

Review

The Neurocognitive Basis of Oral Health Decline in Schizophrenia: From Functional Impairment to Prevention

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Abstract

Background: Schizophrenia is strongly associated with severe oral health deterioration, driven by cognitive deficits, behavioral dysfunction, and medication-related biological changes. **Objective:** To examine how neurocognitive dysfunction in schizophrenia, particularly cognitive deficits, is associated with poorer oral hygiene control, motivation, and self-regulation, contributes to oral health decline by disrupting everyday oral hygiene behaviors and dental care engagement, and to discuss the implications of this framework for interdisciplinary prevention strategies. **Methods:** This manuscript follows a narrative review design aimed at conceptually integrating evidence on neurocognitive mechanisms underlying oral health decline in schizophrenia. To identify relevant literature, a targeted search of PubMed/MEDLINE, Scopus, and Web of Science was conducted, covering publications from 2000 to 2025. The search strategy was used to support thematic exploration and conceptual synthesis, rather than to perform a systematic study selection or quantitative evidence aggregation. This narrative review summarizes findings from 90 peer-reviewed studies selected from the available literature. **Results:** Executive dysfunction, attentional deficits, and low motivation impair routine oral hygiene and delay dental care-seeking. Antipsychotic-induced xerostomia, metabolic disturbances, oxidative stress, immune dysregulation, and oral microbiome dysbiosis accelerate periodontal breakdown and caries progression. These interacting processes generate a self-reinforcing cycle of inflammation, tissue destruction, and treatment avoidance. Epidemiological data show markedly elevated DMFT/DMFS indices and up to a three-fold higher risk of edentulism compared with the general population. Emerging evidence suggests that integrated psychiatric–dental care models may be associated with improvements in oral health and care engagement, although current findings are largely preliminary and based on small or heterogeneous study populations, including related neurocognitive disorders. **Conclusions:** Unlike existing epidemiological syntheses, this review highlights oral health deterioration in schizophrenia as a functionally mediated consequence of neurocognitive impairment, underscoring the need for preventive approaches aligned with patients’ cognitive and motivational capacities.

Keywords: schizophrenia; oral health; neurocognitive dysfunction; interdisciplinary care; antipsychotic medication



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1. Introduction

People with schizophrenia experience long-term disturbances in perception and thought processes with consequences for regulatory control and behavior. More than 20 million individuals worldwide are affected, making schizophrenia one of the leading contributors to chronic disability globally. While extensive research has focused on core

psychopathological dimensions—positive symptoms, negative symptoms, and cognitive impairment—an under-recognized but clinically consequential aspect of the disorder is its impact on oral health and functional self-care behaviors.

A growing body of evidence indicates that the prevalence of dental caries, periodontitis, tooth loss, mucosal inflammation, and opportunistic oral infections is significantly higher in individuals with schizophrenia compared to the general population [1,2]. These oral health disparities contribute to chronic pain, nutritional deficits, impaired social interaction, reduced communicative confidence, and overall diminished quality of life.

The etiology of oral health decline in schizophrenia is multifactorial, encompassing behavioral, pharmacological, and biological pathways. However, recent neurocognitive models suggest that cognitive dysfunction, particularly deficits in executive functioning, working memory, and attentional control, plays a central mechanistic role in mediating this deterioration. Neurobiological models of schizophrenia, particularly those emphasizing dopaminergic dysregulation, highlight impairments in motivation, reward processing, and goal-directed behavior [3], which may indirectly contribute to reduced engagement in daily self-care activities, including oral hygiene.

At a neurobiological level, dysfunction within fronto-striatal circuits may impair the initiation and consistency of goal-directed behaviors such as tooth brushing, while alterations in hippocampal–limbic networks may disrupt habit formation and contextual learning necessary for maintaining daily oral hygiene routines.

Importantly, these cognitive disturbances are supported by dysfunction within partially distinct neural systems. Executive planning, initiation, and monitoring of goal-directed behaviors—including daily oral hygiene—are closely linked to dorsolateral prefrontal cortex–centered networks and their interactions with fronto-striatal circuits, whereas the hippocampus and associated limbic structures contribute to contextual learning, routine formation, and the consolidation of health-related habits. Disruption across these systems may therefore translate neurocognitive impairment into inconsistent or ineffective oral self-care behaviors [2,3].

Disruptions in decision-making, planning, initiation, and self-monitoring make it difficult for individuals to engage consistently in preventive oral hygiene behaviors. Activities that require sequential planning, such as brushing and flossing, scheduling and attending dental appointments, or adhering to multi-step prosthetic care routines, are disproportionately affected. Negative symptoms such as avolition, anhedonia, and cognitive disorganization further exacerbate these difficulties, contributing to persistent patterns of self-neglect [4,5].

Xerostomia, metabolic dysregulation, and altered immune responses all increase periodontal vulnerability, and antipsychotic medication adds to the list of problems due to behavioral variables alone [6]. The gradual association of oral dysfunctions with changes in fronto-striatal and limbic circuitry, as shown by neuroimaging and neuropsychological studies, emphasizes cognitive deficits and motivational difficulties in schizophrenia [7]. This same neural basis suggests that poor oral health is a systemic manifestation of brain-behavior dysregulation, rather than a mere lifestyle effect [8].

From a public health and preventive perspective, the recognition of oral disease as a neurocognitively mediated comorbidity opens new interdisciplinary avenues for intervention. Cognitive remediation, psychosocial rehabilitation, and integrated psychiatric–dental care models could jointly mitigate the downstream effects of cognitive decline on oral hygiene and treatment adherence [9,10].

Unlike existing meta-analyses that primarily quantify oral disease prevalence and risk, the present review focuses on how neurocognitive and motivational impairments

translate into everyday oral hygiene behaviors and care engagement, offering a functional and mechanistic perspective with direct preventive implications.

Therefore, the present review aims to synthesize current evidence on the neurocognitive mechanisms underlying oral health decline in schizophrenia, emphasizing the interplay between cognitive domains, neural circuit dysfunction, symptom expression, and self-care behavior. The review further discusses preventive, rehabilitative, and interdisciplinary approaches bridging neuropsychiatry and dentistry, providing a translational framework for improving oral and systemic wellbeing in this vulnerable population.

2. Literature Review

Over the past two decades, growing interdisciplinary attention has been directed toward understanding how neurocognitive dysfunction in schizophrenia contributes to broader health disparities, including oral health decline. While behavioral, pharmacological, and socioeconomic explanations have been extensively documented, recent evidence suggests that alterations in executive control, attention, and motivation represent key mediators linking brain dysfunction to impaired oral self-care and disease progression [11,12].

The present article follows a narrative review design, aiming to synthesize and interpret current evidence regarding the neurocognitive mechanisms underlying oral health decline in schizophrenia. Literature selection was guided by conceptual relevance rather than by a formal systematic protocol. A comprehensive literature search was performed across PubMed/MEDLINE, Scopus, and Web of Science databases, covering publications from 2000 to November 2025.

The search strategy combined Medical Subject Headings (MeSH) and free-text terms, including “schizophrenia” [MeSH], “oral health” [MeSH], “periodontal diseases” [MeSH], “dental caries” [MeSH], “cognitive dysfunction” [MeSH], “executive function”, “self-care”, “motivation”, and “neurocognition”. Boolean operators (AND, OR) were used to refine the query and ensure inclusion of all relevant combinations. The selection process focused on studies addressing at least one of the following domains:

- (i) Oral health outcomes in schizophrenia;
- (ii) Neurocognitive, behavioral, or motivational impairments affecting self-care;
- (iii) Biological, immunological, or microbiome-related mechanisms;
- (iv) Pharmacological effects of antipsychotic treatment with oral health implications.

Although a formal PRISMA-based screening procedure was not applied, the overall process of identification, thematic screening, and inclusion is illustrated using a PRISMA-inspired flow diagram, as in Figure 1 below, included to enhance transparency and to clarify how the final body of literature was assembled within a narrative framework.

Eligible studies included:

- (1) Original clinical, observational, or neuroimaging research assessing oral health or periodontal outcomes in schizophrenia;
- (2) Investigations exploring cognitive, behavioral, or neurobiological correlates of oral hygiene;
- (3) Systematic or narrative reviews addressing the same topic.

Only peer-reviewed articles published in English were retained. Exclusion criteria comprised case reports, editorials, conference abstracts, and non-indexed material.

Exclusion criteria were applied to maintain conceptual coherence and scientific rigor. Non-indexed material was defined as sources not included in major bibliographic databases, such as non-peer-reviewed reports, opinion pieces, unpublished manuscripts, and conference abstracts without full-text availability. As this review follows a narrative design, no formal risk-of-bias assessment or quantitative quality appraisal tools were applied. Instead,

studies were evaluated qualitatively based on their methodological clarity, relevance to neurocognitive and behavioral mechanisms, and contribution to the conceptual framework discussed. Gray literature was not systematically searched or included, as the primary objective of the review was to synthesize peer-reviewed evidence rather than to perform comprehensive evidence mapping or grading, all results being illustrated in Figure 1 below:

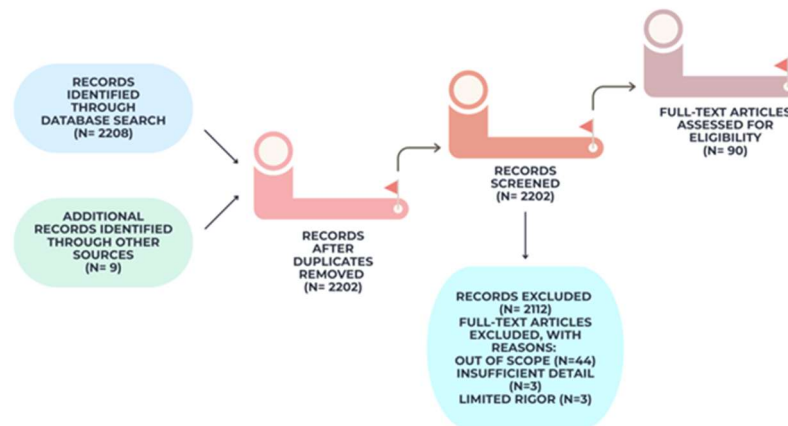


Figure 1. Flow diagram illustrating the literature identification and selection process for the narrative review.

2.1. Oral Health Burden in Schizophrenia

Carries prevalence, periodontitis incidence, and tooth loss are all significantly higher in the schizophrenic population compared to the general population, according to observational cohorts and meta-analyses. Individuals with severe mental illness (SMI) had significantly higher DMFT indices and were almost three times as likely to be toothless as community controls (OR = 2.81; 95% CI 1.73–4.57), according to a pivotal meta-analysis [13]. This points to a significant treatment gap when it comes to dental care access and utilization.

Multiple epidemiological investigations reinforce this pattern, consistently showing that individuals with schizophrenia access dental services less frequently, demonstrate poorer clinical oral parameters, and maintain significantly inferior daily hygiene routines compared with matched general-population controls [14]. Clinically, these behavioral and access-related disparities manifest as delayed presentation, progression to advanced stages of caries and periodontal pathology, a higher likelihood of requiring extractions instead of conservative restorative procedures, and overall reduced potential for long-term preservation of oral function.

Converging evidence also indicates a clear association between schizophrenia, antipsychotic therapy, and an elevated risk of periodontal disease. Systematic syntheses of periodontal outcomes demonstrate that people with severe mental illness—particularly those diagnosed with schizophrenia—exhibit consistently worse periodontal indices than healthy individuals, both at early onset and throughout chronic disease evolution [15]. These periodontal impairments are further aggravated by pharmacologically induced xerostomia, hyposalivation, and antipsychotic-related alterations in oral microbiota, which collectively generate a pathogenic environment conducive to accelerated caries progression and periodontal tissue breakdown [16,17].

Supporting this, data from large cohort studies confirm that dental caries and periodontal disease are disproportionately prevalent among individuals with mental health disorders, emphasizing the need for integrated care models that acknowledge the bidirectional links between psychological well-being and oral health [13]. More recent quantitative analyses deepen this understanding: a meta-analysis focused specifically on schizophrenia reported markedly elevated DMFT scores compared with healthy controls (mean

difference = 7.77; 95% CI: 3.27–12.27), indicating a substantially higher cumulative burden of dental disease across the lifespan [18]. Similarly, periodontitis appears to be nearly twice as prevalent in individuals with severe mental illness (OR = 1.97; 95% CI: 1.15–3.35), who also present increased odds for both shallow and deep periodontal pockets relative to the general population [19].

Additional complexity is provided by genetic-epidemiological data: a two-sample Mendelian randomization analysis conducted in 2024 found a causal relationship between the liability for schizophrenia and the risk of periodontitis (IVW OR $\approx$ 1.03; 95% CI 1.01–1.05). Using a Mendelian randomization approach, Cao et al. [20] did not find evidence supporting a causal relationship between periodontitis and schizophrenia. However, given the inherent assumptions and limitations of Mendelian randomization analyses, these findings should be interpreted with caution and do not definitively exclude more complex or indirect causal pathways. Shared inflammatory and behavioral mechanisms may mediate this unbalanced association [20]. These results are supported by large observational datasets, which show that people with severe mental illness have worse oral health and do not use dental treatments enough when compared to the general population [21].

Meta-analytic syntheses consistently demonstrate a markedly increased oral disease burden in individuals with schizophrenia. Pooled estimates indicate a two- to three-fold higher risk of edentulism compared with the general population, with reported odds ratios ranging between approximately 2.8 and 3.4. Importantly, these relative risk estimates are accompanied by large absolute differences in cumulative caries experience, with mean DMFT differences of approximately 7–8 affected teeth, reflecting a substantial lifetime disease burden rather than a marginal statistical association.

These effect sizes, derived from large population-based and clinical datasets, indicate a clinically meaningful deterioration of oral health, with direct implications for mastication, nutrition, and quality of life. As such, the quantitative magnitude reported in these meta-analyses supports the interpretation that oral disease in schizophrenia represents a major comorbidity, not merely an epiphenomenon of socioeconomic disadvantage or healthcare access.

Further complexity arises from the pharmacological side effects of long-term antipsychotic therapy. Drugs associated with hyperprolactinemia and xerostomia exacerbate periodontal and mucosal disease progression, reduce mechanical cleansing from saliva, and impair oral immune responses [22]. Taken together, these data underscore the multifactorial nature of oral health deterioration in schizophrenia, integrating cognitive impairment, medication effects, behavioral neglect, socioeconomic disadvantage, and physiological dysregulation into a unified explanatory framework [23].

2.2. Neurocognitive and Behavioral Mechanisms Linking Schizophrenia to Oral Disease

Neurocognitive impairment is a fundamental characteristic of schizophrenia, profoundly affecting everyday functioning, self-care, and overall health maintenance. Executive dysfunction, attentional deficiencies, and reduced working memory hinder the capacity to organize, initiate, and maintain routine health behaviors, such as toothbrushing and dentist appointments [24].

At the neurobiological level, neuroimaging evidence highlights reduced activation within the dorsolateral prefrontal cortex (DLPFC), anterior cingulate cortex (ACC), and insula, regions crucial for self-monitoring, decision-making, and motivation [25]. Dysfunction in these interconnected networks weakens the regulation of goal-directed behavior and contributes to diminished awareness or prioritization of health-related needs. As a

result, patients often fail to translate cognitive understanding of hygiene importance into consistent behavioral action—a phenomenon described as the intention–action gap [26].

Beyond its descriptive relevance, the intention–action gap has also been addressed through targeted behavioral interventions in psychiatric and neurocognitive populations. Approaches such as implementation intentions, which translate abstract goals into concrete, context-specific action plans, have shown potential in improving health-related behaviors in individuals with cognitive and motivational impairments. Although evidence specific to oral hygiene in schizophrenia remains limited, these interventions conceptually align with the neurocognitive barriers discussed and may represent a promising avenue for reducing the discrepancy between intention and daily oral self-care behaviors.

Importantly, the intention–action gap has also been addressed through targeted behavioral interventions in psychiatric populations. Approaches such as implementation intentions, which translate abstract goals into concrete, context-specific action plans, have shown potential in improving health-related behaviors in individuals with cognitive and motivational impairments.

Socioeconomic status, structural impediments, and specific neurocognitive and behavioral variables all play a role in the oral health disparities seen in schizophrenia. People with cognitive impairments, particularly those affecting attention, working memory, and executive function, may find it difficult to maintain regular dental hygiene routines and seek out preventive dental therapy [27]. As a result of the avolition, anhedonia, and unpleasant symptoms that accompany these deficits, the desire for self-care and treatment adherence is even more diminished [28]. Even when oral hygiene knowledge is present, motivational deficits and blunted affect reduce adherence to preventive behaviors. Cognitive-behavioral studies have shown that patients frequently underestimate the severity of dental symptoms or postpone care-seeking until pain or infection becomes disabling [28]. Moreover, deficits in theory of mind and social cognition impair communication with healthcare providers, contributing to missed appointments and inadequate follow-up [29].

More than 60% of people with schizophrenia who smoke also suffer from nicotine addiction [29]. This causes periodontal tissue to deteriorate more rapidly and takes longer to heal [30]. There is an association between a diet low in fruits, vitamins, and hard, carbohydrate-rich foods and an increased risk of developing cavities [31]. Alcohol and illicit drug use, common in this population, also exacerbate xerostomia, poor dietary choices, and parafunctional habits such as bruxism [32,33]. These cumulative factors create an oral environment prone to chronic inflammation, caries, and soft-tissue trauma.

Socioeconomic disadvantages, institutionalization, and stigma exert synergistic effects on oral neglect. Social withdrawal and perceived discrimination discourage patients from engaging with dental professionals or attending community clinics [34]. Studies report that individuals with schizophrenia are two to three times less likely to receive preventive dental care than the general population [35]. In addition, disorganized thinking and poor problem-solving skills impair the ability to navigate healthcare systems, schedule appointments, and comply with treatment regimens [36]. The lack of family or institutional support, combined with fragmented communication between psychiatric and dental services, often results in oral conditions being deprioritized relative to psychiatric stabilization. Consequently, acute infections or gingival inflammation are ignored until they become severe or interfere with essential functions such as mastication and speech [37].

This cycle of neglect is further exacerbated by socioeconomic disadvantages, including financial constraints and reliance on institutionalized care settings. Over time, this cumulative load turns oral conditions that may be managed into chronic, debilitating disorders that affect nutrition, speech, and general quality of life [35], as seen in Figure 2:

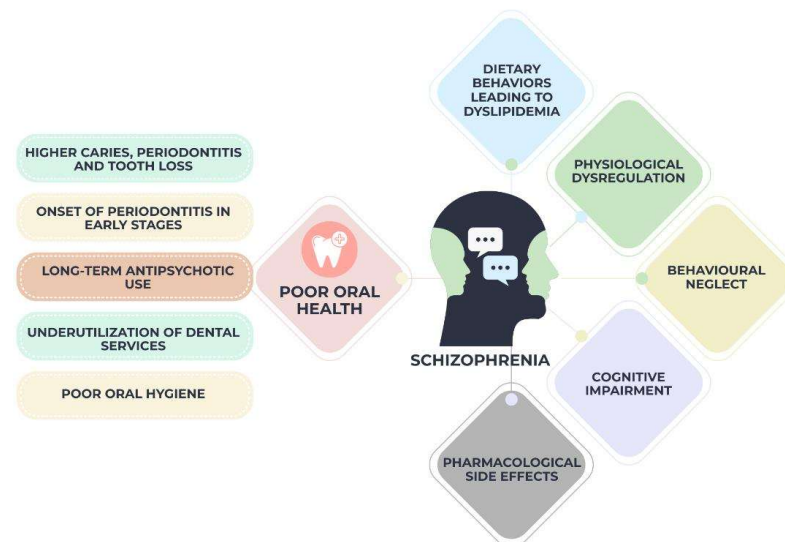


Figure 2. Neurocognitive and Behavioral Mechanisms Linking Schizophrenia to Oral Disease. This schematic illustrates the interaction between executive dysfunction, attentional deficits, motivational impairment, and behavioral neglect, contributing to poor oral hygiene, delayed care-seeking, and disease progression. Arrows indicate conceptual directional relationships between mechanisms rather than quantitative effect sizes.

Dietary and behavioral habits reinforce this downward spiral. Schizophrenic patients frequently consume soft, carbohydrate-rich foods due to limited chewing capacity, dental pain, or medication-induced appetite changes, promoting cariogenic biofilms and plaque accumulation [38]. Psychotropic-induced xerostomia compounds this by reducing salivary flow, buffering capacity, and antimicrobial protection [39]. Over time, this combination of cognitive deficits, behavioral patterns, and pharmacological side effects creates a self-reinforcing trajectory of oral health decline, progressively compromising nutrition, speech, and psychosocial functioning [40].

Recent strategies suggest that combining dental care with psychiatric therapies may help stop these mechanisms. Multidisciplinary programs that include motivational interviewing, supervised brushing, and on-site dental exams in psychiatric facilities have led to measurable improvements in plaque index, gingival bleeding, and overall oral hygiene [41]. Moreover, deficits in attention switching and working memory may limit the patient's ability to follow complex hygiene instructions or adhere to treatment regimens. The cumulative effect of these cognitive and motivational impairments leads to functional oral neglect, characterized by inconsistent plaque control, delayed response to dental symptoms, and avoidance of professional care [42]. This supports the idea that dealing with the neurobehavioral aspects of schizophrenia is important for achieving long-lasting dental health results.

In addition to these cognitive and behavioral aspects, biological and pharmacological factors—such as modified immunological responses, systemic inflammation, and the impact of antipsychotic medication—exacerbate the decline of oral health. This integrated model posits that neurocognitive vulnerability interacts dynamically with systemic physiological systems, establishing the foundation for the multifactorial burden.

2.3. Biological and Pharmacological Pathways Contributing to Oral Health Decline

In addition to cognitive and behavioral mechanisms, biological and pharmacological factors significantly contribute to the oral health deterioration observed in schizophrenia, as illustrated in Figure 3 below:

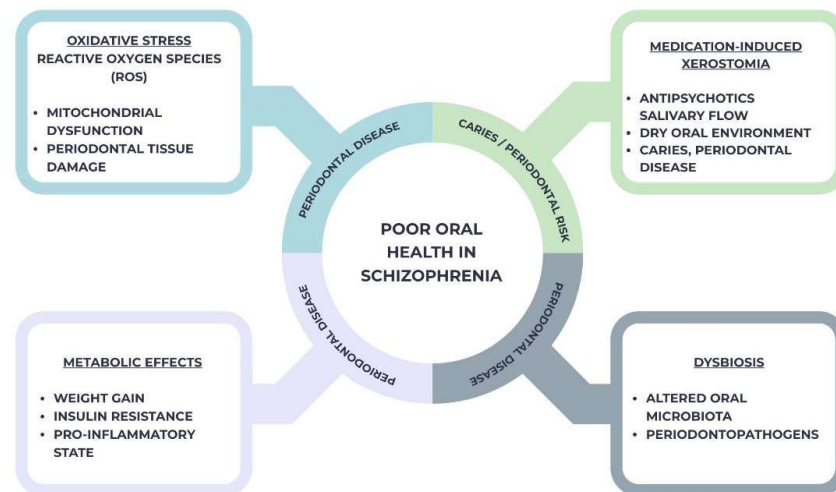


Figure 3. Poor oral health in schizophrenia. The figure summarizes major biological and treatment-related factors, including systemic inflammation, immune dysregulation, oxidative stress, metabolic alterations, and antipsychotic-induced xerostomia, which collectively increase vulnerability to caries and periodontal disease. The illustration represents a conceptual model; arrows denote mechanistic associations without implying magnitude or temporality.

The disorder is increasingly viewed as a systemic condition in which chronic inflammation, immune dysregulation, metabolic imbalance, and the side effects of antipsychotic medication act synergistically to compromise oral tissues. Elevated pro-inflammatory cytokines, altered immune responses, and drug-induced xerostomia or metabolic disturbances further intensify periodontal breakdown and caries development [43,44].

These mechanisms form an overlapping network where systemic biological changes and pharmacological effects reinforce each other, amplifying the risk of oral disease. Recognizing this interplay is essential for understanding why oral health management in schizophrenia must address both local and systemic determinants of pathology, as well as the iatrogenic consequences of long-term psychiatric treatment [45].

Chronic low-grade inflammation is a key biological hallmark of schizophrenia and represents a major pathway through which systemic alterations influence oral health. Elevated levels of inflammatory biomarkers such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF- α) have been consistently reported in both medicated and unmedicated patients [46,47]. These cytokines are known mediators of periodontal inflammation and contribute to connective tissue degradation, alveolar bone resorption, and impaired wound healing. Dysregulation of the immune-inflammatory axis increases susceptibility to exaggerated local responses against bacterial biofilm, accelerating periodontal tissue breakdown and loss of attachment [48]. Furthermore, systemic inflammation may perpetuate a bidirectional cycle, whereby oral infections amplify neuroinflammatory pathways implicated in schizophrenia pathophysiology [49], as illustrated in Figure 4 below:

Immune system abnormalities further compromise host defense mechanisms within the oral cavity. Schizophrenia has been associated with reduced T-helper cell activity, altered monocyte activation, and impaired neutrophil chemotaxis, leading to inefficient bacterial clearance [50]. This immunological imbalance is parallel by significant shifts in the composition of the oral microbiome. Recent metagenomic analyses have shown increased colonization by periodontopathogen species, particularly *Porphyromonas gingivalis*, *Prevotella intermedia*, and *Fusobacterium nucleatum* [51,52]. These microorganisms secrete virulence factors such as gingipains and lipopolysaccharides that amplify local inflammation and contribute to systemic inflammation. The resulting dysbiosis reinforces

both oral and systemic inflammatory states, exacerbating the overall disease burden in schizophrenic individuals [53], as seen in Figure 5:

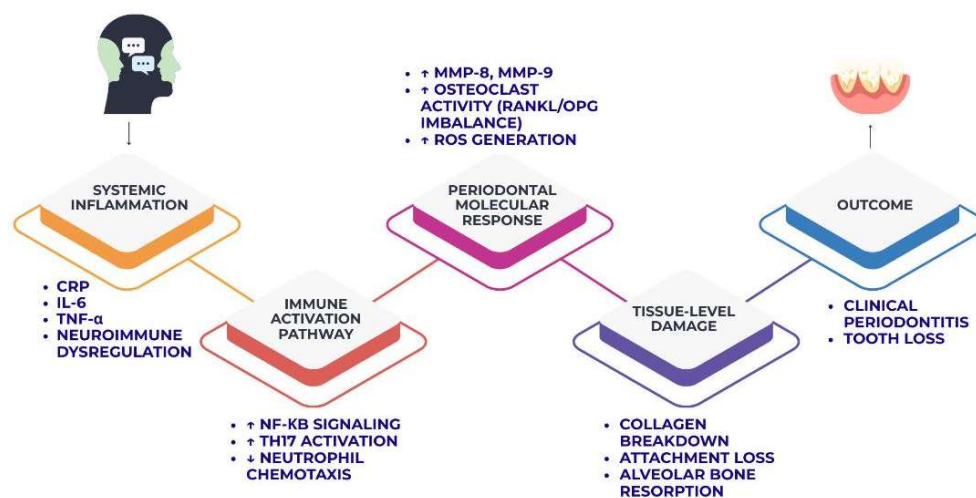


Figure 4. Chronic Systemic Inflammation in Schizophrenia—elevated inflammatory mediators such as CRP, IL-6, and TNF- α , resulting from systemic neuroinflammatory activation, contribute to exaggerated immune responses within periodontal tissues. Elevated inflammatory mediators, including C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF- α), contribute to exaggerated immune responses within periodontal tissues, promoting connective tissue degradation and bone loss. The figure is a schematic representation of inflammatory pathways and does not depict quantitative cytokine concentrations.

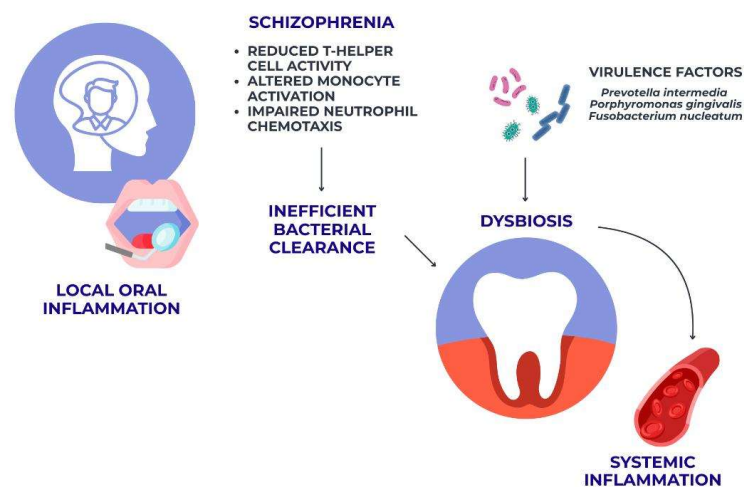


Figure 5. Immune Dysfunction and Oral Microbiome Changes in Schizophrenia—schizophrenia-associated immune alterations—such as reduced T-helper cell activity, impaired neutrophil chemotaxis, and altered monocyte activation—compromise host defense and lead to inefficient bacterial clearance. The diagram illustrates schizophrenia-related immune alterations, including impaired neutrophil chemotaxis, reduced T-helper cell activity, and altered monocyte function, alongside shifts toward a dysbiotic oral microbiome dominated by periodontopathogenic taxa. Microbial representations are symbolic and not proportional to actual abundance. Arrows indicate conceptual interactions rather than causal direction.

Both first-generation and second-generation antipsychotics can reduce salivary secretion through anticholinergic or dopaminergic blockade, impairing the natural cleansing and buffering capacity of saliva [54]. The resulting dry oral environment promotes bacterial overgrowth, mucosal irritation, and opportunistic infections such as *Candida albicans* [55].

Furthermore, oxidative stress appears to be a converging biological mechanism between schizophrenia and periodontitis [56]. Excess ROS (Reactive Oxygen Species) impair endothelial function, reduce gingival perfusion, and promote collagen degradation, thereby weakening the periodontium, as seen in Figure 6:

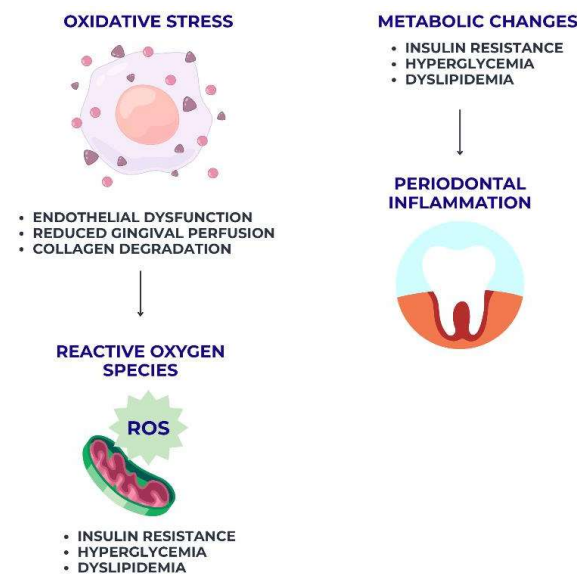


Figure 6. Oxidative Stress and Metabolic Dysfunction in Schizophrenia—Elevated reactive oxygen species (ROS) impair endothelial function, decrease gingival perfusion, and promote collagen degradation. Concurrently, antipsychotic-induced metabolic changes—such as insulin resistance, hyperglycemia, and dyslipidemia—exacerbate inflammatory pathways and delay tissue repair, resulting in progressive weakening of periodontal structures. Excess reactive oxygen species (ROS), mitochondrial dysfunction, and antipsychotic-induced metabolic alterations (including insulin resistance and dyslipidemia) converge to impair gingival perfusion, collagen integrity, and tissue repair. The figure depicts conceptual pathophysiological pathways and does not imply quantitative relationships or temporal sequencing.

In parallel, metabolic alterations—many of which are induced or exacerbated by long-term antipsychotic therapy—such as insulin resistance, hyperglycemia, and dyslipidemia, further intensify periodontal inflammation [57,58]. The presence of metabolic syndrome and type 2 diabetes among patients with schizophrenia not only worsens oral inflammatory outcomes but also delays tissue regeneration and healing responses following dental interventions [59]. Mitochondrial dysfunction and oxidative imbalance induced by antipsychotics may exacerbate periodontal tissue damage through enhanced lipid peroxidation and fibroblast apoptosis [60].

New microbiome research has shown that schizophrenia is linked to a distinct pattern of oral dysbiosis, characterized by a reduction in health-associated commensal organisms (such as *Streptococcus* and *Actinomyces* species) and a proliferation of anaerobic, pro-inflammatory taxa including *Prevotella*, *Veillonella*, and *Fusobacterium*, which are strongly implicated in the pathogenesis of chronic periodontitis [61–63]. This shift is not simply an incidental finding but reflects a broader disruption in host–microbe homeostasis, where protective microbial functions such as pH regulation, mucosal barrier maintenance, and competitive inhibition of pathogenic strains are weakened [64].

Finally, systemic immune dysfunction, which has been documented as a core biological feature of schizophrenia, may impair local oral immune surveillance and inflammatory resolution, thereby reinforcing dysbiosis [65].

Together, these processes create a biological feedback loop in which oral dysbiosis amplifies systemic inflammation (e.g., elevated IL-6, CRP), while systemic inflammation in

turn further disrupts oral microbial stability. This bidirectional interaction suggests that oral microbiome alterations in schizophrenia are not merely consequences of behavioral neglect but may actively contribute to neuroimmune dysregulation and symptom severity. Thus, managing oral dysbiosis may have implications not only for dental outcomes but also for broader psychiatric and metabolic health trajectories in this population [66].

These biological and pharmacological factors suggest that the decline in oral health in schizophrenia results not only from behavioral neglect but also from a complex interaction of drug-induced physiological alterations, immune dysfunction, and microbial imbalance [67], as seen in Table 1 below:

Table 1. Biological and pharmacological pathways contributing to oral health decline in schizophrenia.

Mechanism Type	Biological/Pharmacological Process	Oral Implications	Systemic/Pathophysiological Implications	Representative References
Salivary dysfunction (local)	Antipsychotic-induced anticholinergic and dopaminergic blockade reduces salivary secretion and alters pH.	Xerostomia, mucosal dryness, increased plaque accumulation, and higher caries incidence.	Impaired mucosal defense, microbial overgrowth, discomfort, and reduced oral clearance.	[39–41]
Metabolic and inflammatory alterations (systemic)	Antipsychotic therapy promotes weight gain, dyslipidemia, and insulin resistance, increasing IL-6, TNF- α , and CRP levels.	Gingival bleeding, periodontal inflammation, impaired healing.	Chronic low-grade inflammation, vascular endothelial dysfunction, and higher cardiometabolic risk.	[42–44]
Oxidative stress and mitochondrial dysfunction (shared axis)	Excessive ROS generation and impaired antioxidant defense affect both neural and periodontal cells.	Fibroblast apoptosis, collagen degradation, and periodontal tissue loss.	Neuroinflammation and mitochondrial injury reinforce oxidative imbalance.	[45,46]
Oral microbiome dysbiosis (microbial feedback loop)	Reduction in beneficial commensal bacteria; increase in <i>Prevotella</i> , <i>Veillonella</i> , <i>Fusobacterium</i> .	Periodontitis progression, biofilm resilience, halitosis.	Systemic dissemination of bacterial metabolites; inflammatory cytokine surge.	[47,48]
Integrative pathophysiological loop	Interaction between drug effects, immune dysfunction, and microbial imbalance.	Combined caries and periodontitis burden; mucosal vulnerability.	Bidirectional neuroimmune dysregulation between oral and systemic inflammation.	[49]

It is important to understand these interactions in order to come up with comprehensive preventive and therapeutic plans that work for both oral and systemic health.

Pharmacological therapy remains the cornerstone of schizophrenia management, yet long-term exposure to antipsychotic and adjunctive medications exerts profound effects on oral physiology and tissue homeostasis. Both first-generation (typical) and second-generation (atypical) antipsychotics possess anticholinergic and antidopaminergic properties that significantly alter salivary gland function, leading to xerostomia, pH reduction, and diminished antimicrobial activity [63,64]. Clozapine, olanzapine, and risperidone are particularly implicated in severe hyposalivation and altered salivary composition, predisposing patients to caries, mucosal infections, and halitosis [65]. Reduced salivary clearance facilitates plaque accumulation and *Candida* colonization, while impaired lubrication compromises denture retention and comfort [66].

Beyond local salivary changes, systemic metabolic disturbances induced by antipsychotic therapy contribute indirectly to oral pathology. Weight gain, insulin resistance, and dyslipidemia—especially under clozapine and olanzapine—promote chronic inflammation and delay tissue healing [67]. These metabolic side effects exacerbate periodontal disease progression and complicate postoperative recovery following oral interventions [68]. Furthermore, hematologic toxicity such as clozapine-induced agranulocytosis reduces neutrophil-mediated defense, thereby increasing susceptibility to acute infections and necrotizing gingivitis [69].

Adjuvant medications commonly prescribed in schizophrenia, including antidepressants, anxiolytics, and antiparkinsonian agents, further intensify oral complications. Tricyclic antidepressants and anticholinergic agents (e.g., trihexyphenidyl) exhibit cumulative xerostomic effects, whereas selective serotonin reuptake inhibitors (SSRIs) are associated with bruxism, gingival trauma, and mucosal sensitivity [70]. Lithium and valproate, used for mood stabilization, may induce gingival hypertrophy, hypersalivation, or dysgeusia, adding to oral discomfort and reduced compliance with hygiene routines [71]. The interplay of these pharmacological effects reinforces the bidirectional link between systemic treatment and oral vulnerability, emphasizing the necessity of integrated care models that monitor and manage iatrogenic oral side effects throughout psychiatric therapy [72].

2.4. Clinical and Public Health Implications

The deterioration of dental health in individuals with schizophrenia has measurable clinical consequences and considerable implications for healthcare structure and policy. A growing body of epidemiological research, including meta-analyses and large cohort studies, demonstrates that patients with SMI have markedly poorer oral health outcomes relative to the general population, as illustrated in Table 2 below:

Table 2. Clinical and Public Health Studies Linking Schizophrenia and Oral Health.

Study (Year)	Population/Setting	Design	Oral Outcomes	Population/Exposure	Main Findings
Kisely et al. (2011) [9]	SMI vs. community controls	Systematic review and meta-analysis	Edentulism; DMFT; DMFS	Severe Mental Illness (incl. schizophrenia)	Individuals with severe mental illness exhibited a significantly increased risk of edentulism (OR = 3.4; 95% CI: 1.6–7.2), together with substantially higher DMFT and DMFS scores compared with community controls.
Kisely et al. (2015) [13]	SMI vs. general population	Systematic review and meta-analysis	Edentulism	Severe Mental Illness	Meta-analysis confirmed a significantly increased likelihood of edentulism among individuals with severe mental illness (OR = 2.8; 95% CI: 1.7–4.6), consistent across included populations
Yang et al. (2018) [14]	Schizophrenia	Review/meta-analysis	Poor oral health indicators	Schizophrenia diagnosis	Patients with schizophrenia demonstrated significantly poorer oral health outcomes, including a higher risk of edentulism (OR ≈ 2.8), alongside increased caries and periodontal disease burden
Chu et al. (2012) [73]	Schizophrenia inpatients vs. disabled vs. general	Comparative cohort	Caries, periodontal indices, dental care use	Schizophrenia inpatient status	Psychiatric inpatients with schizophrenia exhibited poorer access to dental care and significantly worse oral health outcomes compared with community-dwelling individuals and the general population.
Shalaby et al. (2023) [74]	Patients on antipsychotics	Systematic review	Periodontitis progression	Antipsychotic medication (prolactin-raising)	All antipsychotic agents were associated with an increased periodontal risk, with a more pronounced effect observed for prolactin-inducing medications.
Chen et al. (2024) [50]	Mental health populations on antipsychotics	Review	Xerostomia; caries risk	Antipsychotics	Xerostomia was common, and reduced salivary flow was associated with an increased risk of dental caries

Table 2. *Cont.*

Study (Year)	Population/Setting	Design	Oral Outcomes	Population/Exposure	Main Findings
Kuo et al. (2020) [55]	Psychiatric inpatients	Interventional study	Plaque index; gingival index	Education + supervised hygiene	The intervention group showed significant reductions in plaque index and gingival index following an oral health promotion program with supervised hygiene
Kuipers et al. (2021) [56]	Mental health disorders	Scoping review (interventions)	Caries, PI, GI, attendance	Behavioral/educational/integrated care	Multi-component oral health programs showed promising results; however, robust randomized controlled trials are still needed.

The probability of edentulism is around three times higher (OR 2.8–3.4), and both the DMFT and DMFS indices consistently demonstrate a greater cumulative caries burden across a lifetime. There is a huge disparity between the number of people who have the disorder and the number of people who can access treatment. This is especially true for institutionalized patients, elderly adults, and people from low-income households [9,10].

The clinical interplay of cognitive impairment, apathy, and negative symptoms results in diminished desire and self-care, whereas medication-induced xerostomia, decreased salivary buffering, and modified oral microbiota heighten vulnerability to dental caries and mucosal infections. Antipsychotic-induced alterations—especially from prolactin-elevating or anticholinergic medications—intensify periodontal tissue inflammation and hasten bone resorption [73]. As a result, patients frequently exhibit severe periodontal damage, persistent mucosal lesions, or significant tooth loss, symptoms that hinder mastication, speech, and nutritional intake, consequently exacerbating overall morbidity [74,75].

Administering dental care for individuals with schizophrenia poses unique therapeutic challenges. Impaired cognitive processing may hinder understanding of treatment recommendations, while anxiety and distrust towards healthcare practitioners can limit collaboration [76]. Dentists may have difficulties in communicating with psychiatric teams, suffer obstacles in accessing medical information, and experience ambiguity regarding pharmacological adverse effects or contraindications [77].

It is also important to consider individual patient responses to sedation and potential interactions between antipsychotic medication and dental anesthetics when planning invasive procedures. In this context, clinical literature describes organizational adaptations—such as structured, time-efficient appointments, coordinated follow-up, and preventive-oriented approaches—as pragmatic strategies to facilitate dental care delivery in patients with severe psychiatric disorders, taking into account their psychological and pharmacological profiles [78]. These considerations are schematically summarized in Figure 7.

From a public health perspective, the significance of proper dental hygiene in mental health treatment is undervalued. Even in health care systems where everyone has access, people with SMI still do not obtain the preventive dental care they need. Reducing plaque and gingival indices following educational and hygienic treatments is one of the demonstrable effects of oral health promotion in psychiatric rehabilitation programs [79,80]. Behavior modification, professional cleaning, and positive reinforcement are just a few of the methods that have been proven effective in larger trials. However, they stress the importance of continuing use and high-quality randomized controlled studies [81–83].

Interventions at the system level are necessary for effective integration. In order to ensure prompt referrals, mental health providers should be trained to conduct basic oral exams and identify the symptoms of gum disease, candidiasis, and mucosal damage [84]. Evidence suggests that adherence and emergency visits can be improved by integrating

oral health indicators into mental electronic health records and creating shared clinical pathways between dental and mental services [85]. The goals of public policy should be to incorporate oral health into national mental health plans, provide continuity of care between inpatient and community-based programs, and provide subsidies for preventive dental care for psychiatric populations [86].

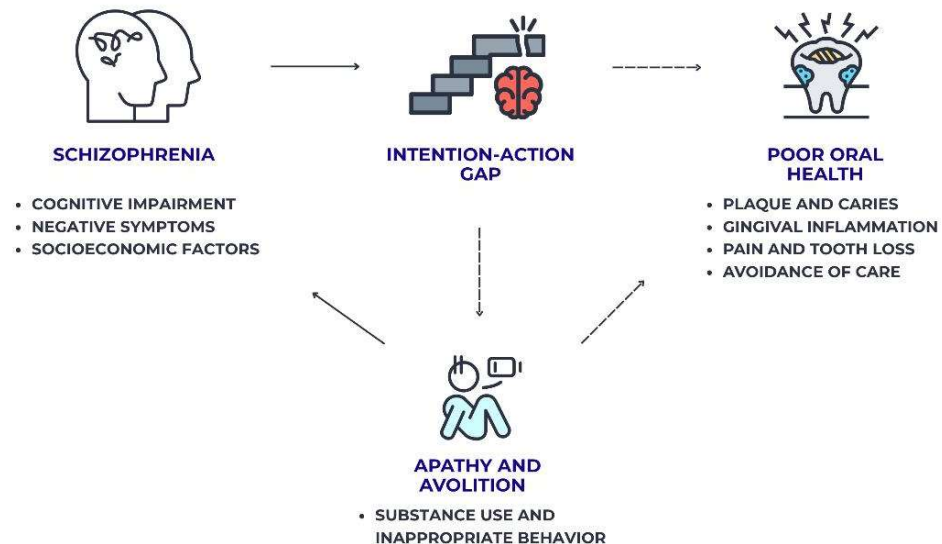


Figure 7. Clinical and Therapeutic Implications of Oral Health Decline in Schizophrenia—multifactorial origins and consequences of oral health deterioration in schizophrenia. This conceptual framework integrates neurocognitive impairment, behavioral dysfunction, biological vulnerability, and healthcare access barriers, highlighting their combined impact on clinical outcomes and interdisciplinary management strategies. Boxes represent thematic domains rather than measured variables.

2.5. Integrated Neuropsychiatric–Dental Care Models

The growing recognition that oral health deterioration in schizophrenia arises from the interaction between cognitive dysfunction, motivational deficits, medication side effects, and structural barriers to care has led to the emergence of integrated service delivery models that combine psychiatric, psychological, and dental expertise. These programs challenge the traditional segmentation of medical and dental care by acknowledging that oral disease in severe mental illness is not merely a behavioral outcome but a functional expression of neurocognitive impairment and reduced capacity for self-management [84].

In integrated care frameworks, oral health is embedded into psychiatric treatment pathways rather than treated as an optional or secondary concern. For example, routine oral health assessments can be conducted during psychiatric follow-up visits, facilitating early detection of caries, periodontal inflammation, prosthetic instability, and xerostomia-associated mucosal damage [85].

This approach enables intervention before disease progression leads to irreversible tissue loss or the need for emergency extractions, which are common when dental care is fragmented [86].

Collaborative clinical settings in outpatient psychiatric clinics, day-hospital programs, and assisted living facilities demonstrate that the co-location of dental professionals within mental health teams enhances treatment adherence, reduces missed appointments, and improves long-term maintenance outcomes. These models often incorporate supervised toothbrushing routines, motivational interviewing, graded behavioral prompts, and simplified oral hygiene plans tailored to the cognitive profile of each patient. Such interventions

have produced measurable reductions in plaque index, bleeding on probing, and acute dental visits among schizophrenia cohorts [86].

Equally important is the role of caregiver and family education, particularly for individuals with negative symptoms of dominance (avolition, apathy) or impaired executive functioning. Providing caregivers with structured guidance on oral hygiene support and symptom-responsive communication strategies can significantly improve consistency of daily oral care and reduce behavioral resistance to treatment [87].

Telehealth-based follow-up, mobile dental units, and domiciliary care services represent additional extensions of integrated care, particularly relevant for patients with low mobility or heightened anxiety toward clinical environments. These service models help bridge the treatment access gap documented in epidemiological studies, where individuals with schizophrenia are less likely to initiate or complete oral health interventions [88,89].

Ultimately, integrated neuropsychiatric–dental models demonstrate that meaningful improvement in oral health outcomes occurs only when behavioral, cognitive, pharmacological, and social determinants of health are addressed concurrently, rather than sequentially or in isolation. This paradigm reframes oral health not as an optional adjunct but as a core component of recovery-oriented psychiatric care and patient well-being [90].

To avoid etiological conflation, it should be explicitly stated that the following evidence from neurodevelopmental and neurodegenerative conditions is not cited to suggest shared pathophysiological mechanisms with schizophrenia. Rather, these studies are included as trans-diagnostic functional models, illustrating how persistent cognitive impairment and reduced adaptive capacity—regardless of underlying disease etiology—can lead to compromised oral self-care behaviors and increased vulnerability to oral health deterioration.

As a narrative review, this study is inherently subject to selection bias, as the inclusion of evidence was guided by thematic relevance and conceptual coherence rather than by a formal systematic screening protocol. Although multiple databases were consulted and recent high-quality literature was prioritized, the final body of included studies may reflect publication availability, indexing practices, and language restrictions, potentially favoring well-represented research areas while underrepresenting negative or less frequently reported findings.

Furthermore, the heterogeneity of included study designs, ranging from epidemiological cohorts and meta-analyses to neurocognitive, microbiological, and pharmacological investigations—may introduce uneven evidentiary weight across domains, limiting direct comparability.

This selection bias does not undermine the validity of individual findings but constrains the ability to draw quantitative or causal conclusions. Accordingly, the interpretations offered aim to contextualize convergent evidence and mechanistic patterns, rather than to provide pooled estimates or definitive causal inferences.

3. Future Directions

Future research should aim to elucidate the bidirectional relationship between schizophrenia and oral health by integrating neurocognitive, immunological, and behavioral frameworks into unified longitudinal models. High-resolution neuroimaging combined with oral microbiome sequencing could reveal how specific neural circuit disruptions (particularly within the DLPFC–ACC–insula network) modulate inflammatory signaling and oral microbial ecology. Understanding these neuroimmune–microbiome interactions would enable precision-based interventions targeting both neural regulation and oral homeostasis.

As a narrative review, this study is limited by the absence of a systematic search strategy and formal risk-of-bias assessment. Although efforts were made to include the most recent and relevant literature, the possibility of publication bias and selective reporting cannot be excluded. The heterogeneity of included studies—spanning neurocognitive, immunological, behavioral, and pharmacological domains—makes direct comparisons challenging and limits the ability to establish causal inferences.

A major limitation of this review is the reliance on indirect evidence from dementia research regarding integrated care models, due to the scarcity of specific data for schizophrenia.

Furthermore, much of the current evidence relies on cross-sectional or small-sample observational studies, with limited representation of institutionalized or low-income populations. Few investigations have employed integrated methodologies combining neuroimaging, microbiome analysis, and clinical dental outcomes, which constrain the translational interpretation of mechanistic findings. Future systematic reviews and longitudinal cohort studies are warranted to validate the proposed multifactorial pathways and to clarify temporal and bidirectional relationships between schizophrenia and oral health decline.

Another critical direction involves the implementation of multidisciplinary preventive programs that merge psychiatry, dentistry, and behavioral science. Interventions such as digital adherence tools, cognitive remediation combined with oral hygiene training, and motivational interviewing protocols should be tested in controlled clinical trials to assess their long-term efficacy and cost-effectiveness. Parallel efforts are required to develop pharmacological strategies that mitigate medication-induced xerostomia and metabolic dysfunction without compromising psychiatric stability.

From a policy standpoint, integrating oral health assessment into psychiatric follow-up protocols could markedly improve outcomes, especially for institutionalized or socioeconomically disadvantaged patients. Collaborative frameworks between community mental health services and dental care networks are essential to reduce disparities in access and to ensure continuity of care. Ultimately, the advancement of translational models linking neural, immunological, and behavioral mechanisms will redefine how oral health is monitored and managed in schizophrenia, shifting from reactive treatment toward proactive, integrated prevention.

Future research should prioritize cognitive remediation strategies with direct relevance to daily self-care behaviors. Interventions targeting executive functioning and planning (e.g., goal management training), prospective memory support (e.g., reminder-based or cueing interventions), and habit formation techniques (such as implementation intentions and structured routines) may be particularly relevant for improving oral hygiene behaviors. These approaches are feasible within psychiatric care settings and could be integrated with dental education to enhance adherence to daily oral self-care in individuals with schizophrenia.

This review has inherent limitations related to its narrative design. Although the literature search was broad and conceptually structured, it did not follow a systematic PRISMA protocol, and therefore the selection of studies may be influenced by publication availability and reporting variability.

The evidence synthesized originates from highly heterogeneous sources—ranging from observational cohorts and meta-analyses to neurocognitive, microbiological, and pharmacological investigations—which limit the comparability of findings and prevent the formulation of quantitative conclusions.

Much of the existing research relies on cross-sectional designs or small clinical samples, reducing the ability to establish temporality or causality between schizophrenia-related mechanisms and oral health deterioration. Consequently, the interpretations offered here

aim to illuminate emerging mechanisms and interdisciplinary links rather than deliver definitive causal estimates.

4. Conclusions

The evidence reviewed highlights that oral health deterioration in schizophrenia is a multifactorial process shaped by the interplay between neurocognitive dysfunction, behavioral impairment, systemic inflammation, and medication-induced physiological changes.

Deficits in executive functioning and motivation, coupled with immune dysregulation, oxidative stress, and pharmacological side effects, converge to produce a persistent cycle of neglect, inflammation, and tissue breakdown.

Understanding schizophrenia as a systemic condition—rather than solely a psychiatric disorder—broadens the clinical perspective and emphasizes the importance of interdisciplinary management. Integrating dental, psychiatric, and behavioral care within shared protocols can significantly improve both oral and general health outcomes.

Emerging evidence suggests that integrated psychiatric–dental care models may be associated with improvements in oral health outcomes and care engagement; however, the current literature is limited by small sample sizes, preliminary study designs, and, in some cases, extrapolation from related neurocognitive populations such as dementia. Consequently, these findings should be interpreted as promising but provisional, underscoring the need for schizophrenia-specific, methodologically robust investigations.

Direct correlational data linking performance on specific neurocognitive measures—such as the Wisconsin Card Sorting Test (WCST) or Trail Making Test (TMT)—with standardized oral health indices (e.g., DMFT, CPI) are limited in the schizophrenia literature. While executive dysfunction and slowed processing speed are well documented, corresponding statistical associations with dental caries or periodontal indices have not been systematically reported, highlighting an important gap for future empirical research.

Continued translational research combining neuroscience, immunology, and clinical dentistry is essential to refine preventive strategies, personalize treatment, and mitigate the long-term burden of oral disease in individuals with schizophrenia.

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Abbreviations

The following abbreviations are used in this manuscript:

SMI	Severe Mental Illness
DMFT	Decayed, Missing, and Filled Teeth
DMFS	Decayed, Missing and Filled Surfaces
DLPFC	Dorsolateral Prefrontal Cortex

ACC	Anterior Cingulate Cortex
CRP	C-reactive protein
IL-6	Interleukin-6
TNF- α	Tumor Necrosis Factor-Alpha
ROS	Reactive Oxygen Species
SSRIS	Selective Serotonin Reuptake Inhibitors

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