JOURNAL OF EDUCATION, HEALTH AND SPORT
eISSN 2391-8306 · Open Access · Peer-reviewed
apcz.umk.pl/JEHS · Nicolaus Copernicus University in Toruń

Cite as: STEFANIK, Zofia, STAŃKO, Mikołaj, STEFANIK, Natalia, SMAGOWSKA, Julia, MUSIAŁ, Paulina, PIECZARA, Szymon, PASTUSZKA, Jakub and WILK, Julia. Biopsychosocial Pathways Linking Depression and Oral Health: A Literature Review. Journal of Education, Health and Sport. 2026;92:72462. https://doi.org/10.12775/JEHS.2026.92.72462

ARTICLE TIMELINE
Received: 23.05.2026 Revised: 25.05.2026
Accepted: 26.05.2026 Published: 10.06.2026

INDEXING & EVALUATION
MEiN points: 40 Unique ID: 201159
Disciplines: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).

The journal has been awarded 40 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32318). Unique Journal Identifier: 201159. Scientific disciplines: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).

Punkty Ministerialne z 2019 – aktualny rok 40 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).

Biopsychosocial Pathways Linking Depression and Oral Health: A Literature Review

Zofia Stefanik, ORCID https://orcid.org/0009-0005-0722-2700
Email: stefanik.zosia@gmail.com
University Dental Center of the Medical University of Silesia in Katowice, Katowice, Poland

Mikołaj Stańko, ORCID https://orcid.org/0009-0006-8703-4482
Email: mikolaj.stanko17501@student.akademiaslaska.pl
Academy of Silesia, Faculty of Medicine, Katowice, Poland

Natalia Stefanik, ORCID https://orcid.org/0000-0003-0764-846X
Email: nstefanik@sum.edu.pl
Department of Periodontal Diseases and Oral Mucosa Diseases, Faculty of Medical Sciences in Zabrze, Medical University of Silesia, Katowice, Poland

Julia Smagowska, ORCID https://orcid.org/0009-0003-4275-0846
Email: juliasmagowska1@gmail.com
University Dental Center of the Medical University of Silesia in Katowice, Katowice, Poland

Paulina Musiał, ORCID https://orcid.org/0009-0001-1386-6087
Email: paulinamusial2002@gmail.com
University Dental Center of the Medical University of Silesia in Katowice, Katowice, Poland

Szymon Pieczara, ORCID https://orcid.org/0009-0003-0355-4762
Email: szym.pieczara@gmail.com
Wrocław Medical University, Wrocław, Poland

Jakub Pastuszka, ORCID https://orcid.org/0009-0002-4423-4979
Email: jakubpass0@gmail.com
Academy of Silesia, Faculty of Medicine, Katowice, Poland

Julia Wilk, ORCID https://orcid.org/0009-0009-3471-833X
Email: juliawilk70@gmail.com
Academy of Silesia, Faculty of Medicine, Katowice, Poland

Corresponding Author: Zofia Stefanik — stefanik.zosia@gmail.com

Abstract

Background: Despite their high global prevalence, depression and oral diseases are rarely addressed within a unified clinical framework. Recent evidence highlights a bidirectional relationship with overlapping biological and behavioral underpinnings.

Aim: This review consolidates current knowledge on the mutual influence of depression and oral health and explores the underlying mechanisms.

Methods: A structured search of PubMed/MEDLINE, EMBASE, PsycINFO, and the Cochrane Library identified systematic reviews, meta-analyses, observational, and mechanistic studies published between 2016 and 2025.

Results: Depression is consistently linked to caries, periodontitis, tooth loss, and xerostomia, while periodontal inflammation may contribute to depressive symptomatology. Underlying mechanisms span neuroendocrine, immune, microbial, behavioral, pharmacological, and psychosocial domains.

Conclusions: Recognizing depression and oral disease as mutually reinforcing conditions supports the case for integrated, interdisciplinary care models that incorporate routine cross-screening.

Keywords: *Depressive Disorder, Major; Periodontal Diseases; Oral Health; Neuroinflammation; Integrated Care*

Introduction

Major depressive disorder (MDD) and oral diseases rank among the most prevalent health burdens globally, yet their clinical interconnection remains insufficiently recognized in practice. MDD, projected to rank as the second leading cause of disability worldwide, affects millions of individuals and frequently coexists with poor oral health outcomes [1,4]. Accumulating evidence indicates that this relationship is bidirectional and mechanistically complex, involving biological, behavioural, and psychosocial pathways. [5,6].

Epidemiological studies consistently demonstrate significant associations between depression and multiple oral health conditions

1,3,4[](https://pubmed.ncbi.nlm.nih.gov/33103263). Meta-analytic evidence indicates that depression is associated with increased odds of dental caries, tooth loss, and edentulism, while periodontal disease predicts the development of depression 1,7. Additional meta-analytic data confirm that periodontal disease is positively associated with both depression and anxiety 3. The magnitude of these associations intensifies with increasing severity of both conditions, and a dose-response relationship exists between the number of oral health conditions and depression severity 1,8.

A recent scoping review of 165 longitudinal studies identified 35 independent associations between oral exposures and mental disorder outcomes, with depression linked bidirectionally to temporomandibular disorders (TMD) and periodontitis 6. Longitudinal data from nationally representative cohorts demonstrate that internalizing problems predict bleeding gums and tooth extraction at two-year follow-up 9. Data from the Azar cohort study demonstrate that individuals with depression, particularly those not receiving antidepressant medication, have a significantly higher prevalence of untreated decayed teeth compared to non-depressed individuals 10.

Understanding the mechanisms underlying this bidirectional relationship requires a comprehensive biopsychosocial framework that integrates neuroendocrine dysregulation, systemic inflammation, behavioral factors, pharmacological effects, and psychosocial consequences 3,5,11[](https://pubmed.ncbi.nlm.nih.gov/35153869). This review synthesizes current evidence on these interconnected pathways and discusses implications for integrated clinical care.

Research materials and methods

This literature review synthesizes current evidence on the bidirectional relationship between depression and oral health. The review integrates findings across biological, behavioral, pharmacological, and psychosocial domains to provide a comprehensive biopsychosocial perspective on this relationship 12-15[](https://pubmed.ncbi.nlm.nih.gov/21800117).

Search Strategy

A comprehensive literature search was conducted across four major electronic databases: PubMed/MEDLINE, EMBASE, PsycINFO, and the Cochrane Library. The search was performed using a combination of Medical Subject Headings (MeSH) terms and free-text keywords. The primary MeSH terms included: "Depressive Disorder, Major", "Periodontal Diseases", "Dental Caries", "Oral Health", "Inflammation", "Xerostomia", "Antidepressive Agents", and "Delivery of Health Care, Integrated".

Free-text keywords included: "depression," "oral health," "periodontitis," "dental caries," "HPA axis," "cortisol," "neuroinflammation," "blood-brain barrier," "oral microbiome," "xerostomia," "dry mouth," "antidepressants," "SSRIs," "tricyclic antidepressants," "oral health-related quality of life," "integrated care," and "biopsychosocial." Boolean operators (AND, OR) were used to combine search terms across thematic domains.

Eligibility Criteria

Studies were included if they met the following criteria:

1. published in English;

2. published primarily between 2016 and 2025, with seminal earlier works included where relevant;

3. addressed the relationship between depression or depressive symptoms and oral health outcomes (dental caries, periodontal disease, tooth loss, edentulism, xerostomia, or oral health-related quality of life);

4. were systematic reviews, meta-analyses, randomized controlled trials, cohort studies, cross-sectional studies with adequate sample sizes, preclinical mechanistic studies, or qualitative studies on integrated care models.

Studies were excluded if they:

1. were case reports or case series with fewer than 10 participants;

2. were published in languages other than English without available translation;

3. focused exclusively on psychiatric conditions other than depression (e.g., schizophrenia, bipolar disorder) without relevance to depressive symptomatology;

4. lacked sufficient methodological detail to assess study quality.

Study Selection and Data Extraction

Titles and abstracts were screened for relevance to the five thematic domains of the review:

1. biological pathways (neuroendocrine and inflammatory mechanisms);

2. behavioral mechanisms (oral hygiene, diet, healthcare-seeking);

3. pharmacological factors (antidepressant-induced oral side effects);

4. psychosocial impact (bidirectional relationship, quality of life);

5. clinical implications (integrated care models, interventions).

Full texts of potentially relevant articles were retrieved and assessed for inclusion. Reference lists of included systematic reviews and meta-analyses were hand-searched to identify additional relevant studies Data were extracted focusing on study design, population characteristics, key findings, effect sizes (where reported), and clinical implications. Priority was given to systematic reviews and meta-analyses as the highest level of evidence, followed by randomized controlled trials, prospective cohort studies, cross-sectional studies, preclinical studies, and qualitative research [14,15]**.**

Quality Assessment

The quality of included systematic reviews and meta-analyses was assessed based on adherence to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [14]. For observational studies, the Newcastle-Ottawa Scale (NOS) criteria were considered as reported by the original authors [3,16]. For qualitative studies, methodological rigor was assessed based on transparency of methods, sampling strategy, and analytical approach [15]. No formal critical appraisal scoring was applied. Quality considerations were integrated into the evidence synthesis, with priority given to systematic reviews and meta-analyses as higher-level evidence [12,13].

Evidence Synthesis

Findings were synthesized thematically across the five domains described above. Where multiple meta-analyses addressed the same association, results were compared and any discrepancies noted. The strength and direction of associations were summarized qualitatively, with statistical measures reported where they added clarity. Evidence gaps and limitations were identified within each thematic domain and consolidated in a dedicated section.

Research Results

Epidemiological Associations Between Depression and Oral Health

Meta-analytic evidence demonstrates consistent associations between depression and adverse oral health outcomes [1,3,4]. Depression is associated with increased odds of dental caries, tooth loss, and edentulism [1]. Conversely, periodontal disease predicts the development of depression [1,7]. People with severe mental illness have nearly three times the odds of having lost all their teeth compared to the general community, and those with depression have between 1.17 and 1.32 times the odds [2].

The relationship between periodontal disease and emotional disorders is supported by multiple meta-analyses 3,16. Subjects with periodontal disease demonstrate higher depression scale scores and anxiety scale scores compared to periodontally healthy controls 3. However, one recent meta-analysis found no significant increased risk of depression among subjects with periodontal disease when analyzing pooled estimates from longitudinal studies, highlighting the need for cautious interpretation 17.

A dose-response relationship exists between oral health burden and depression severity 1,8. Individuals experiencing multiple concurrent oral health conditions demonstrate progressively higher odds of depression 8. Individuals with moderately severe to severe depressive symptoms demonstrate substantially higher odds of experiencing oral health problems that interfere with daily functioning, including difficulty obtaining needed dental care and job-related impairment due to oral pain 18.

Biological Pathways

HPA Axis Dysregulation and Cortisol

Salivary cortisol levels are significantly elevated in patients with periodontitis, with the highest concentrations observed in individuals experiencing concurrent psychological stress 19,20. The cortisol/dehydroepiandrosterone (DHEA) ratio, an indicator of chronic stress burden, demonstrates strong associations with periodontal disease severity 19. Cortisol levels increase with periodontal disease severity, being highest in the periodontitis group compared to gingivitis and healthy controls, and chromogranin A levels follow a similar pattern. Recent evidence demonstrates strong correlations between *Porphyromonas gingivalis* levels and salivary stress markers including lactoferrin, cortisol, and DHEA 19,21.

Cortisol and catecholamines possess immunomodulatory properties that can alter periodontal bacterial growth patterns and enhance the expression of virulence factors in periodontal pathogens 22,23. Psychosocial stress may contribute to a proinflammatory immunity that is implicated in periodontal disease pathobiology 23. However, prospective observational studies and Mendelian randomization (MR) analyses show mixed results regarding the causal role of cortisol in periodontitis pathology 24.

Nonsurgical periodontal treatment reduces gingival crevicular fluid cortisol levels in patients with chronic periodontitis, suggesting that periodontal inflammation contributes to local stress hormone elevation 25.

Systemic Inflammation and Neuroinflammation

Preclinical models combining experimental periodontitis with chronic mild stress demonstrate region-specific neuroinflammatory responses in the frontal cortex and hippocampus [26,27,28]. These regions exhibit increased expression of pro-inflammatory mediators in combined periodontitis-depression models, accompanied by microglial activation and morphological changes [26,28]. These microglial alterations include increased complexity and heterogeneity of parenchymal microglia, along with elevated expression of inducible nitric oxide synthase (iNOS), a marker of pro-inflammatory microglial phenotype [28].

In preclinical models, bacterial lipopolysaccharide (LPS) from periodontal pathogens such as *Porphyromonas gingivalis* and *Fusobacterium nucleatum* enters systemic circulation and can be transported to the brain via apolipoprotein A1 (APOA1)-mediated mechanisms [26]. Notably, *F. nucleatum* has been detected in brain parenchyma in preclinical models, suggesting direct bacterial translocation as an additional pathway [26].

Treatment of periodontitis reduces systemic inflammatory markers. Meta-analyses confirm reductions of high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), and plasma glucose after periodontal treatment [29,30]. Intensive nonsurgical periodontal therapy (NSPT) leads to significantly greater reductions in tumor necrosis factor-alpha (TNF-α), IL-6, and hs-CRP compared to standard therapy [31]. These findings support a causal association between periodontitis and systemic inflammation and suggest that periodontal treatment may attenuate the inflammatory pathways linking oral disease to depression [29,30].

Blood-Brain Barrier Disruption

In combined periodontitis-depression models, blood-brain barrier (BBB) integrity is significantly compromised, as evidenced by decreased expression of tight junction proteins including zonula occludens-1 (ZO-1) and occludin, alongside increased expression of adhesion molecules (intercellular adhesion molecule-1 (ICAM-1), vascular cell adhesion molecule-1 (VCAM-1), and matrix metalloproteinase 9 (MMP9) [27]. This BBB disruption facilitates the entry of peripheral inflammatory mediators and potentially periodontal pathogens into the central nervous system (CNS), amplifying neuroinflammatory responses [27].

Neuroplasticity and Neurotransmitter Alterations

Preclinical studies demonstrate decreased levels of brain-derived neurotrophic factor (BDNF) and synaptophysin in combined periodontitis-depression models. The proBDNF/mature BDNF (mBDNF) ratio becomes imbalanced, contributing to depressive-like

behavior 28. Intracellular signaling pathways critical for neuroplasticity, including phosphatidylinositol-3-kinase (PI3K)/protein kinase B (Akt) and mammalian target of rapamycin (mTOR), show reduced activity 28.

Endocannabinoid signaling is also disrupted, with decreased expression of endocannabinoid metabolic enzymes (N-acyl phosphatidylethanolamine-specific phospholipase D (NAPE-PLD), diacylglycerol lipase (DAGL), monoacylglycerol lipase (MAGL)) and cannabinoid receptor 1 (CB1), along with downstream signaling molecules (PI3K, Akt, extracellular signal-regulated kinase 1/2 (ERK1/2)) 28.

The Oral Microbiome-Brain Axis

Studies examining the salivary microbiome in individuals with depression demonstrate subtle but significant differences in alpha and beta diversity compared to healthy controls. A total of 21 bacterial taxa have been found to be differentially abundant in depressed cohorts, including increased *Neisseria* spp. and *Prevotella nigrescens* 32.

Large-scale population-based studies confirm that mental and periodontal health variables influence the overall composition of the oral microbiome 33. Posttraumatic stress disorder (PTSD) symptoms correlate with altered abundance of specific bacterial species, and functional prediction analysis reveals a potential role for tryptophan metabolism/degradation in the oral-brain axis, confirmed by lower plasma serotonin levels across symptomatic groups 33. Oral microbiota dysbiosis may contribute to depression through immune, inflammatory, and neuroactive pathways 34,35. Preclinical evidence demonstrates that oral microbiota dysbiosis alters chronic restraint stress-induced depression-like behaviors by modulating host metabolism 36.

Behavioral Mechanisms

Oral Hygiene and Self-Care

Reduced toothbrushing frequency serves as a significant mediator of the systemic determinants of relationship between depressive symptoms and dental caries 1,4. Anhedonia directly undermines motivation for self-care behaviors, while psychomotor retardation reduces the physical capacity to perform routine oral care tasks 4. Qualitative research reveals that oral care is often among the first self-care activities to be neglected in individuals with depression 4,37. Depression is frequently associated with disinterest in performing appropriate oral hygiene techniques, cariogenic diet, and diminished salivary flow 2,4.

Dietary Patterns

High sugar intake, associated with depressive symptoms, represents both a coping mechanism and a consequence of altered reward processing in depression. Individuals with depression may consume more processed foods high in refined carbohydrates while reducing intake of nutrient-dense foods, creating a dietary pattern that promotes both systemic inflammation and dental caries 1,4.

Healthcare-Seeking Behaviors

Individuals with moderately severe to severe depressive symptoms are substantially less likely to obtain needed oral health care despite experiencing greater oral health needs [](https://pubmed.ncbi.nlm.nih.gov/41201412)9,18[](https://pubmed.ncbi.nlm.nih.gov/37246825). Dental phobia and anxiety, which frequently co-occur with depression, create additional barriers to accessing dental care 4,38. The combination of depression and dental anxiety can result in avoidance of dental services until oral health problems become severe 4.

Pharmacological Factors

Xerostomia Prevalence and Risk

Xerostomia (dry mouth) is reported in 91% of psychotropic medications across all classes 39. Serotonin-norepinephrine reuptake inhibitors (SNRIs) are associated with a significantly greater risk of dry mouth compared to selective serotonin reuptake inhibitors (SSRIs) 40. Tricyclic antidepressants (TCAs) demonstrate the highest risk of dry mouth 39,40. Network meta-analyses reveal that duloxetine, levomilnacipran, and vilazodine carry higher risk of inducing dry mouth, whereas trazodone, amitriptyline, agomelatine, and mirtazapine tend to be better tolerated 40.

Mechanisms of Salivary Dysfunction

TCAs exert antimuscarinic effects at the glandular level, directly inhibiting methacholine-evoked salivary secretion. In contrast, SSRIs and SNRIs primarily affect salivary secretion through central inhibition of the salivary reflex, reducing citric acid-evoked secretion by 40--60% without direct antimuscarinic effects at salivary glands 41. While all three antidepressant types inhibit reflex-evoked secretion, only TCAs inhibit methacholine-evoked secretion, whereas SSRIs and SNRIs actually increase methacholine-evoked secretion, particularly salivary protein output.41

Specific Antidepressant Profiles and Oral Consequences

Among commonly prescribed antidepressants, fluoxetine demonstrates relatively lower prevalence of dry mouth compared to other agents [42]. Sertraline is associated with higher oral side effects and should be avoided when oral health is a primary concern [39]. Dysgeusia (taste disturbance) affects approximately 65% of antidepressants [39,42]. Systematic reviews demonstrate consistent associations between antidepressant use and increased risk of dental caries and periodontal disease [39]. SSRIs have been linked with higher implant failure, potentially through their anabolic effect on bone, reducing turnover [2].

Psychosocial Impact

Oral Health as a Predictor of Depression

Beyond cross-sectional associations, longitudinal evidence demonstrates that periodontal disease and edentulism predict incident depression [1,7]. A recent scoping review identified that depressive and anxiety disorders were linked bidirectionally with TMD and eating disorders [6]. The dose-response relationship between oral health burden and depression severity provides additional evidence for causality [1].

Functional and Aesthetic Mediators

Mediation analyses reveal that difficulty chewing accounts for approximately one-fifth of the relationship between tooth loss and depression, with difficulty speaking and problems with smiling also contributing substantially [1]. Oral health-related quality of life (OHRQoL) demonstrates moderate positive correlations with both depression and anxiety, and poorer OHRQoL significantly predicts incident depression [1,43,44].

Genetic Evidence

Mendelian randomization studies demonstrate associations between mouth ulcers and both MDD and bipolar disorder (BIP), as well as between MDD and periodontitis [45]. However, socioeconomic status represents an important confounding factor, with poverty explaining the majority of the observed association between depression and periodontitis [46].

Barriers and Facilitators to Integrated Care

Qualitative research has identified key barriers for maintaining oral health among individuals with severe mental illness, including impact of mental ill-health, lack of patient involvement and tailored approach, and accessibility and availability of dental services. The

main facilitators identified include service providers' effective communication skills and further support through the involvement of carers 37.

From multiple perspectives (patients, psychiatrists, dentists), barriers include access to dental care, fear of dental care, characteristics of mental illness, lack of oral health screening by psychiatrists, lack of education and training, stigma of mental illness, and lack of communication. Facilitators are linked to the need for education and training, financial support, dentists' chairside manner, community support, and interprofessional communication 38.

Four types of oral health interventions in mental health have been identified:

I. educational interventions;

II. (II) physical interventions;

III. (III) interventions combining behavioral and educational elements; and

IV. (IV) interventions combining educational and physical elements.

Studies demonstrate positive effects on oral health knowledge, oral health behavior, and physical oral health outcomes in different diagnostic patient groups 47.

Learning collaboratives have demonstrated that virtual interprofessional education is an accessible and productive avenue to improve interprofessional education and achieve practical progress in integrated care 48. Sites participating in such collaboratives displayed improvement in the percentages of patients screened and referred 48.

Discussion

Synthesis of Biological Evidence

The biological pathways linking depression and periodontal disease converge on a shared inflammatory substrate [](https://pubmed.ncbi.nlm.nih.gov/30072865)5,11. The hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic-adrenal-medullary (SAM) system represent the primary neuroendocrine pathways through which chronic stress, a hallmark of depression, influences periodontal disease susceptibility 22,23. Activation of these systems triggers sustained cortisol elevation, which possesses immunomodulatory properties that alter periodontal bacterial growth patterns and enhance virulence factor expression 22. The "cytokine hypothesis" of depression posits that peripheral inflammation, including that originating from periodontitis, increases CNS pro-inflammatory cytokines (TNF-α, interleukin-1 beta (IL-1β), nuclear factor kappa B (NF-κB)), thereby contributing to depressive symptomatology [](https://pubmed.ncbi.nlm.nih.gov/30072865)5,11.

The preclinical evidence demonstrating BBB disruption, microglial activation, and neuroplasticity alterations in combined periodontitis-depression models provides mechanistic plausibility for the epidemiological associations observed 26,27,28. The detection of *F. nucleatum* in brain parenchyma suggests that direct bacterial translocation may represent an additional pathway beyond cytokine-mediated neuroinflammation 26. The emerging evidence on the oral microbiome-brain axis adds a further dimension, with oral microbiota dysbiosis potentially contributing to depression through immune, inflammatory, and neuroactive pathways 32,33,35.

Importantly, the finding that periodontal treatment reduces systemic inflammatory markers (hs-CRP, IL-6, TNF-α) supports the hypothesis that periodontitis is a modifiable contributor to systemic inflammation 29,30,31. If periodontitis contributes to depression through inflammatory pathways, then periodontal treatment may represent a novel adjunctive approach to depression management, though this hypothesis requires direct testing in clinical trials 11.

Behavioral and Pharmacological Considerations

The behavioral mechanisms linking depression to poor oral health are clinically intuitive but empirically well-supported 1,4. The core symptoms of depression-anhedonia, fatigue, psychomotor retardation, and cognitive impairment-create cascading barriers to oral self-care that extend from reduced brushing frequency to dietary deterioration and avoidance of dental services 4,9,18. The paradox of increased oral health need coupled with decreased healthcare utilization in depressed individuals represents a critical clinical challenge 18.

Pharmacological factors compound these behavioral risks 39,40,49. The high prevalence of xerostomia across antidepressant classes creates an iatrogenic pathway to dental caries and periodontal disease that is often overlooked in prescribing decisions 39. The mechanistic differences between TCA-induced xerostomia (peripheral anticholinergic) and SSRI/SNRI-induced xerostomia (central inhibition of salivary reflex) have practical implications for management strategies 41. Clinicians should consider oral side effect profiles when selecting antidepressants, particularly for patients with pre-existing oral health concerns 39,49.

The Role of Socioeconomic Confounding

A critical finding from the current evidence is the substantial role of socioeconomic status in mediating the depression-periodontitis relationship. The observation that poverty explains the majority of the observed association challenges simplistic causal models and highlights the importance of addressing social determinants of health 46. Both depression and periodontal

disease share common upstream risk factors including low income, limited education, food insecurity, and restricted access to healthcare [](https://pubmed.ncbi.nlm.nih.gov/41201872)23,46. This does not negate the biological and behavioral pathways described above but rather contextualizes them within a broader socioeconomic framework that must inform both research design and clinical intervention 46.

Clinical Implications for Integrated Care

The convergence of biological, behavioral, pharmacological, and psychosocial evidence supports the need for integrated care models that bridge dental and mental health services 2,4,37. Both mental and oral health rank among the leading causes of disability worldwide, yet clinical care typically addresses these domains in isolation 1,2.

Dental practices represent viable venues for mental health screening, as many individuals access dental care more regularly than mental health services. Standardized screening tools can be efficiently integrated into dental intake procedures 2,4. Conversely, standard oral health checklists can be completed by non-dental personnel in mental health settings, enabling systematic screening without requiring dental expertise 4,50.

Effective integration requires systematic approaches to interprofessional collaboration 37,38,51. Education and training represent foundational elements, with mental health professionals requiring oral health education and dental professionals needing mental health literacy and communication skills training. Communication protocols must be established to enable information sharing between dental and mental health providers 37,38.

Behavioral interventions should employ evidence-based behavior change techniques, including clear instructions, demonstrations of proper oral hygiene techniques, and involvement of carers or care coordinators 37,47,51. Pharmacological management should address iatrogenic xerostomia through saliva substitutes and anticaries agents containing fluoride 4,39. Prevention should be a priority, including the promotion of dental care and the management of xerostomia when psychopharmacologic agents are prescribed 2,4.

At the systems level, service integration models that co-locate dental and mental health care, establish formal referral networks, and create shared care protocols can reduce fragmentation 37,48,51. Policy implications include the need for reimbursement structures that support integrated care models and public health initiatives that address the social determinants underlying both depression and poor oral health 2,46.

Evidence Gaps and Limitations

Several important gaps in the current evidence base warrant attention:

1. **Causality and Temporality:** While meta-analyses demonstrate consistent associations between depression and oral diseases, the cross-sectional design of most included studies limits causal inference. More longitudinal studies are required to test causal and temporal relationships 1,4.

2. **Heterogeneity:** High degrees of heterogeneity among studies should be considered when interpreting pooled estimates. Findings for periodontal disease are more equivocal than for dental caries, possibly because of study heterogeneity. One meta-analysis found no significant increased risk of depression among subjects with periodontal disease, contrasting with other pooled analyses 2,3,17.

3. **Intervention Trials:** There is a paucity of randomized controlled trials testing integrated care models for individuals with comorbid depression and oral disease. Similarly, no clinical trials have directly tested whether periodontal treatment improves depression outcomes 47.

4. **Mechanistic Translation:** The majority of mechanistic evidence derives from preclinical animal models. Clinical studies are needed to validate these pathways in human populations and quantify the relative contribution of each biological mechanism 26,27,28.

5. **Population Diversity:** Mendelian randomization studies are limited to European populations, restricting generalizability. More diverse population-based studies are needed 45.

6. **Socioeconomic Confounding:** The substantial role of socioeconomic status in mediating the depression-periodontitis relationship highlights the need for studies that adequately control for social determinants of health 46.

7. **Oral Microbiome Research:** While emerging evidence supports the oral-brain axis, further studies are warranted to investigate whether shifts in oral microbiota play a role in the underlying etiology of depression and to develop microbiome-based therapeutic strategies 32,33,35

Conclusions

The relationship between depression and oral health is bidirectional and mechanistically complex, involving neuroendocrine dysregulation, systemic inflammation, behavioral factors,

pharmacological effects, and psychosocial consequences 1,3,5,11. The bidirectional nature of this relationship with depression increasing risk for oral disease and poor oral health predicting depression creates opportunities for integrated interventions that address both conditions simultaneously 1,6.

Biological mechanisms linking depression and periodontal disease include HPA axis activation with cortisol elevation, systemic inflammation with neuroinflammatory consequences, BBB disruption, and alterations in neuroplasticity and neurotransmitter systems [](https://pubmed.ncbi.nlm.nih.gov/41289041)11,19,22,26,27,28. Treatment of periodontitis reduces systemic inflammatory markers, supporting the hypothesis that periodontal disease is a modifiable contributor to systemic inflammation and potentially to depression 29,30.

Behavioral mechanisms, including depression-related neglect of oral hygiene, cariogenic dietary patterns, and reduced dental service utilization, translate psychological symptoms into tangible oral health consequences 1,4,9,18. Pharmacological factors, particularly antidepressant-induced xerostomia, create additional risk for dental caries and periodontal disease 39,40. Psychosocial impacts, including functional impairments and aesthetic concerns related to poor oral health, can precipitate or exacerbate depression, perpetuating a bidirectional cycle 1,18,43.

Clinical implications emphasize the need for integrated care models that bridge dental and mental health services. 2,4,37. Mental health screening in dental settings and oral health screening in mental health settings can facilitate early identification of comorbidities 2,4. Interprofessional collaboration, supported by cross-disciplinary education, systematic communication protocols, and tailored care plans, represents the foundation for effective integrated care 37,38,48,51.

Future research should prioritize longitudinal studies establishing causal and temporal relationships, intervention trials testing whether periodontal treatment improves depression outcomes, clinical validation of preclinical mechanistic findings, and investigation of the oral microbiome-brain axis 3,6,32,35. From a public health perspective, addressing socioeconomic factors that mediate the depression-oral health relationship is essential for achieving health equity 46.

Recognition of the profound interconnections between oral and mental health should inform clinical practice, health policy, and resource allocation 1,2. By adopting integrated approaches that address both domains simultaneously, healthcare systems can reduce the substantial burden of these comorbid conditions and improve outcomes for affected individuals 1,2,9.

Disclosure

Author Contributions

Conceptualization: Z.S., M.S., N.S., J.S., P.M., S.P. and J.P.; Formal analysis: Z.S., M.S., N.S., J.S., P.M., S.P. and J.W.; Investigation: Z.S., J.S., P.M., S.P. and J.P.; Writing rough preparation: Z.S., M.S., N.S., J.S., P.M., S.P. and J.W.; Writing review and editing: Z.S., M.S., N.S., J.S., P.M., S.P., J.P. and J.W.; Supervision: N.S., J.P.and J.W.

All authors have read and agreed to the published version of the manuscript.

AI Statement

Artificial intelligence tools were used exclusively for linguistic refinement, grammar correction, stylistic improvement, and organizational assistance under full human supervision. Specifically, the paid version of ChatGPT (OpenAI) and Grammarly (https://www.grammarly.com) were utilized to support academic English editing, improve clarity and readability of the manuscript, and assist with language refinement. No AI tools were used for data fabrication, generation of scientific results, interpretation of findings, or autonomous scientific decision-making. All scientific analysis, interpretation of the literature, conclusions, and final editorial decisions were performed solely by the authors, who take full responsibility for the content of the manuscript.

Funding: This study received no external funding

Institutional Review Board Statement: Not applicable.

Informed Consent Statement Not applicable.

Data Availability Statement The original contributions presented in the study are included in the article, further inquiries can be directed to the corresponding authors.

Conflicts of Interest: The authors declare no conflicts of interest.

References

1. Cademartori MG, Gastal MT, Nascimento GG, Demarco FF, Corrêa MB. Is depression associated with oral health outcomes in adults and elders? A systematic review and meta-analysis. *Clin Oral Investig*. 2018 Nov;22(8):2685-2702. doi: 10.1007/s00784-018-2611-y.](https://pubmed.ncbi.nlm.nih.gov/30191327/) https://pubmed.ncbi.nlm.nih.gov/30191327/

2. Choi J, Price J, Ryder S, Siskind D, Solmi M, Kisely S. Prevalence of dental disorders among people with mental illness: An umbrella review. *Aust N Z J Psychiatry*. 2022 Aug;56(8):949-963. doi: 10.1177/00048674211042239. Epub 2021 Aug 30. PMID: 34461748

3. Zheng DX, Kang XN, Wang YX, Huang YN, Pang CF, Chen YX, Kuang ZL, Peng Y. Periodontal disease and emotional disorders: A meta-analysis. *J Clin Periodontol*. 2021 Feb;48(2):180-204. doi: 10.1111/jcpe.13395. Epub 2020 Nov 11. PMID: 33103263

4. Kisely S, Sawyer E, Siskind D, Lalloo R. *The oral health of people with anxiety and depressive disorders - a systematic review and meta-analysis. J Affect Disord*. 2016 Aug;200:119-32. doi: 10.1016/j.jad.2016.04.040. Epub 2016 Apr 21. PMID: 27130961

5. Martínez M, Postolache TT, García-Bueno B, Leza JC, Figuero E, Lowry CA, Malan-Müller S. The Role of the Oral Microbiota Related to Periodontal Diseases in Anxiety, Mood and Trauma- and Stress-Related Disorders. *Front Psychiatry*. 2022 Jan 27;12:814177. doi: 10.3389/fpsyt.2021.814177. PMID: 35153869; PMCID: PMC8833739

6. Neupane SP, Onyeka IN, Bull VH, Daray FM. Associations between diseases of the mouth and mental disorders: A scoping review of longitudinal studies. *Neurobiol Dis*. 2026 Mar;220:107285. doi: 10.1016/j.nbd.2026.107285. Epub 2026 Jan 26. PMID: 41605332

7. Hsu CC, Hsu YC, Chen HJ, Lin CC, Chang KH, Lee CY, Chong LW, Kao CH. Association of Periodontitis and Subsequent Depression: A Nationwide Population-Based Study. *Medicine (Baltimore)*. 2015 Dec;94(51):e2347. doi: 10.1097/MD.0000000000002347. PMID: 26705230; PMCID: PMC4697996

8. O'Neil A, Berk M, Venugopal K, Kim SW, Williams LJ, Jacka FN. The association between poor dental health and depression: findings from a large-scale, population-based study (the NHANES study). *Gen Hosp Psychiatry*. 2014 May-Jun;36(3):266-70. doi: 10.1016/j.genhosppsych.2014.01.009. Epub 2014 Jan 31. PMID: 24636212

9. Kalaigian A, Chaffee BW. *Mental Health and Oral Health in a Nationally Representative Cohort. J Dent Res*. 2023 Aug;102(9):1007-1014. doi: 10.1177/00220345231171108. Epub 2023 May 29. PMID: 37246825; PMCID: PMC10403957

10. Katebi K, Alamdari SS, Mahboobi Z, Faramarzi E, Sharififard N. Depression and Untreated Dental Caries Among the Azar Cohort Population: A Cross-Sectional Study. *Int Dent J*. 2025 Oct;75(5):100885. doi: 10.1016/j.identj.2025.100885. Epub 2025 Jul 11. PMID: 40651327; PMCID: PMC12275032

11. Hashioka S, Inoue K, Hayashida M, Wake R, Oh-Nishi A, Miyaoka T. *Implications of Systemic Inflammation and Periodontitis for Major Depression. Front Neurosci*. 2018 Jul 18;12:483. doi: 10.3389/fnins.2018.00483. PMID: 30072865; PMCID: PMC6058051

12. Henry BM, Skinningsrud B, Vikse J, Pękala PA, Walocha JA, Loukas M, Tubbs RS, Tomaszewski KA. Systematic reviews versus narrative reviews in clinical anatomy: Methodological approaches in the era of evidence-based anatomy. *Clin Anat*. 2018 Apr;31(3):364-367. doi: 10.1002/ca.23042. Epub 2018 Jan 25. PMID: 29322553

13. Gasparyan AY, Ayvazyan L, Blackmore H, Kitas GD. Writing a narrative biomedical review: considerations for authors, peer reviewers, and editors. *Rheumatol Int*. 2011 Nov;31(11):1409-17. doi: 10.1007/s00296-011-1999-3. Epub 2011 Jul 29. PMID: 21800117

14. Parums DV. Editorial: Review Articles, Systematic Reviews, Meta-Analysis, and the Updated Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 Guidelines. *Med Sci Monit*. 2021 Aug 23;27:e934475. doi: 10.12659/MSM.934475. PMID: 34421116; PMCID: PMC8394590

15. Siddaway AP, Wood AM, Hedges LV. How to Do a Systematic Review: A Best Practice Guide for Conducting and Reporting Narrative Reviews, Meta-Analyses, and Meta-Syntheses. *Annu Rev Psychol*. 2019 Jan 4;70:747-770. doi: 10.1146/annurev-psych-010418-102803. Epub 2018 Aug 8. PMID: 30089228

16. Liu F, Wen YF, Zhou Y, Lei G, Guo QY, Dang YH. *A meta-analysis of emotional disorders as possible risk factors for chronic periodontitis. Medicine (Baltimore)*. 2018 Jul;97(28):e11434. doi: 10.1097/MD.0000000000011434. Erratum in: Medicine (Baltimore). 2023 Feb 3;102(5):e32891. doi: 10.1097/MD.0000000000032891. PMID: 29995795; PMCID: PMC6076092

17. Dibello V, Custodero C, Cavalcanti R, Lafornara D, Dibello A, Lozupone M, Daniele A, Pilotto A, Panza F, Solfrizzi V. Impact of periodontal disease on cognitive disorders, dementia, and depression: a systematic review and meta-analysis. *Geroscience*. 2024 Oct;46(5):5133-5169. doi: 10.1007/s11357-024-01243-8. Epub 2024 Jun 28. PMID: 38943006; PMCID: PMC11336026

18. Llaneza AJ, Zetino Y, Llaneza DH, Seward J. Investigating the relationship between depression and oral health among adults in the United States, 2017-2020. *J Am Dent Assoc*. 2026 Feb;157(2):153-162. doi: 10.1016/j.adaj.2025.09.008. Epub 2025 Nov 7. PMID: 41201412

19. Lee YH, Suk C, Shin SI, Hong JY. Salivary cortisol, dehydroepiandrosterone, and chromogranin A levels in patients with gingivitis and periodontitis and a novel biomarker for psychological stress. *Front Endocrinol (Lausanne)*. 2023 Apr 11;14:1147739. doi: 10.3389/fendo.2023.1147739. PMID: 37113482; PMCID: PMC10126469

20. Zhang H, Chen B, Pan C, Zhang A. To evaluate the serum cortisol, salivary cortisol, and serum interleukin-1 B level in patients of chronic periodontitis with smoking and stress and without smoking and stress. *Medicine (Baltimore)*. 2021 Aug 6;100(31):e26757. doi: 10.1097/MD.0000000000026757. PMID: 34397819; PMCID: PMC8341332

21. Antequera D, Buetas E, García-Esteban S, Romualdi D, Carrero L, Municio C, Mira A, Carro E. Association of salivary lactoferrin, *Porphyromoras gingivalis* and stress hormone levels in patients with periodontitis: a pilot study. Front Immunol. 2025 Sep 23;16:1681095. doi: 10.3389/fimmu.2025.1681095. PMID: 41063989; PMCID: PMC12500692

22. Skrypnyk M, Spahr A, Berkovsky S, Yatsenko T, Xu C, Zuieva O, Osada T, Takahashi S, Hattori N, Takahashi K, Hattori K, Heissig B. *Chronic stress and the IL-10-mediated immunoregulatory loop in the pathogenesis of periodontitis. Clin Sci (Lond)*. 2025 Nov 25;139(22):1469--87. doi: 10.1042/CS20256843. PMID: 41289041; PMCID: PMC12751072

23. Gomaa N, Glogauer M, Nicolau B, Tenenbaum H, Siddiqi A, Fine N, Quiñonez C. Stressed-Out Oral Immunity: A Gateway From Socioeconomic Adversity to Periodontal Disease. *Psychosom Med*. 2020 Feb/Mar;82(2):126-137. doi: 10.1097/PSY.0000000000000774. PMID: 31860530

24. Baumeister SE, Reckelkamm SL, Grabe HJ, Nauck M, Klinger-König J, Völzke H, Kocher T, Friedrich N, Holtfreter B. Cortisol and periodontitis: Prospective observational and Mendelian

randomization studies. *Front Endocrinol (Lausanne)*. 2023 Mar 15;14:1100985. doi: 10.3389/fendo.2023.1100985. PMID: 37008927; PMCID: PMC10050732

25. Cakmak O, Alkan BA, Saatci E, Tasdemir Z. The effect of nonsurgical periodontal treatment on gingival crevicular fluid stress hormone levels: A prospective study. *Oral Dis*. 2019 Jan;25(1):250-257. doi: 10.1111/odi.12973. Epub 2018 Sep 27. PMID: 30187981

26. Martínez M, Martín-Hernández D, Virto L, MacDowell KS, Montero E, González-Bris Á, Marín MJ, Ambrosio N, Herrera D, Leza JC, Sanz M, García-Bueno B, Figuero E. Periodontal diseases and depression: A pre-clinical in vivo study. *J Clin Periodontol*. 2021 Apr;48(4):503-527. doi: 10.1111/jcpe.13420. Epub 2021 Feb 9. PMID: 33432590

27. Martín-Hernández D, Martínez M, Robledo-Montaña J, Muñoz-López M, Virto L, Ambrosio N, Marín MJ, Montero E, Herrera D, Sanz M, Leza JC, Figuero E, García-Bueno B. *Neuroinflammation related to the blood-brain barrier and sphingosine-1-phosphate in a pre-clinical model of periodontal diseases and depression in rats. J Clin Periodontol*. 2023 May;50(5):642-656. doi: 10.1111/jcpe.13780. Epub 2023 Jan 27. PMID: 36644813

28. Robledo-Montaña J, Díaz-García C, Martínez M, Ambrosio N, Montero E, Marín MJ, Virto L, Muñoz-López M, Herrera D, Sanz M, Leza JC, García-Bueno B, Figuero E, Martín-Hernández D. *Microglial morphological/inflammatory phenotypes and endocannabinoid signaling in a preclinical model of periodontitis and depression. J Neuroinflammation*. 2024 Sep 8;21(1):219. doi: 10.1186/s12974-024-03213-5. PMID: 39245706; PMCID: PMC11382403

29. Orlandi M, Muñoz Aguilera E, Marletta D, Petrie A, Suvan J, D'Aiuto F. Impact of the treatment of periodontitis on systemic health and quality of life: A systematic review. *J Clin Periodontol*. 2022 Jun;49 Suppl 24:314-327. doi: 10.1111/jcpe.13554. Epub 2021 Nov 17. PMID: 34791686

30. Luthra S, Orlandi M, Hussain SB, Leira Y, Botelho J, Machado V, Mendes JJ, Marletta D, Harden S, D'Aiuto F. Treatment of periodontitis and C-reactive protein: A systematic review and meta-analysis of randomized clinical trials. *J Clin Periodontol*. 2023 Jan;50(1):45-60. doi: 10.1111/jcpe.13709. Epub 2022 Aug 29. PMID: 35946825; PMCID: PMC10087558

31. Cardisciani M, Di Nicolantonio S, Altamura S, Ortu E, Del Pinto R, Pietropaoli D. Temporal dynamics of early inflammatory markers after professional dental cleaning: a meta-analysis and spline-based meta-regression of TNF-α, IL-1β, IL-6, and (hs)CRP. *Front Immunol*. 2025 Aug 28;16:1634622. doi: 10.3389/fimmu.2025.1634622. PMID: 40948802; PMCID: PMC12423065

32. Wingfield B, Lapsley C, McDowell A, Miliotis G, McLafferty M, O'Neill SM, Coleman S, McGinnity TM, Bjourson AJ, Murray EK. *Variations in the oral microbiome are associated with depression in young adults. Sci Rep*. 2021 Jul 22;11(1):15009. doi: 10.1038/s41598-021-94498-6. PMID: 34294835; PMCID: PMC8298414

33. Malan-Müller S, Vidal R, O'Shea E, Montero E, Figuero E, Zorrilla I, de Diego-Adeliño J, Cano M, García-Portilla MP, González-Pinto A, Leza JC. Probing the oral-brain connection: oral microbiome patterns in a large community cohort with anxiety, depression, and trauma symptoms, and periodontal outcomes. *Transl Psychiatry*. 2024 Oct 5;14(1):419. doi: 10.1038/s41398-024-03122-4. PMID: 39368974; PMCID: PMC11455920

34. Liu F, Yang Y, Zhang N, Wang S, Wang CX. *The association of depression and oral microbiota. Sci Rep*. 2025 Nov 27;15(1):42434. doi: 10.1038/s41598-025-26526-8. PMID: 41309831; PMCID: PMC12661023

35. Rafique QT, Gogoi V, Barah P. Beyond the Gut: Integrating Oral Microbiota into the Microbiota-Brain Axis in Depression. *Mol Neurobiol*. 2025 Nov 29;63(1):207. doi: 10.1007/s12035-025-05518-9. PMID: 41317233

36. Lou F, Luo S, Kang N, Yan L, Long H, Yang L, Wang H, Liu Y, Pu J, Xie P, Ji P, Jin X. *Oral microbiota dysbiosis alters chronic restraint stress-induced depression-like behaviors by modulating host metabolism. Pharmacol Res*. 2024 Jun;204:107214. doi: 10.1016/j.phrs.2024.107214. Epub 2024 May 17. PMID: 38763328

37. Mishu MP, Faisal MR, Macnamara A, Sabbah W, Peckham E, Newbronner L, Gilbody S, Gega L. A Qualitative Study Exploring the Barriers and Facilitators for Maintaining Oral Health and Using Dental Service in People with Severe Mental Illness: Perspectives from Service Users and Service Providers. *Int J Environ Res Public Health*. 2022 Apr 5;19(7):4344. doi: 10.3390/ijerph19074344. PMID: 35410025; PMCID: PMC8998854

38. Wright WG, Averett PE, Benjamin J, Nowlin JP, Lee JGL, Anand V. Barriers to and Facilitators of Oral Health Among Persons Living With Mental Illness: A Qualitative Study. *Psychiatr Serv*. 2021 Feb 1;72(2):156-162. doi: 10.1176/appi.ps.201900535. Epub 2020 Nov 25. PMID: 33234051

39. Alcázar-Hernández JM, Pecci-Lloret MR, Guerrero-Gironés J. Oral Manifestations in Patients in Treatment with Antidepressants: A Systematic Review. *J Clin Med*. 2024 Nov 18;13(22):6945. doi: 10.3390/jcm13226945. PMID: 39598089; PMCID: PMC11594724

40. Cappetta K, Beyer C, Johnson JA, Bloch MH. Meta-analysis: Risk of dry mouth with second generation antidepressants. *Prog Neuropsychopharmacol Biol Psychiatry*. 2018 Jun 8;84(Pt A):282-293. doi: 10.1016/j.pnpbp.2017.12.012. Epub 2017 Dec 20. PMID: 29274375

41. Johnsson M, Winder M, Zawia H, Lödöen I, Tobin G, Götrick B. *In vivo studies of effects of antidepressants on parotid salivary secretion in the rat. Arch Oral Biol*. 2016 Jul;67:54-60. doi: 10.1016/j.archoralbio.2016.03.010. Epub 2016 Mar 19. PMID: 27023402

42. Sarrión Pérez MG, Jiménez Y, Bagán L, Bagán J. Oral side effects of fluoxetine in patients with depressive disorder: A systematic review. *Med Oral Patol Oral Cir Bucal*. 2025 May 1;30(3):e373-e382. doi: 10.4317/medoral.26947. PMID: 39954277; PMCID: PMC12019662

43. Alsaif MI, Bin Rahmah AS, Naser AY. Exploring the link between depression, anxiety, and oral health-related quality of life: A cross-sectional study in 2 Arab countries. *Medicine (Baltimore)*. 2026 Jan 2;105(1):e46968. doi: 10.1097/MD.0000000000046968. PMID: 41496080; PMCID: PMC12778151

44. Xin T, Hu F, Xu L, Kong F, Yang H, Li L, Qin A, Bao B, Chen C, Lan J. *Association and mediators between the impact of oral health-related quality of life and depression. Oral Dis*. 2024 Sep;30(6):4001-4010. doi: 10.1111/odi.14832. Epub 2023 Dec 9. PMID: 38071189

45. Pi Y, Jiao Z, Wang L, Zhao Y, Hu W, Chen F, Yu J, Zhang X, Zhao P, Jiang H, Zhang M, Wang F. Genetic evidence strengthens the bidirectional connection between oral health status and psychiatric disorders: A two-sample Mendelian randomization study. *J Affect Disord*. 2024 Apr 15;351:661-670. doi: 10.1016/j.jad.2024.01.232. Epub 2024 Feb 1. PMID: 38309483

46. Jungo S, Smail-Faugeron V, Guez D, Hoertel N, Lemogne C, Carra MC. *Cross-sectional analysis of role of socioeconomic status in the association between depression and periodontitis. J Periodontol*. 2025 Nov 7. doi: 10.1002/jper.70012. Epub ahead of print. PMID: 41201872

47. Kuipers S, Boonstra N, Kronenberg L, Keuning-Plantinga A, Castelein S. Oral Health Interventions in Patients with a Mental Health Disorder: A Scoping Review with Critical Appraisal of the Literature. *Int J Environ Res Public Health*. 2021 Jul 30;18(15):8113. doi: 10.3390/ijerph18158113. PMID: 34360406; PMCID: PMC8346069

48. Cardenas K, Weilnau T, Aguilar C, Ali A, Eidelman A, Ponnala S, Russel T, Schwanderla J, Sievers K, Wu H, Silk H, Hunt LS, Barrow J, Riedy CA, Phillips RS. Partnering for Integrated Care: A Learning Collaborative for Primary Care and Oral Health Teams. *Ann Fam Med*. 2023 Feb;21(Suppl 2):S22-S30. doi: 10.1370/afm.2918. PMID: 36849470; PMCID: PMC9970677

49. Cockburn N, Pradhan A, Taing MW, Kisely S, Ford PJ. *Oral health impacts of medications used to treat mental illness. J Affect Disord*. 2017 Dec 1;223:184-193. doi: 10.1016/j.jad.2017.07.037. Epub 2017 Jul 23. PMID: 28759866

50. Khokhar MA, Khokhar WA, Clifton AV, Tosh GE. Oral health education (advice and training) for people with serious mental illness. Cochrane Database of Systematic Reviews 2016, Issue 9. Art. No.: CD008802. DOI: 10.1002/14651858.CD008802.pub3. Accessed 06 May 2026

51. Mishu MP, Faisal MR, Macnamara A, Sabbah W, Peckham E, Newbronner L, Gilbody S, Gega L. Exploring the contextual factors, behaviour change techniques, barriers and facilitators of interventions to improve oral health in people with severe mental illness: A qualitative study. *Front Psychiatry*. 2022 Oct 11;13:971328. doi: 10.3389/fpsyt.2022.971328. PMID: 36304558; PMCID: PMC9592713