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ORAL MICROBIOME DISTURBANCE, SALIVARY DYSFUNCTION, AND COGNITIVE HEALTH IN OLDER ADULTS: AN ACADEMIC REVIEW OF THE ORAL-BRAIN AXIS

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ABSTRACT

Multimorbidity, polypharmacy, decreased salivary flow, tooth loss, periodontal disease, denture use, changes in diet, diminished self-care, and immunosenescence represent the oral physiology changes related to ageing. All of these factors cause reconfiguration of the oral microbial ecosystem. Biological state relevant isn't just reduced amount of 'normal flora' but depleted ecological stability, reduced in numbers of good flora, increased presence of 'pathobionts', depleted in numbers of nitrate-reducing flora and chronic periodontal inflammation. Thus, this review aims at exploring the potential linkage which may exist between age-associated, oral dysbiosis and cognitive impairment, dementia and Alzheimer disease. Mechanisms for their spread have been proposed to involve systemic exposure to inflammatory mediators and microbial products, microperfusion, blood-brain barrier disruption, activation of microglia, amyloidogenic responses, tau-related injury, involvement of vascular pathways, nitric-oxide pathways, nutritional and/or quality of life decline, and reduced mastication after tooth loss. There is general support for a relationship between periodontitis and loss of teeth, oral microbial signatures and cognitive decline in observational studies and systematic reviews, although causality has not been shown with the majority of the decline in cognition thought to cause oral decline. Although till now, evidence does not support microbiome-directed interventions, they should be part of oral health assessment and periodontal care that forms part of healthy ageing. Future longitudinal studies need to include standardized dental phenotyping, saliva measurements, shotgun metagenomics, inflammatory measures and trajectories, cognitive trajectories, medication data and trials of intervention.

KEYWORDS: Oral microbiome; ageing; xerostomia; periodontitis; mild cognitive impairment; dementia; Alzheimer disease; oral-brain axis.

Review approach

This is a narrative review of the epidemiological, microbiome, mechanistic and clinical literature on oral health and cognitive ageing. Emphasis was placed on study design, reverse causation, confounding, oral sampling site and the difference between measurers of microbial abundance and dysbiosis. The numbers in the text refer to the reference list.

Key conceptual distinctions

Term	Meaning	Research implication
Low microbial biomass	Reduced absolute number of organisms.	Requires quantitative methods, not relative abundance alone.
Low diversity	Fewer taxa or less evenness.	May be harmful, neutral, or site-specific.
Loss of commensals	Depletion of health-associated organisms/functions.	Can reduce colonization resistance or nitrate metabolism.
Oral dysbiosis	Disease-associated ecological and functional imbalance.	Most relevant framework for periodontitis and cognition.

1. Introduction and Conceptual Clarification

The oral cavity is one of the most diverse microbial communities of the human body. Cultivating in a niche-specific manner, bacteria, fungi, archaea and viruses reside on the tongue, teeth, gingival sulcus, mucosa, saliva and surfaces of the prostheses. In health, these organisms represent a dynamic ecosystem which is important in colonization resistance, the metabolism of nitric oxide, immune education and maintaining local homeostasis. Hence, when talking about the oral normal flora, it is incomplete, because the question should be the oral normal flora lower than what? When sampling an environment, the concentration, diversity, structure, functional potential, and anatomic location of the microbes has to be separated.[1,2,25-27]

It may be a reflection of an apparently reduced abundance of selected commensals, coupled with an increase in overall plaque biomass, followed microbiota expansion, a decrease in ecological resilience, and/or increase in fungal colonization, in older adults. The more appropriate term is oral dysbiosis, a community composition and function change that is linked to a reduction in the body's defence or a disease. A mental or thinking response is similar expansive as well. Typically, research evaluates global cognition, memory, executive function, mild cognitive impairment, dementia diagnosis, neuropsychiatric symptoms, or Alzheimer disease biomarkers and not a single ‘mental response.[1,2,25-27]

The oral-brain axis has emerged as an important research specialty area, since oral disease is prevalent, may be amenable to change and is biologically linked to systemic inflammation and vascular health. Meanwhile, however, it can be over-hyped. Direct links from oral bacteria to dementia are associations only and the reverse causation is probably true – a reduction in cognitive function may lead to less flossing, less eating and nutritious food, less medication compliance, less oral care visits, and ultimately increased oral disease. A balanced review is needed to consider both directions.[1,2,25-27]

2. Ageing, Salivary Function, and the Oral Ecosystem

As for much more severe salivary gland failure, although this is not inevitable in healthy ageing, a high prevalence of xerostomia and hyposalivation exists in older populations due to medications, dehydration, systemic disease, radiotherapy, autoimmune disease, and reduced functional reserve of their salivary glands. Common xerogenic drug classes include analgesics, diuretics, antihistamines, anti-resistant, some analgesics and anticholinergics. Polypharmacy

can have add-on effects. In the normal state, saliva lubricates tissues, buffers acids, provides calcium and phosphate, helps swallow food and taste, and contains antimicrobial agents including: lactoferrin, peroxidases, histatins and secretory immunoglobulin A.

With decreased salivary flow, the pH, oxygen gradients, nutrient availability, clearance, and epithelial protection change. These changes could lead to changes in dental caries, candidiasis, mucosal injury, and halitosis as well as denture problems. Ageing and xerostomia has been reported to change the composition of micro-organisms in the mouth and to decrease their stability, though the changes in alpha diversity differ in various studies.[1,2] This heterogeneity is attributable to variations in oral phenotypology, dentition, sampling niche, geography, sequencing platform, drug use, and frailty.

Another aspect of ageing is a host response modification. Immmunosenescence may affect the ability to respond to microbial stimuli and helps to generate unregulated inflammatory signals, named "inflammaging. Diabetes, malnutrition, smoking history and low manual dexterity are additional factors of stress. Thus, age should not be considered a stand-alone causal determinant; age is a proxy for multiple biological, behavioral and environmental exposures.

3. Oral Dysbiosis, Periodontitis, and Loss of Beneficial Functions

Periodontitis is a chronic inflammatory condition in which a shift towards dysbiotic subgingival communities and susceptible host response leads to destruction of periodontal attachment and alveolar bone. Common organisms that are often cited to have a colloquially notable involvement include the anaerobic Porphyromonas gingivalis, Treponema denticola, Tannerella forsythia, as well as Fusobacterium nucleatum. Controls that focus on interactions at the community level and host-mediated destruction of tissue are emphasised in modern ecological models.

The importance of changes to beneficial taxa lies in benefits of commensals in filling niches, controlling pathogens, metabolizing food residues and regulating immunity. The oral nitrate-reducing bacteria participate in the enterosalivary nitrate-nitrite-nitric oxide pathway which helps maintain the vascular system. It is reasonable to hypothesise that this loss of function may be associated with cerebrovascular/cognitive health outcomes and is associated with oral dysbiosis, although such direct causal evidence is in the early stages.

Ecological alterations are also created by the loss of teeth and the use of dentures. Edentulism eliminates non-shedding tooth surfaces and the periodontal pockets and instroduces acrylic surfaces which can support biofilms. Minimized mastication can impact nutrition, cerebral blood flow and sensory input. So ‘less flora' following tooth loss is not necessarily healthier, as the anatomical habitat and functional consequences have changed.

4. Cognitive Ageing, Dementia, and Alzheimer Disease

Cognitive ageing can include normal ageing, mild cognitive impairment and major neurocognitive disorder. The clinical dementias are characterized by a functional deficit and could include several pathologies such as Alzheimer's, vascular, Lewy-body, other pathologies, etc.rogate Alzheimer disease is characterized by a biological continuum with amyloid beta and tau pathology. Mixed pathology is more frequently seen in older subjects. Because of this complexity, oral exposures can be simply related to inflammatory or vascular process and affect cognition without being specific to the Alzheimer disease.

The best available evidence for oral health is not in the context of a single microbial signature but for periodontitis and tooth loss and for general cognitive outcomes. Effect sizes are small and heterogeneity is high, but most meta-analyses do suggest that there is a significant correlation between periodontal disease and the preponderance or incidence of cognitive impairment and dementia. Although there are various microbiome sequencing studies comparing cognitively normal elderly over the age of 60 years with individuals with mild cognitive impairment, the results are not yet sufficiently uniform to use for diagnosis.[7-10]

The current evidence is only associative, and mechanistically plausible. A diagnosis of dementia can't be made from an oral swab -- and there's no evidence of anything else but restoring a specific bacteria to stop developing an Alzheimer disease.

5. Proposed Biological Pathways of the Oral-Brain Axis

A possible mechanism is systemic inflammation. Bacterial products and inflammatory mediators easily enter into the circulation via the ulcerated periodontal pocket epithelium, which is present in large quantity. Periodontitis is linked to increased levels of C-reactive protein, interleukin-6, signaling with tumor necrosis factor and other inflammatory factors. Systemic inflammation, especially of a chronic nature, can influence endothelial function and blood-brain barrier integrity and microglial activation.[16-18,25,28-33]

The second pathway is the episodic bacteremia and the microbial products. Oral bacteria may be transiently 'blooded' as a result of tooth brushing, chewing and dental procedures, especially in inflamed periodontal tissues. Perhaps bacterial DNA, lipopolysaccharide (LPS), proteases, and other virulence factors play a role in systemic immune activation. Although experimental and post mortem studies have suggested various roles for P. gingivalis and its gingipain proteases, the interpretation of these findings and results in such studies does not imply causation or directionality.[16-18,25,28-33]

Amyloidogenic/tau related signaling is a third pathway. Amyloid-beta is known to have antimicrobial and innate immune function and the possibility was suggested that chronic microbial stimulation could affect amyloid deposition. Microbial virulence factors or inflammatory signaling may also include models to this state of tau phosphorylation. These mechanisms are possible but the jump from cell/ animal model to the prevention of human disease is large.[16-18,25,28-33]

The 4th pathway is vascular. Endothelial dysfunction and cardiovascular risk factors connected with periodontitis contribute to the vascular cognitive dysfunction and expression of Alzheimer disease. A disruption of the nitrate reducing oral community might, therefore, lead to a decrease in nitric oxide supply. A fifth.

The nutritional and mechanical pathway is that tooth loss and oral pain and xerostomia may impact dietary quality, protein intake and chewing function, with possible consequences on frailty and cognition.[16-18,25,28-33]

6. Human Microbiome Studies

Comparisons of samples collected from saliva, tongue or subgingival have been performed using 16S rRNA gene-sequencing in cognitively normal adults, mild cognitive impairment and dementia. Oral taxon and associations with inflammatory markers were different in older persons with mild cognitive impairment.[7] In a community sample, Da and colleagues found associations between features of salivary microbiomes and mild cognitive impairment.[8] Oral

microbial composition has also been correlated with executive functions and subjective memory measures in the analysis of the nationally representative data.[9]

Subgingival sampling may be especially pertinent given that it represents periodontal ecology but it is technically more challenging and greatly affected by pocket depth, gingival bleeding, number of teeth sampled and recent dental care. The unique subgingival taxonomic and functional signatures at different levels of cognitive function have been reported in 2024.[10] Recent studies have also investigated the relationship of APOE genotype and nitric oxide related microbial functions.[11] These results were obtained in favour of an oral-brain research model which may be subject to confounding and multiple-comparison error.

None of the taxa has proven to be reproducible biomarkers. Some research focuses on the augmentation of periodontal pathobionts and some on the loss of periodontal health promoting organisms or the disruption of a web of organisms. Different DNA extraction method, selection of primers, length of sequencing, bioinformatic pipeline, oral niche and population characteristics may generate divergent results. Although 16S metagenomics can give taxonomic information, shotgun metagenomics and metabolomics may give functional information with more resolution.[7-11,13-15]

7. Epidemiologic Evidence for Periodontitis, Tooth Loss, and Cognition

Overall, systematic reviews indicate that there is a relationship between periodontitis and cognitive disorders. A meta-analysis with the assessment of study-design factors reported higher risks in cross sectional and longitudinal evidence with significant heterogeneity.[4] A 2024 periodontal disease synthesis showed that the risk ratio for the association of periodontal disease with cognitive disorders ranged near 1.25, varying among studies according to the type of outcome and study design assessed.[5] It has also been noted that low dentition and/or tooth losses are associated with cognition problems: however, the lifetime health practices, smoking habits, diabetes, and economic status play a role in tooth loss.[6]

Even though longitudinal designs provide more information than comparisons of images at a single point in time, they do not rid the data of the effects of residual confounding. Dementia can take years or even decades to develop and oral disease is a potential cause and impact. Repeated dental assessments and repeated cognitive assessments must be conducted in studies (dental and cognitive starting before functional decline). The possibility for "competing mortality" and "differential dental attendance" can bias older cohorts.[3-6,19-24]

In an intervention study, there was one report of positive association of periodontal treatment to a measure of Alzheimer-related brain atrophy.[12] The latter is a very important observation, but it provides no proof that treatment prevents dementia. Currently, there are limited clinical intervention trials with cognitive outcomes and periodontal therapy is best indicated by known oral and systemic benefits, not promising a cure for Alzheimer.

8. Reverse Causation and Confounding

These patients may suffer from cognitive impairments that directly impact upon oral health. Factors that result in memory deficits and missed brushing/appointments; factors that result in apraxia and poor brushing technique; factors that result in decreased cooperation because of behavioral symptoms; factors affecting oral ecology from a dysphagia/hasshfood change in diet; factors affecting caregiver proficiency; and factors related to medications reducing saliva. Older adults who have been institutionalized are a special group of risk. Hence, poorer oral microbiological

parameters in dementia may be a consequence of disease rather than being a causative factor.[3-6,13,15,22,23]

Important confounders are age, education, income, smoking, alcohol drinking, diabetes, cardiovascular disease, diet, BMI, depression, frailty, physical activity, number of medications, dentition, access to dental services, and APOE genotype. The microbiome can be greatly altered by exposure to antibiotic or antiseptic mouthwashes. The effect of these factors may have not been controlled for in the studies and may be responsible for the overestimation of independent oral effects.[3-6,13,15,22,23]

The compositionality issue is another analysis issue. Sequencing is done in terms of the relative abundance: if one taxon grows in abundance, another may look reduced. To determine if a community has been depleted, either absolute bacterial load or flow cytometry or spike-in standards and/or quantitative PCR can be helpful. This will be a key consideration in weighing the term 'lower than normal flora.[3-6,13,15,22,23]

9. Clinical Implications for Older Adults

There is current evidence which supports comprehensive oral care within healthy ageing. Clinicians should examine the dentition, periodontal inflammation, oral pain, chewing ability, swallowing function, denture hygiene and xerostomia together with the patient's own ability to self-manage, and to check for the presence of candida albicans. A medication review can indicate if there is anything you can do to prevent dry mouth. Hydration, saliva replacements, sugar free chew where possible, fluoride and treatment with dentists can help minimize complications.[1,2,13,22,23]

With cognitively impaired patients, oral-care plans should be easily accessible, visually clear and caregiver-supported. Powered toothbrushes and high fluoride products as directed, interdental aids, removal and cleaning of dentures as well as a regular professional review may be beneficial. It is important to educate the carer on signs of pain, bleeding, loose teeth, oral infection, and decreased feeding.[1,2,13,22,23]

Probiotics, prebiotics, microbiome transplants, nitrate supplement, and targeting antimicrobial are experimental in this space. Global oral microbial suppression is not a logical and appropriate objective as beneficial functions would also be lost. Goals for treatment should be to restore ecological health by means of plaque control, periodontal treatment, salivary intervention, nutrition, and systemic risk factor management.[1,2,13,22,23]

10. Recommended Research Design

It is essential to use a well-designed study with standardized diagnostic criteria for the selection of cognitively normal older adults, a MCI group, and a dementia group. Oral assessment forms for oral assessment should encompass periodontal probing, bleeding on probing, clinical attachment loss, number of teeth, caries, denture status, mucosal disease, oral hygiene indices, salivary flow, pH and xerostomia symptoms. Saliva, tongue, supragingival plaque and sub gingival plaque should be differentiated in sampling.[2-11,13-15]

Negative extraction controls, mock communities, and randomized batch processing, absolute-load measurement, and prespecified bioinformatics are essential to microbiome analysis. Evaluations of inflammatory, nitrate reducing, proteolytic and virulence genes can be made using shotgun metagenomics. Measurements of salivary/serum inflammatory markers and nitric oxide metabolites, as well as nutritional status and vascular measures would enhance mechanistic interpretation.[2-11,13-15]

Longitudinal follow up must be done. A repeated cognitive assessment is necessary to assess memory, executive function, attention, language and function at home. Medication exposure should be updated over time, as well as dental treatment, antibiotics and diet as well as caregiver supports. A nested periodontal-treatment trial could evaluate the effect of reduction in oral inflammation on the systemic biomarkers, or rate of change in cognition.[2-11,13-15]

11. Limitations of the Current Literature

The majority of studies conducted are observational, a significant number of these are cross-sectional and sample sizes are frequently small. Definitions of periodontitis, cognitive impairment and oral dysbiosis vary. Sequencing can be done in different ways, and at times may be "pooled" orally. An important limitation is that of the presence of unmeasured confounding and reverse causation. This may be due to publication bias, which tended to publish positive associations.[3-6,13-15]

Causality can be better established mechanistically in experimental systems and is not well established in human systems. The presence of a periodontal organism or its product in brain does not mean that it is responsible for the start of the disease. The pathological process of Alzheimer can start decades prior to dementia, and late life sampling in the mouth could not reflect the crucial time of exposure.[3-6,13-15]

The need is for greater terminology clarity in the field. The notion of 'normal flora' suggests a "list" that is healthy, when in reality, the oral microbiome is context dependent and unique to each person. There are a number of different consequences of 'low flora'—low biomass, insufficient richness, loss of good flora, and antibiotic suppression.[3-6,13-15]

12. CONCLUSION

For older people, salivary dysfunction, oral disease, tooth loss, frailty and polypharmacy, can lead to salivary disturbance in the oral ecosystem. A correlation has been observed between periodontitis, low dentition, and certain oral microbes and reduced cognitive functioning for a variety of reasons, including inflammatory, microbial, vascular, and nutritional mechanisms. But direction of causation has not been definitely determined and it seems probable that there is exchange of causation between the two phenomena.

One guarded notion is that oral health is potentially a modifiable aspect of healthy ageing and could impact systemic and cognitive resilience. Prevention dentistry, periodontal treatment, salivary control and caregiver-assisted hygiene are appropriate at this time. Longitudinal and interventional confirmation is needed for microbiome specific therapies and for claims of dementias to be preventable.

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