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Review

Could Periodontitis Aggravate Psoriasis?—An Update by Systematic Review

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Abstract: (1) Background: Psoriasis is a chronic and inflammatory systemic disease that has been associated with periodontal pathologies, specifically periodontitis. The aim of this research is to answer the following question: Could periodontitis aggravate psoriasis? (2) Methods: We carried out a systematic review following the PRISMA guide using PubMed, Embase, Scopus, and WOS; (3) Results: A total of 111 studies were identified in the databases and 11 were obtained after screening. The selection included nine case–control studies, one cross-sectional study, and one cohort study. Most of the publications report an increase in bleeding on probing and the presence of periodontal pockets in patients with psoriasis, confirming that inflammation caused by periodontitis can contribute to systemic inflammation worsening psoriasis. To summarize, the scientific literature indicates that local periodontal inflammation could aggravate psoriasis.

Keywords: psoriasis; periodontitis; inflammatory disease; periodontal disease; risk factors for psoriasis

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1. Introduction

Psoriasis is a chronic, immune-mediated inflammatory skin disease, characterized by red and scaly plaques occurring more frequently on the elbows, knees, scalp, and lower back [1]. Worldwide, in 2017, an estimated 29.5 million adults had psoriasis, corresponding to a physician-diagnosed lifetime prevalence of 0.59% of the adult population [1]. Plaque psoriasis is the most frequent type, representing more than 80% of psoriasis cases [2]. The pathogenesis of plaque psoriasis consists of a feed-forward mechanism of inflammation involving primarily the T-helper cell type 17 (TH17) pathway [2]. In the past, psoriasis was considered a disease that was limited to the skin and was treated with topical agents or phototherapy. With recent advances, research has focused on clarifying the roles of specific proinflammatory cytokines that contribute to the disease's pathogenesis [3].

Psoriasis is a visible skin disease, and therefore relationships with other people can be disturbed. Many patients encounter prejudice and rejection and feel that their attractiveness is diminished. Therapy and skin care are time-consuming, so psoriasis patients may be limited in their work, leisure time, and freedom of movement due to physical symptoms [1,4]. Apart from comorbidities [5], people with psoriasis not only have to cope with physical limitations but also with severe psychological burdens such as depression, anxiety, and suicidal thoughts [1,4].

Guideline-indicated therapeutic options involve topical treatments, phototherapy, and systemic therapies which encompass both oral treatments and injectable biologics. Eighty per cent of patients with psoriasis have mild-to-moderate forms of psoriasis and can be treated exclusively with topical agents such as corticosteroids and vitamin D analogs. Phototherapy and systemic agents are recommended for patients with moderate-to-severe psoriasis, where the extent of the disease makes topical therapy of all lesions impractical [6].

Recently, psoriasis has been related to other chronic inflammatory conditions, such as periodontitis. Accumulated epidemiologic, genetic, and pathogenetic evidence indicates that psoriasis is associated with this condition [7]. The American Academy of Periodontics defines periodontal disease as an inflammation of the supporting tissues of the tooth [8]. It is a progressive destruction process that leads to the loss of the supporting bone of the tooth and its periodontal ligament [9]. The prevalence of periodontitis is reported to vary from 20% to 50% around the world. Additionally, periodontitis is one of the major causes of tooth loss, which can undermine function, aesthetics, self-confidence, and quality of life [10].

Periodontitis is a chronic inflammatory condition provoked by a bacterial infection that activates the host immune response [10–16]. Psoriasis could cause periodontal lesions and sometimes white plaque and erythema lesions on the oral mucosa and palate [11,16]. This disease is an inflammatory condition considered a result of the complex interaction between the oral microbial community and the host response, modified by genetics and environmental factors. In recent decades, there has been increasing evidence supporting a strong relationship between periodontitis and systemic conditions. These conditions include cardiovascular diseases, metabolic syndrome, obesity, rheumatoid arthritis, polycystic ovary syndrome, and adverse outcomes during pregnancy [11–13].

Since psoriasis and periodontitis have similar pathogenic mechanisms and associated conditions in common, there has been a renewed interest in research into possible links between these diseases. The current hypothesis of common etiopathogenic processes between the conditions comprises several possible mechanisms, such as amplified inflammatory response and T-cell activation and a lower concentration of salivary IgA and lysozymes [11]. Some studies have already indicated that patients with psoriasis have a significantly elevated risk of periodontitis compared with controls without psoriasis [14–20]. This was especially observed in patients with severe psoriasis [21]. In addition, a meta-analysis reported that patients with periodontitis have a significantly increased risk of psoriasis [22]. Therefore, the aim of this study was to establish if periodontitis could aggravate psoriasis and if psoriasis patients had more risk of developing periodontal disease or presenting worse periodontal status.

2. Materials and Methods

We carried out a systematic review following the PRISMA guide (Preferred Reporting Items for Systematic Reviews and Meta-analyses), and the PRISMA check list is available as Supplementary Materials [23]. We built an evidence-based research method for incorporating a PECO question model (PECO: Participants, Exposure, Control and Outcomes). The focused question was: Could periodontitis aggravate psoriasis? Patients (P): patients with psoriasis; Exposure (E): exposition to periodontitis; Control (C): periodontal healthy patients. Outcome (O): periodontal and clinical parameters. It was intended to establish if periodontitis can be considered a risk cofactor for aggravating periodontal problems in patients in comparison with healthy patients. The chronic inflammation that exists in periodontitis could cause higher vulnerability to developing more clinical manifestations of psoriasis.

2.1. Search Strategy

To carry out the systematic review we used the following databases: PubMed (National Library of Medicine, Washington, DC, USA), Embase, Scopus (Elsevier B.V., Amsterdam, The Netherlands), and Web of Science Core Collection. The keywords employed were: periodontitis, psoriasis, inflammation, and inflammatory disorder. The search strategy was performed using MeSH terms. To facilitate the reproducibility of the search strategy, a QR code was generated for each database used (Figure 1). This systematic review was registered in PROSPERO, the international prospective register of systematic reviews, with the following registration code: CRD42021261141.

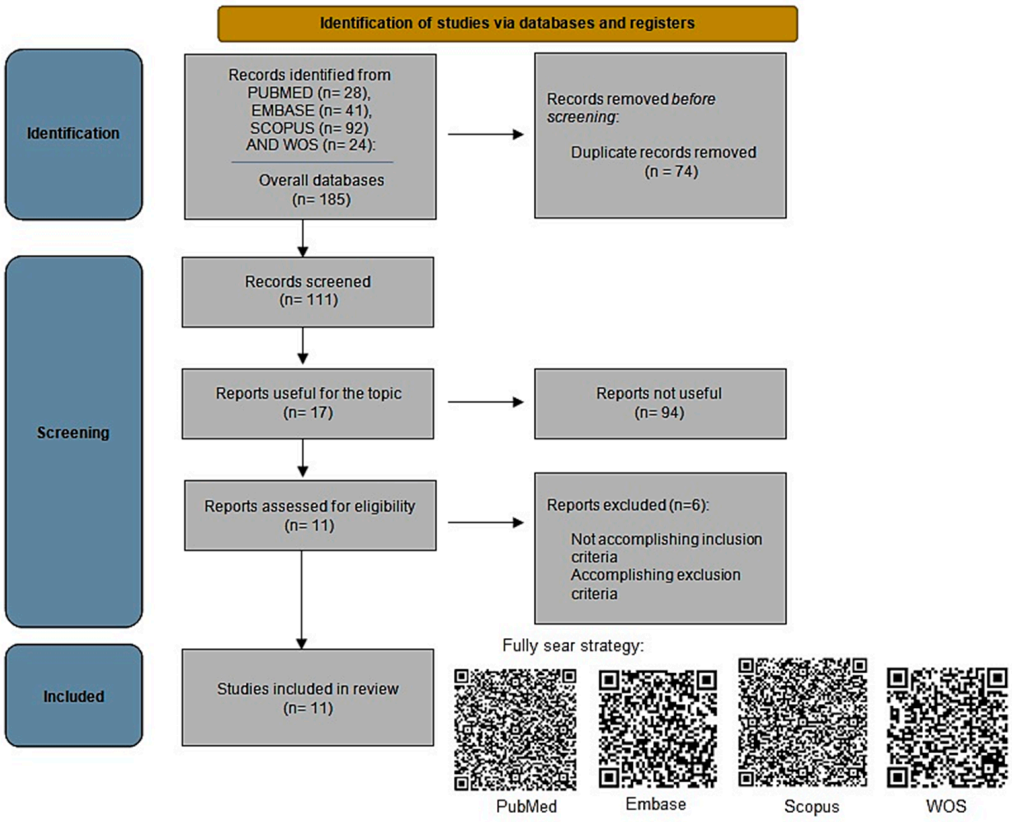

Figure 1. Flow chart following the PRISMA guide [23].

2.2. Selection of Articles and Eligibility Criteria

The inclusion criteria were: original research articles reporting longitudinal studies, cross-sectional studies, clinical trials, cohort studies, or case–control studies in the previous 5 years. Besides, the sample size included was research with 30 or more individuals and articles with quality score of 4 or higher. The exclusion criteria were: topic reviews, case reports, and low quality articles. After conducting a bibliographic search, duplicate articles were removed. Moreover, studies that were not deemed useful for the topic were excluded. Finally, those articles which did not meet the inclusion criteria were not considered for this systematic review. Thus, a selection of articles was established to answer the PECO question (Figure 1).

2.3. Quality Control of Articles

The Newcastle–Ottawa scale for quality control was used. This scale checks the selection of the study groups, the comparability of the groups, and the outcomes (exposure for case–control studies). It is a "gold standard" system that assesses these 3 features, with each comprising several items depending on the type of publication: case–control study (Table 1), cross-sectional study (Table 2), and cohort study (Table 3).

Table 1. Newcastle–Ottawa scale for case–control study.

Author, Year	Selection Items				Comparability Item	Exposure Items			Total
	1	2	3	4	5	6	7	8	
Sezer et al., 2016			*	*	*	*	*		6/9
Painsi et al., 2017				*	*	*	*		4/9
Sarac et al., 2017		*	*	*	*	*	*		6/9
Woeste et al., 2019	*	*	*	*	*	*			6/9
Macklis et al., 2019	*	*	*		*	*			5/9
Mendes et al., 2019	*	*	*	*	*	*			6/9
Barros et al., 2020	*			*	*	*	*		5/9
Belstrøm et al., 2020	*				*	*	*		4/9
Skutnik-Radziszewska et al., 2020		*	*		*	*	*		5/9

Selection: 1: Is the case definition adequate? (1 point); 2: Representativeness of the cases (1 point); 3: Selection of controls (1 point); 4: Definition of controls (1 point); Comparability: 5: Comparability of cases and controls on the basis of the design (1 point) or/and analysis (1 point); Exposure: 6: Ascertainment of exposure (1 point); 7: Same method of ascertainment for cases and controls (1 point); 8: Non-response rate (1 point). *: Corresponds to 1 point from total score when the research adequately meets the item.

Table 2. Newcastle-Ottawa scale for cross-sectional study.

Author, Year	Selection Items				Comparability Item	Outcome Items		Total
	1	2	3	4	5	6	7	
Ligia et al., 2019	*			**	*	**		6/9

Selection: 1: Representativeness of the sample (1 point); 2: Sample size (1 point); 3: Non-respondents (1 point); 4: Ascertainment of exposure (2 points); Comparability: 5: Subjects in different outcomes are comparable, based on the study design or analysis, and confounding factors are controlled (1 point); Outcome: 6: Assessment of the outcome (2 points); 7: Statistical test (1 point). *: Corresponds to 1 point from total score when the research adequately meets the item. **: 2 points.

Table 3. Newcastle-Ottawa Scale for cohort study.

Author, Year	Selection Items				Comparability Item	Outcome Items			Total
	1	2	3	4	5	6	7	8	
Egeberg et al., 2017	*	*	*		**	*	*		7/9

Selection: 1: Representativeness of the exposed cohort (1 point); 2: Selection of the non-exposed cohort (1 point); 3: Ascertainment of exposure (1 point); 4: Demonstration that outcome of interest was not present at start of study (1 point); Comparability: 5: Comparability of cohorts on the basis of the design or analysis (2 points); Outcome: 6: Assessment of outcome (1 point); 7: Was follow up long enough for outcomes to occur (1 point); 8: Adequacy of follow up of cohorts (1 point). *: Corresponds to 1 point from total score when the research adequately meets the item. **: 2 points.

3. Results

In this systematic review, a total of 111 studies were identified in the databases, and 11 were obtained after screening (Figure 1). The selection included nine case–control studies, one cross-sectional study, and one cohort study. The general characteristics of the studies analyzed in this review are presented in Table 4. All the articles used large samples of between 71 and 5,470,428 individuals. Regarding the case–control studies, these mainly compared the results of periodontal tests and epidemiological indexes (prevalence and incidence) between a group exposed to psoriasis and a control group. In the cross-sectional study, the periodontal status of the sample was analyzed. In the cohort study, the different periodontal states of different groups were compared, including psoriasis patient groups

and control groups. Most of the publications report an increase in bleeding on probing and the presence of periodontal pockets in patients with psoriasis and indicate that local periodontal inflammation could aggravate psoriasis.

Table 4. General characteristics of the studies analysed in this review.

Author, Year	Type of Study	Sample Size	Periodontal Evaluation	Conclusions
Sezer et al., 2016	Case–control	100 cases 20 controls	PI, PD, CAL, and BOP%	There were no differences between periodontitis (systemically healthy-chronic periodontitis, psoriasis-chronic periodontitis, psoriatic arthritis-chronic periodontitis) groups
Painsi et al., 2017	Case–control	209 cases 91 controls	Data from patient who underwent an inflammatory focus screening, including a dental check up	Higher periodontitis prevalence in psoriasis patients compared with chronic spontaneous urticaria (OR = 3.76; 95% CI 1.60–10.27; $p = 0.001$)
Sarac et al., 2017	Case–control	76 cases 76 controls	CPITN	In the psoriasis group, there were higher values in CPITN
Woeste et al., 2019	Case–control	100 cases 101 controls	BOP, CPITN, and dental parameters according to the DMFTI	The author found higher values in BOP and CPITN in psoriasis group. There were no differences for DMFTI. Periodontitis could aggravate psoriasis simptosms.
Macklis et al., 2019	Case–control	100 cases 165 controls	Validated WHO survey for adult oral hygiene practices including gingivitis and periodontitis signs	Patients who reported poor or very poor gum health showed more symptoms of severe psoriasis. Periodontitis could aggravate psoriasis simptosms.
Mendes et al., 2019	Case–control	397 cases 325 controls	PI, PD, CAL, and BOP	Psoriasis patients had higher PI, PD, BOP, and CAL values. Psoriasis individuals showed more probability of suffering periodontitis when compared with controls (OR = 1.72; 95% CI 1.28–2.32; $p < 0.001$).
Barros et al., 2020	Case–control	69 cases 74 controls	PI, PD, CAL, BOP, and DMFTI	Psoriasis patients had lower PI values, higher CAL and DMFT values, and fewer teeth. More prevalence of severe and generalized periodontitis. Severe periodontitis can be considered a risk factor for psoriasis (OR = 3.7; 95% CI 1.5–9.0; $p < 0.003$). BOP was not significantly different.
Belstrøm et al., 2020	Case–control	85 cases 52 controls	PI, BOP, PD, and CAL	Patients with psoriasis had good periodontal health (regularly attending dental care). No differences in missing teeth, PD, or CAL. Relatively high percentages of BOP and PI. Lower salivary levels of NGAL and transferrin.
Skutnik-Radziszewska et al., 2020	Case–control	40 cases 40 controls	Dental status, DMFTI, PBI, and GI	There were no differences in the values of DMFTI, PBI, or GI

Table 4. *Cont.*

Author, Year	Type of Study	Sample Size	Periodontal Evaluation	Conclusions
Ligia et al., 2019	Cross-sectional	71 participants	PI, BOP, CAL, and PD	Periodontal disease was frequent in patients with psoriasis. Nevertheless, there was no statistically significant (small sample). No data for periodontal indexes.
Egeberg et al., 2017	Cohort	5,470,428 participants	Patients with periodontitis were identified by their first inpatient or outpatient (ambulatory) hospital diagnosis of periodontitis	Increased risk of periodontitis in mild psoriasis linebreak (IRR: 1.66; 95% CI 1.43–1.94; $p < 0.001$), severe psoriasis (IRR: 2.24; 95% CI 1.46–3.44; $p < 0.001$) and psoriatic arthritis (IRR: 3.48; 95% CI 2.46–4.92; $p < 0.001$). Periodontitis could agravate psoriasis simptosms.

Abbreviations: PI: plaque index; CAL: clinical attachment loss; BOP: bleeding on probing; PD: probing depth; GI: gingival index; CPITN: community periodontal index of treatment needs; DMFTI: decayed, missing, and filled teeth index; D: decayed teeth; M: missing teeth; FT: filled teeth; PBI: papilla bleeding index; IRR: incidence rate ratio.

3.1. Periodontal Parameters

The periodontal parameters used in most of the investigations were probing depth (PD) [18,19,24–26], clinical attachment loss (CAL) [18,19,24–26], and the community periodontal index of treatment need (CPITN) (15,16). All of these indices are indicative of the stage of periodontitis according to the new classification of 2017 [27]. PD had higher values in one study [18], but in the others that analyzed it there were no differences between cases and controls [19,24–26]. CAL had higher values in some of the studies reviewed [18,19] but not in others [24–26]. There were significant differences in CPITN in the two studies involving it [15,16]. In addition, plaque index (PI) was used to assess the oral hygiene of some patients [18,19,24–26], with most studies finding no differences in this measure between cases and controls.

On the other hand, other authors have used some less frequent indexes, such as bleeding on probing (BOP) [16,18,19,24–26]; the decayed, missing, and filled teeth index (DMFTI) [16,19,28]; the papilla bleeding index (PBI) [28]; and the gingival index (GI) [28] (Table 4).

3.2. Epidemiological Parameters

Likewise, it is especially important to mention the epidemiological factors identified in this review. Some of the studies used prevalence indicators [14,18,19] while other publications used incidence rates [20]. Thus, the prevalence of periodontitis was 23.9–46.1% in psoriasis patients compared with 7.7–33.1% in healthy controls [14,18]. An OR of between 1.72 (95% CI 1.28–2.32; $p < 0.001$) [18] and 3.76 (95% CI 1.60–10.27; $p = 0.001$) [14] was found for the risk of psoriasis patients suffering periodontitis. Regarding the incidence of periodontitis in the cohort study [20], the authors found significant differences between cases and controls. The incidence rate ratio (IRR) in mild psoriasis was 1.66 (95% CI 1.43–1.94; $p < 0.001$), 2.24 (95% CI 1.46–3.44; $p < 0.001$) in psoriatic arthritis, and 3.48 (95% CI 2.46–4.92; $p < 0.001$) in severe psoriasis. Finally, Macklis [17] used a validated WHO survey to establish the state of gums in adult patients with psoriasis compared with healthy controls, and it was observed that psoriasis patients who considered their gum health to be poor or very poor had significantly more severe psoriasis symptoms.

4. Discussion

This systematic review has given our PECO question an affirmative answer with periodontitis being a disease that could aggravate the clinical manifestations of psoriasis. Patients with psoriasis present an increase in proinflammatory cytokines that leads to a bidirectional association between both pathologies [14–20,24–26,28]. This topic has attracted interest because of the effects that both diseases have on patients and because of the large number of people who suffer from both of them. These two conditions share several common immunologic, micro-biological, and environmental pathogenetic factors. Although the etiopathogenesis is not fully understood, it is proposed that the environmental factors modify the diversity of the local microbiome and produce dysbiosis. Altogether, these factors lead to T-cell activation and cytokine production [7], which starts the inflammatory process. Thus, there has been increasing attention in establishing if psoriasis and periodontitis have a relationship.

In two previous similar studies it was determined that psoriasis patients had higher chances of suffering from periodontitis. Qiao et al. [21] carried out a meta-analysis of eight articles, finding significant differences in BOP, PD, CAL, and remaining and missing teeth, as well as in the level of alveolar bone loss. There were no differences in PI and GI. The authors elucidated that psoriasis patients suffer from worse periodontal health compared with non-psoriasis subjects, and, despite a more detailed investigation being needed, it was concluded that the confounding factors should be taken into much more consideration. Moreover, it was stated that there were not enough studies to establish solid conclusions for some indexes and that more papers should undertake adequate quality meta-analysis. Zhang et al. [11] performed a systematic review, concluding that psoriasis and periodontitis were bidirectionally related, but the authors mention that there was high heterogeneity among the papers and a higher number of articles was needed. Zang et al. [11] also report that the role of confounding factors such as age, gender, or systemic conditions should be highlighted. Additionally, establishing precise and common criteria for the diagnosis of periodontitis was deemed critical. Regarding the present paper, there were three studies that did not find any differences between the psoriasis patients and the control groups [25,26,28]. Ligia et al. (24) also show no statistical significance between groups, although periodontitis was more frequent in psoriasis patients. The remaining seven articles [13–19] gathered significant evidence that patients with psoriasis were more susceptible to suffering periodontal disease. These articles [14–20] used epidemiological indexes (prevalence [14,18,19] and incidence [20]), periodontal indexes [15,16,18,19], and questionnaires [17]. In several studies [13,17,18], it was found that there was a higher prevalence of periodontitis in psoriasis patients with an OR (95% CI) of between 1.72 (1.28–2.32, $p < 0.001$) [18] and 3.76 (1.60–10.27, $p = 0.001$) [14]. Eberg et al.'s cohort study [20] has to be highlighted as the initial sample was all individuals aged 18 or over from Denmark, with a final sample was composed of 5,470,428 individuals. Their results show through the IRR that there is an increased risk of periodontitis in mild psoriasis (IRR: 1.66; 95% CI 1.43–1.94; $p < 0.001$), severe psoriasis (IRR: 2.24; 95% CI 1.46–3.44; $p < 0.001$), and psoriatic arthritis (IRR: 3.48; 95% CI 2.46–4.92; $p < 0.001$).

However, the results are not as clear for the PD measure with four out of five articles not finding any differences [19,24–26]. For the CAL measure, three out of five papers found no differences between cases and controls [24–26]. Furthermore, all of the studies that analyzed PI did not find any differences [18,19,24–26]. Regarding prevalence, three out of three studies showed significant differences [14,18,19]. All of this may indicate that psoriasis can be a risk factor for developing periodontal disease. Nevertheless, the diagnosis of periodontitis in these three publications was different. While Painsi et al. [14] used registers to identify periodontal disease patients, Mendes et al. [18] employed interproximal CAL and/or PD and Barros et al. [19] only used the CAL measure in interproximal sites. The different diagnosis methods in the studies increases the heterogeneity of the results and the subsequent conclusions. For further studies, there should be criteria for always establishing the same method of diagnosis since without this, it is difficult to compare and generalize

results, especially in relation to registers. Although it is a good method for large studies, it reduces their precision. Thus, a goal for this line of research would be having a common diagnosis for both periodontitis and psoriasis.

In addition, there is the question of confounding factors, with age, gender, or systemic conditions having been mentioned already. However, there may be other factors that have the capacity to modify the results. Socioeconomic status is likely to alter periodontitis outcomes, so patients with less access to healthcare or healthy conditions are more likely to have worse outcomes. Another confounding factor is the presence of plaque, as there will not be the same outcomes for people with poor hygiene vs. people with good oral care. There have been articles involved in some systematic reviews with very high PI values, and these values are going to change the results because the periodontium is not going to react in the same way to good hygiene as it does to poor hygiene. Both diseases have been shown to cause inflammatory changes in the form of increased cytokine values [14–16,19,20,24–26]. Since they are essential in the pathogenesis and progression of periodontitis and psoriasis, it can be speculated that increased cytokine values may favor the development of periodontitis [14–16,18,26]. This altered state would render the individual susceptible to developing inflammatory diseases. Therefore, it has been shown that if periodontal disease is treated, the psoriasis condition improves [29,30]. In addition, systemic psoriasis therapy could lead to better periodontal parameters [29–31].

An association between psoriasis and periodontitis has been shown, and increased concentrations of proinflammatory cytokines such as TNF-α and IL-1β have been found in saliva from patients with psoriasis [3,30–32]. Activated TH17 cells producing IL-17 are key pathogenic players in psoriasis, and bacterial infection, including infection with *P. gingivalis*, may also activate TH17 cells. This bacterial infection can activate inflammatory pathways, promoting secretion of interleukins and increasing the clinical manifestations of psoriasis by contributing to systemic inflammation. Moreover, activated TH17 cells have been found in periodontal lesions and in mild psoriasis, and increased IL-17 levels have been demonstrated in crevicular fluid from patients with mild psoriasis [2,3,30–32]. These findings show that TH17 hyperactivation could be a pathway that connects both pathologies, sharing pathophysiological mechanisms present in psoriasis and periodontitis [2,3,7,17–19,31–33].

However, there is some heterogeneity in the results of recent articles on this topic, which could be because the investigations used different diagnosis methods for periodontitis. Prospective and more detailed research is required to obtain more evidence. In any case, this manuscript has carried out a review following the PRISMA guidelines [23] and using quality scales with a thorough protocol developed by three researches with experience in this field. Moreover, this publication covers the most recent articles on the subject and has incorporated the most cited papers on the relationship between periodontitis and psoriasis.

5. Conclusions

The scientific literature available up to now affirms that periodontitis could aggravate the clinical manifestations of psoriasis. A bidirectional association between both pathologies is proposed: On the one hand, patients with psoriasis typically present oral lesions that make them more at risk of developing periodontal diseases [1,4,6,11,16], and on the other hand, periodontitis in patients with psoriasis may increase the poll of proinflammatory cytokines and in this way aggravate the clinical manifestations of psoriasis [14–20,24–26,28,31,32]. Clearly, the role of the dentist is of great importance, since a dental examination can identify the presence of periodontitis and provide treatment to patients. This treatment can improve the systemic inflammatory process and improve the evolution of psoriasis, so periodontal treatment would also improve the consequences of psoriasis. In addition, since the evidence points to periodontitis and psoriasis having a bidirectional relationship, dental practitioners should carry out a comprehensive dental checkup in these populations. Patients diagnosed with psoriasis could also undergo specific gum surveillance, since there is evidence that psoriasis may be a risk factor for periodontitis too.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/oral3010006/s1, The PRISMA check list.

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