franke A, Gladman DD, Gudjonsson JE, Gulliver W, Hüffmeier U, Kingo K, Kõks S, Liao W, Løset M, Mägi R, Nair RP, Rahman P, Reis A, Smith CH, Di Meglio P, Barker JN, Tsoi LC, Simpson MA, Elder JT. GWAS meta-analysis of psoriasis identifies new susceptibility alleles impacting disease mechanisms and therapeutic targets. medRxiv [Preprint]. 2023 Oct 5:2023.10.04.23296543. doi: 10.1101/2023.10.04.23296543. Update in: Nat Commun. 2025 Feb 28;16(1):2051. doi: 10.1038/s41467-025-56719-8. PMID: 37873414; PMCID: PMC10593001.

24. Schmutz JL. Médicaments inducteurs de psoriasis : actualités [Psoriasis-inducing drugs: An update]. Ann Dermatol Venereol. 2019 Mar;146(3):260-261. French. doi: 10.1016/j.annder.2019.01.002. Epub 2019 Mar 2. PMID: 30833036.
25. Wu R, Zeng J, Yuan J, Deng X, Huang Y, Chen L, Zhang P, Feng H, Liu Z, Wang Z, Gao X, Wu H, Wang H, Su Y, Zhao M, Lu Q. MicroRNA-210 overexpression promotes psoriasis-like inflammation by inducing Th1 and Th17 cell differentiation. J Clin Invest. 2018 Jun 1;128(6):2551-2568. doi: 10.1172/JCI97426. Epub 2018 May 14. PMID: 29757188; PMCID: PMC5983326.
26. Gonçalves MBS, Fonseca M, Mascarenhas-Melo F, Figueiras A. Advancing insights into psoriasis: from pathogenesis to current and emerging therapies. *Int Immunopharmacol.* 2025 Nov 14;165:115429. doi:
27. Raharja A, Mahil SK, Barker JN. Psoriasis: a brief overview. Clin Med (Lond). 2021 May;21(3):170-173. doi: 10.7861/clinmed.2021-0257. PMID: 34001566; PMCID: PMC8140694.
28. Kim J, Lee J, Lee J, Kim K, Li X, Zhou W, Cao J, Krueger JG. Psoriasis harbors multiple pathogenic type 17 T-cell subsets: Selective modulation by risankizumab. J Allergy Clin Immunol. 2025 Jun;155(6):1898-1912. doi: 10.1016/j.jaci.2025.02.008. Epub 2025 Feb 18. PMID: 39978685; PMCID: PMC12145251.
29. Brembilla NC, Senra L, Boehncke WH. The IL-17 Family of Cytokines in Psoriasis: IL-17A and Beyond. Front Immunol. 2018 Aug 2;9:1682. doi: 10.3389/fimmu.2018.01682. PMID: 30127781; PMCID: PMC6088173.
30. Marrakchi S, Puig L. Pathophysiology of Generalized Pustular Psoriasis. Am J Clin Dermatol. 2022 Jan;23(Suppl 1):13-19. doi: 10.1007/s40257-021-00655-y. Epub 2022 Jan 21. PMID: 35061228; PMCID: PMC8801405.
31. Sieminska I, Pieniawska M, Grzywa TM. The Immunology of Psoriasis-Current Concepts in Pathogenesis. Clin Rev Allergy Immunol. 2024 Apr;66(2):164-191. doi: 10.1007/s12016-024-08991-7. Epub 2024 Apr 20. PMID: 38642273; PMCID: PMC11193704.
32. Zhou X, Chen Y, Cui L, Shi Y, Guo C. Advances in the pathogenesis of psoriasis: from keratinocyte perspective. Cell Death Dis. 2022 Jan 24;13(1):81. doi: 10.1038/s41419-022-04523-3. PMID: 35075118; PMCID: PMC8786887.
33. Zhang L, Wang Y, Qiu L, Wu J. Psoriasis and cardiovascular disease risk in European and East Asian populations: evidence from meta-analysis and Mendelian randomization analysis. BMC Med. 2022 Nov 1;20(1):421. doi: 10.1186/s12916-022-02617-5. PMID: 36320003; PMCID: PMC9628092.
34. Patel N, Nadkarni A, Cardwell LA, Vera N, Frey C, Patel N, Feldman SR. Psoriasis, Depression, and Inflammatory Overlap: A Review. Am J Clin Dermatol. 2017 Oct;18(5):613-620. doi: 10.1007/s40257-017-0279-8. PMID: 28432649.
35. Łasica A, Golec P, Laskus A, Zalewska M, Gędaj M, Popowska M. Periodontitis: etiology, conventional treatments, and emerging bacteriophage and predatory bacteria therapies. Front Microbiol. 2024 Sep 26;15:1469414. doi: 10.3389/fmicb.2024.1469414. PMID: 39391608; PMCID: PMC11464445.
36. GBD 2016 Disease and Injury Incidence and Prevalence Collaborators. Global, regional, and national incidence, prevalence, and years lived with disability for 328 diseases and injuries for 195 countries, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. Lancet. 2017 Sep 16;390(10100):1211-1259. doi:

10.1016/S0140-6736(17)32154-2. Erratum in: Lancet. 2017 Oct 28;390(10106):e38. doi: 10.1016/S0140-6736(17)32647-8. PMID: 28919117; PMCID: PMC5605509.
37. Gasmi Benahmed A, Kumar Mujawdiya P, Noor S, Gasmi A. *Porphyromonas Gingivalis* in the Development of Periodontitis: Impact on Dysbiosis and Inflammation. Arch Razi Inst. 2022 Oct 31;77(5):1539-1551. doi: 10.22092/ARI.2021.356596.1875. PMID: 37123122; PMCID: PMC10133641.
38. Mendonça CD, Mata ADSPD, Azevedo LFR, Marques JF, Silveira JML, Marques DNDS. Probiotics in the non-surgical treatment of periodontitis: a systematic review and network meta-analysis. BMC Oral Health. 2024 Oct 15;24(1):1224. doi: 10.1186/s12903-024-05027-6. PMID: 39407177; PMCID: PMC11481756.
39. Sedghi LM, Bacino M, Kapila YL. Periodontal Disease: The Good, the Bad, and the Unknown. Front Cell Infect Microbiol. 2021 Dec 7;11:766944. doi: 10.3389/fcimb.2021.766944. PMID: 34950607; PMCID: PMC8688827
40. Kawai T, Akira S. The role of pattern-recognition receptors in innate immunity: update on Toll-like receptors. Nat Immunol. 2010 May;11(5):373-84. doi: 10.1038/ni.1863. Epub 2010 Apr 20. PMID: 20404851.
41. Xu W, Zhou W, Wang H, Liang S. Roles of Porphyromonas gingivalis and its virulence factors in periodontitis. Adv Protein Chem Struct Biol. 2020;120:45-84. doi: 10.1016/bs.apcsb.2019.12.001. Epub 2020 Jan 10. PMID: 32085888; PMCID: PMC8204362.
42. Hasan F, Tandon A, AlQallaf H, John V, Sinha M, Gibson MP. Inflammatory Association between Periodontal Disease and Systemic Health. Inflammation. 2025 Aug 6. doi: 10.1007/s10753-025-02317-1. Epub ahead of print. PMID: 40768114.
43. Villoria GEM, Fischer RG, Tinoco EMB, Meyle J, Loos BG. Periodontal disease: A systemic condition. Periodontol 2000. 2024 Oct;96(1):7-19. doi: 10.1111/prd.12616. Epub 2024 Nov 4. PMID: 39494478; PMCID: PMC11579822.
44. Tattar R, da Costa BDC, Neves VCM. The interrelationship between periodontal disease and systemic health. Br Dent J. 2025 Jul;239(2):103-108. doi: 10.1038/s41415-025-8642-2. Epub 2025 Jul 25. PMID: 40715391; PMCID: PMC12296551.
45. Almubarak A, Tanagala KKK, Papapanou PN, Lalla E, Momen-Heravi F. Disruption of Monocyte and Macrophage Homeostasis in Periodontitis. Front Immunol. 2020 Feb 26;11:330. doi: 10.3389/fimmu.2020.00330. PMID: 32210958; PMCID: PMC7067288
46. Mills CD. M1 and M2 Macrophages: Oracles of Health and Disease. Crit Rev Immunol. 2012;32(6):463-88. doi: 10.1615/critrevimmunol.v32.i6.10. PMID: 23428224.
47. Italiani P, Boraschi D. From Monocytes to M1/M2 Macrophages: Phenotypical vs. Functional Differentiation. Front Immunol. 2014 Oct 17;5:514. doi: 10.3389/fimmu.2014.00514. PMID: 25368618; PMCID: PMC4201108.
48. Yin L, Li X, Hou J. Macrophages in periodontitis: A dynamic shift between tissue destruction and repair. Jpn Dent Sci Rev. 2022 Nov;58:336-347. doi: 10.1016/j.jdsr.2022.10.002. Epub 2022 Oct 28. PMID: 36340583; PMCID: PMC9630779.
49. Fan X, Shu P, Wang Y, Ji N, Zhang D. Interactions between neutrophils and T-helper 17 cells. Front Immunol. 2023 Oct 18;14:1279837. doi: 10.3389/fimmu.2023.1279837. PMID: 37920459; PMCID: PMC10619153.
50. Wang Y, Xue N, Wang Z, Zeng X, Ji N, Chen Q. Targeting Th17 cells: a promising strategy to treat oral mucosal inflammatory diseases. Front Immunol. 2023 Jul 26;14:1236856. doi: 10.3389/fimmu.2023.1236856. PMID: 37564654; PMCID: PMC10410157.
51. Huang N, Dong H, Luo Y, Shao B. Th17 Cells in Periodontitis and Its Regulation by A20. Front Immunol. 2021 Sep 7;12:742925. doi: 10.3389/fimmu.2021.742925. PMID: 34557201; PMCID: PMC8453085.
52. Bunte K, Beikler T. Th17 Cells and the IL-23/IL-17 Axis in the Pathogenesis of Periodontitis and Immune-Mediated Inflammatory Diseases. Int J Mol Sci. 2019 Jul 10;20(14):3394. doi: 10.3390/ijms20143394. PMID: 31295952; PMCID: PMC6679067.
53. Kim TS, Silva LM, Theofilou VI, Greenwell-Wild T, Li L, Williams DW, Ikeuchi T, Brenchley L; NIDCD/NIDCR Genomics and Computational Biology Core; Bugge TH, Diaz PI, Kaplan MJ, Carmona-Rivera C, Moutsopoulos NM. Neutrophil extracellular traps and extracellular histones potentiate IL-17 inflammation in periodontitis. J Exp Med. 2023 Sep 4;220(9):e20221751. doi: 10.1084/

jem.20221751. Epub 2023 Jun 1. PMID: 37261457; PMCID: PMC10236943.
54. Sun L, Wang L, Moore BB, Zhang S, Xiao P, Decker AM, Wang HL. IL-17: Balancing Protective Immunity and Pathogenesis. J Immunol Res. 2023 Aug 12;2023:3360310. doi: 10.1155/2023/3360310. PMID: 37600066; PMCID: PMC10439834.
55. McClure FA, Wemyss K, Cox JR, Bridgeman HM, Prise IE, King JI, Jaigirdar S, Whelan A, Jones GW, Grainger JR, Hepworth MR, Konkel JE. Th17-to-Tfh plasticity during periodontitis limits disease pathology. J Exp Med. 2024 Aug 5;221(8):e20232015. doi: 10.1084/jem.20232015. Epub 2024 May 31. PMID: 38819409; PMCID: PMC11143381.
56. Magán-Fernández A, Rasheed Al-Bakri SM, O'Valle F, Benavides-Reyes C, Abadía-Molina F, Mesa F. Neutrophil Extracellular Traps in Periodontitis. Cells. 2020 Jun 19;9(6):1494. doi: 10.3390/cells9061494. PMID: 32575367; PMCID: PMC7349145
57. Qiu W, Guo R, Yu H, Chen X, Chen Z, Ding D, Zhong J, Yang Y, Fang F. Single-cell atlas of human gingiva unveils a NETs-related neutrophil subpopulation regulating periodontal immunity. J Adv Res. 2025 Jun;72:287-301. doi: 10.1016/j.jare.2024.07.028. Epub 2024 Jul 30. PMID: 39084404; PMCID: PMC12147643.
58. Zhang J, Feng Y, Shi D. NETosis of psoriasis: a critical step in amplifying the inflammatory response. Front Immunol. 2024 Aug 1;15:1374934. doi: 10.3389/fimmu.2024.1374934. PMID: 39148738; PMCID: PMC11324545.
59. Sansores-España LD, Melgar-Rodríguez S, Olivares-Sagredo K, Cafferata EA, Martínez-Aguilar VM, Vernal R, Paula-Lima AC, Díaz-Zúñiga J. Oral-Gut-Brain Axis in Experimental Models of Periodontitis: Associating Gut Dysbiosis With Neurodegenerative Diseases. Front Aging. 2021 Dec 10;2:781582. doi: 10.3389/fragi.2021.781582. PMID: 35822001; PMCID: PMC9261337.
60. Xue M, Deng Q, Deng L, Xun T, Huang T, Zhao J, Wei S, Zhao C, Chen X, Zhou Y, Liang Y, Yang X. Alterations of gut microbiota for the onset and treatment of psoriasis: A systematic review. Eur J Pharmacol. 2025 Jul 5;998:177521. doi: 10.1016/j.ejphar.2025.177521. Epub 2025 Mar 17. PMID: 40107339.
61. Di Spirito F, Di Palo MP, Rupe A, Piedepalumbo F, Sessa A, De Benedetto G, Russo Barone S, Contaldo M. Periodontitis in Psoriatic Patients: Epidemiological Insights and Putative Etiopathogenic Links. Epidemiologia (Basel). 2024 Jul 26;5(3):479-498. doi: 10.3390/epidemiologia5030033. PMID: 39189252; PMCID: PMC11348036.
62. Mendes VS, Cota LOM, Costa AA, Oliveira AMSD, Costa FO. Periodontitis as another comorbidity associated with psoriasis: A case-control study. J Periodontol. 2019 Apr;90(4):358-366. doi: 10.1002/JPER.18-0394. Epub 2018 Nov 28. PMID: 30387155.
63. Marruganti C, Malvicini G, Cinotti E, Fuso A, Trovato E, Rubegni P, Grandini S, Gaeta C. Association between apical periodontitis and psoriasis vulgaris: A cross-sectional study. Int Endod J. 2025 Jun;58(6):848-861. doi: 10.1111/iej.14222. Epub 2025 Mar 19. PMID: 40105908; PMCID: PMC12065119.
64. Kwak EJ, Choi YJ, Kim HN, Kim KE, Jeon J, Baek YS. Increased dental comorbidities in patients with psoriasis: a nationwide population-based cohort study in Korea. *Clin Exp Dermatol.* 2023 Dec;48(12):1347–1353. doi: 10.1093/ced/llad286. PMID: 37701322
65. Sharma A, Raman A, Pradeep AR. Association of chronic periodontitis and psoriasis: periodontal status with severity of psoriasis. *Oral Dis.* 2015 Apr;21(3):314–9. doi: 10.1111/odi.12274. PMID: 24989645.
66. Marruganti C, Stefanini M, Guidi R, Giannini D, Baldini C, Giusti L, et al. Treatment of periodontitis ameliorates the severity and extent of psoriasis: A randomized clinical trial. *J Periodontal Res.* 2025 Feb;60(2):134–43. doi: 10.1111/jre.13245. PMID: 38899599.
67. Radoi L, Kadri M, Gosset M. Complications of invasive oral procedures in patients with immune-mediated inflammatory disorders treated with biological and conventional disease-modifying antirheumatic drugs or glucocorticoids: a scoping review of the literature. BMC Oral Health. 2025;25:442. doi:10.1186/s12903-024-05414-z
68. Toader MP, Condurache Hritcu OM, Popa C, Sciuca AM, Onofrei BA, Costan VV, Toader SV. The impact of systemic corticosteroid therapy on oral health: complications and management strategies. *Romanian Journal of Medical and Dental Education*. 2023;12(5):

69. Monson carlos. Periodontal Aspects for Psoriasis: A Systematic Review. Journal of Clinical Research in Dermatology. doi:10.15226/2378-1726/3/4/0014
70. Nijakowski K, Rutkowski R, Eder P, Korybalska K, Witowski J, Surdacka A. Changes in salivary parameters of oral immunity after biologic therapy for inflammatory bowel disease. Life. 2021;11(12):1409. doi:10.3390/life11121409
71. Zhang X, Gu H, Xie S, Su Y. Periodontitis in patients with psoriasis: A systematic review and meta-analysis. Oral Dis. 2022 Jan;28(1):33-43. doi: 10.1111/odi.13617. Epub 2020 Sep 18. PMID: 32852860; PMCID: PMC9290533
72. Hajishengallis G, Chavakis T. Local and systemic mechanisms linking periodontal disease and inflammatory comorbidities. Nat Rev Immunol. 2021;21(7):426-40. doi:10.1038/s41577-020-00488-6
73. Baurecht H, Freuer D, Welker C, Tsoi LC, Elder JT, Ehmke B, Leitzmann MF, Holtfreter B, Baumeister SE. Relationship between periodontitis and psoriasis: A two-sample Mendelian randomization study. J Clin Periodontol. 2022 Jun;49(6):573-579. doi: 10.1111/jcpe.13620. Epub 2022 Apr 1. PMID: 35362630; PMCID: PMC9117489
74. Di Spirito F, Di Palo MP, Rupe A, Piedepalumbo F, Sessa A, De Benedetto G, Russo Barone S, Contaldo M. Periodontitis in Psoriatic Patients: Epidemiological Insights and Putative Etiopathogenic Links. Epidemiologia (Basel). 2024 Jul 26;5(3):479-498. doi: 10.3390/epidemiologia5030033. PMID: 39189252; PMCID: PMC11348036.
75. Mainas G, Ide M, Rizzo M, Magan-Fernandez A, Mesa F, Nibali L. Managing the Systemic Impact of Periodontitis. Medicina (Kaunas). 2022 Apr 29;58(5):621. doi: 10.3390/medicina58050621. PMID: 35630038; PMCID: PMC9147054.
76. Di Spirito F, Di Palo MP, Rupe A, Piedepalumbo F, Sessa A, De Benedetto G, Russo Barone S, Contaldo M. Periodontitis in Psoriatic Patients: Epidemiological Insights and Putative Etiopathogenic Links. Epidemiologia (Basel). 2024 Jul 26;5(3):479-498. doi: 10.3390/epidemiologia5030033. PMID: 39189252; PMCID: PMC11348036
77. Marruganti C, Gaeta C, Falciani C, Cinotti E, Rubegni P, Alovisi M, Scotti N, Baldi A, Bellan C, Defraia C, Fiorino F, Valensin S, Bellini E, De Rosa A, D'Aiuto F, Grandini S. Are periodontitis and psoriasis associated? A pre-clinical murine model. J Clin Periodontol. 2024 Aug;51(8):1044-1053. doi: 10.1111/jcpe.13996. Epub 2024 May 3. PMID: 38699834.

## SAŽETAK

### Psorijaza i parodontitis: zajednička imunopatogeneza, molekularni upalni mehanizmi i kliničke implikacije

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Psorijaza je kronična, autoimuna, genetski uvjetovana bolest kože s naizmjeničnim periodima regresije i egzacerbacije same bolesti koja je obilježena karakterističnim promjenama u obliku eritematoskvamoznog plaka. Nastanak psorijaze uvjetovan je genetskim i okolišnim čimbenicima kao i kompleksnom upalnom kaskadom za koju dan danas ne postoje točni podaci o tome koji je glavni pokretač same upale i nastanka psorijatičnog plaka. Kod pacijenata koji boluju od psorijaze primijećena je povećana učestalost nastanka parodontitisa. Parodontitis čini skupina kroničnih bolesti oralne šupljine koja je izrazito česta u odrasle populacije i povezana je s kroničnim infekcijama oralne šupljine. Kronična upala te imunološki mehanizmi nastanka ovih bolesti i danas su predmet istraživanja i traženja ključnih terapijskih opcija. U ovom preglednom radu istražili smo postoji li povezanost između dvije kronične upalne imunološki posredovane bolesti – psorijaze i parodontitisa – te postoji li potencijalna mogućnost pokretanja multisistemske bolesti kao što je psorijaza kod pacijenata oboljelih od parodontitisa.

**KLJUČNE RIJEČI**

*Psorijaza; Parodontitis; Sustavna upala; Kronične infekcije*