

Dental Health and Dementia: More than Forgetfulness



Course Author(s): Maria L. Geisinger, DDS, MS; Maggie A. Misch, BS

CE Credits: 2 hours

Intended Audience: Dentists, Dental Hygienists, Dental Assistants, Dental Students, Dental Hygiene Students, Dental Assisting Students

Date Course Online: 05/01/2019

Last Revision Date: 12/15/2022

Course Expiration Date: 12/14/2025

Cost: Free

Method: Self-instructional

AGD Subject Code(s): 490, 730, 750

Online Course: www.dentalcare.com/en-us/professional-education/ce-courses/ce566

Disclaimer: Participants must always be aware of the hazards of using limited knowledge in integrating new techniques or procedures into their practice. Only sound evidence-based dentistry should be used in patient therapy.

Conflict of Interest Disclosure Statement

- Dr. Geisinger has been or is currently a co-investigator and/or principal investigator on research funded entirely or in part by The Proctor & Gamble Company. All funds were used for research endeavors and not for personal gain. Dr. Geisinger has not accepted any payment from dentalcare.com for participation in this continuing education course.
- Ms. Misch reports no conflicts of interest associated with this course.

Introduction – Dementia

Dental Health and Dementia: More than Forgetfulness seeks to improve the dental care provider's understanding of the interaction between periodontal disease, dental caries, and dementia. It will also serve as an aid in the clinical decision-making process to optimize dental health for patients with dementia and periodontal disease.

Course Contents

- Overview
- Learning Objectives
- Introduction
- Epidemiology and Classification of Dementia
 - Prevalence and Distribution of Patients with Dementia
 - Are All Dementias the Same?
 - Cognitive Impairment Classifications
- Epidemiology and Pathogenesis of Periodontal Disease
- Proposed Mechanisms of Interaction between Periodontal Disease and Dementia
 - Common Pathways of Immune Response and Inflammatory State
 - Microbial Interactions between Periodontal Disease and Dementia
- Oral Hygiene in Patients with Dementia
 - Delivery of Oral Home Care by Primary Caregivers
 - Adjunctive Therapies to Improve Oral Health in Patients with Dementia
- Interdisciplinary Care for Individuals in Residential Nursing Facilities
- Summary
- Course Test
- References
- About the Authors

Overview

This course seeks to improve the dental care provider's understanding of the interaction between periodontal disease, dental caries, and dementia. It will also serve as an aid in the clinical decision-making process to optimize dental health for patients with dementia and periodontal disease.

Learning Objectives

Upon completion of this course, the dental professional should be able to:

- Understand the current scientific literature about the prevalence, etiology, and stages of dementia.
- Recognize and discuss with patients the association of periodontal diseases and oral bacteria with dementia and Alzheimer's Disease.
- Develop and implement strategies for communication and delivery of oral hygiene and dental care to patients suffering with dementia.
- Evaluate patients' risk factors and oral

hygiene to develop effective intervention and treatment modalities to reduce caries and periodontal disease rates.

- Understand the utility of nonsurgical caries treatment (e.g., topical fluoride, silver diamine fluoride, etc.) in patients with moderate to advanced dementia.
- Deliver instructions to primary care providers for patients with moderate to advanced dementia to increase compliance with oral home care and decrease care resistant behaviors.

Introduction

It is well-established that cognitive impairment and loss of motor skills in elderly individuals can have a direct effect on oral health and the progression and/or severity of oral diseases, including dental caries and periodontal diseases.¹ Multiple recent reports have linked periodontitis, periodontal pathogens and byproducts, and tooth loss with dementias, including Alzheimer's disease and other dementias.¹⁻³ A landmark study published in 2019 identified the presence of enzymes (gingipains) secreted by a bacteria commonly thought to be one of the keystone pathogens for periodontitis, *Porphyromonas gingivalis* (P.g.), in the brain tissues of individuals with pathology and symptoms of Alzheimer's Disease at higher levels than in the brains of individuals without such symptoms.⁴ This report has re-ignited interest in the medical and lay communities in the potential role of periodontal diseases in development of Alzheimer's and other dementias. It is, however, important to note that these findings are based upon a cross-sectional analysis of gingipain presence in tissues, which were found in 96% of all tissues assessed.⁴ This study builds upon earlier animal studies indicating that chronic oral application of P.g. bacteria or the gingipains produced by P.g. increase the production of amyloid beta, a protein indicated in the development of Alzheimer's Disease.⁵ Further prospective randomized controlled trials are necessary to identify causation and/or common disease pathways between dementia and periodontitis. However, emerging evidence suggests that periodontal health may be critical in this population.

Periodontitis has been associated with dementia in epidemiologic studies; patients

with periodontal disease are up to 70% more likely to present with Alzheimer's Disease than those who are periodontally healthy.^{6,7} It has been postulated that this relationship may be due to poorer oral hygiene over time due to deficits associated with dementia, dementia patients' resistance to caregiver delivery of oral care resulting in a reduction of oral hygiene delivery, medication induced xerostomia, or other challenges associated with oral hygiene delivery for patients with dementia leading to larger dental plaque masses and/or more pathologic intraoral bacteria.⁴⁻⁷ It has also been hypothesized that this interaction may be mediated by increases in local and systemic inflammation associated with both diseases, a dysbiotic oral microbiome, and/or immune reactions to oral pathogens.^{8,9} Currently more than 55 million people live with dementia worldwide, and there are nearly 10 million new cases every year.¹⁰ Furthermore, global estimates suggest that the total economic costs caused by dementia increased from US \$279.6 billion in 2000 to \$948 billion in 2016, with an annual growth rate of nearly 16%.¹¹ It is also notable that the older segment of the population are retaining more teeth and retaining teeth for longer. Total edentulism in the US population is decreasing. Complete edentulism is expected to reach as low as 2.6% by 2050.¹² This represents a 30% decrease in complete edentulism, after we account for estimations of population growth and aging.¹² These changes in tooth retention may have a significant impact on the oral health status of older individuals.

Given the projected increased numbers of teeth in the elderly and the prevalence of dementia in that population, we can assume that more and more individuals with dementia will experience oral health challenges. Further, dementia and its sequelae and treatment may impact oral health and oral disease, in particular periodontitis, have the potential for bidirectional influence. For example, medications for dementia often increase symptoms of xerostomia and higher caries rates are seen in patients with dementia, in particular those with moderate to severe disease and/or those that reside in residential nursing care facilities.^{13,14} Conversely, periodontitis has been

implicated in dementia disease progression.⁴⁻⁷ Because of these interactions, it is critical that dental healthcare professionals are familiar with the medical impacts of oral disease and are able to counsel patients and caregivers about effective oral hygiene practices and interventions for dementia patients with caries and/or periodontal diseases.¹⁵⁻¹⁷

Degeneration of cognitive function has been associated with worsening oral health parameters, including dental caries and periodontitis.^{1,18-20} Large-scale epidemiologic studies have also shown that individuals with increased systemic inflammation, including elevated proinflammatory markers, have a higher risk of developing dementia and more rapid progression of dementia symptoms over time.²¹⁻²³ Given that periodontal diseases are initiated by dysbiotic biofilm, which then induces an inflammatory response that ultimately is responsible for soft and hard tissue destruction in the local periodontal environment. This inflammation-induced tissue destruction results in periodontal pocket formation, alveolar bone resorption, and finally, tooth loss.^{24,25}

In recent reports, periodontitis-associated local and systemic elevated inflammatory markers and dysbiotic oral bacteria/bacterial byproducts have been linked to dementia.^{1-15,26-28} Dementia also presents additional behavior management challenges. Patients with dementia and their caregivers must manage delivery of preventative oral hygiene measures, which can be increasingly difficult as dementia severity increases.^{29,30} Due to the number of individuals affected by both periodontal disease³¹ and dementia¹⁰ and the chronic, progressive nature of both diseases, the understanding of the interaction between periodontal disease and dementia and treatment strategies for promotion of optimal oral health in patients suffering with dementia is of utmost importance to the dental practitioner.

Epidemiology and Classification of Dementia

Dementia is not a natural consequence of aging or even extreme forgetfulness. It is a generally progressive chronic syndrome in which patients

experience a deterioration in memory, thinking, behavior, and the ability to perform everyday activities.¹⁰ Dementia is described in the International Classification of Disease version 10 (ICD-10) as:

A syndrome due to disease of the brain, usually of a chronic or progressive nature, in which there is a disturbance of multiple higher cortical functions, including memory, thinking, orientation, comprehension, calculation, learning capacity, language, and judgement. Consciousness is not clouded. The impairments of cognitive function are commonly accompanied, and occasionally preceded, by deterioration in emotional control, social, behavior, or motivation. This syndrome occurs in Alzheimer's disease, in cerebrovascular disease, and in other conditions primarily or secondarily affecting the brain.³²

Nearly 10 million individuals are diagnosed with new cases of dementia each year and that number is expected to increase with increasing life expectancy worldwide.¹⁰ Furthermore, it is estimated the number of people with dementia will increase from an estimated 57.4 (50.4 to 65.1) million cases globally in 2019 to an estimated 152.8 (130.8 to 175.6) million cases in 2050.¹¹

Prevalence and Distribution of Patients with Dementia

Dementia rates are growing rapidly throughout the world, which reflects an aging global population.³³ Neurologic conditions, including dementia, were estimated by the Global Burden of Disease 2010 Study as the third leading cause of years lived with disability at global level.³⁴

The prevalence of dementia increases dramatically in the older elderly population. Approximately 2-3% of those aged 70-75 years are affected with dementia, whereas 20-25% of individuals aged 85 years or more have some form of the disease.³⁴ Data tracking prevalence in individuals over the age of 85 is limited, and therefore, it is not currently clear if dementia prevalence keeps increasing or stabilizes in these individuals.³⁶

Are All Dementias the Same?

Alzheimer's disease is the most common form of dementia. Approximately two out of every three cases of dementia is related to Alzheimer's disease. Other common dementias include: vascular dementia (up to 20% of patients with dementia), dementia with Lewy bodies (approximately 15% of all dementia cases), and frontotemporal dementia (less than 5% of dementia cases).³⁷ In many cases the diagnostic criteria for these dementias overlap and patients may present with symptoms for multiple forms of dementia.¹⁰ Further, dementia may be a comorbidity with other diseases.¹⁰ Finally, a small number of dementia cases are associated with less common underlying causes. These may include Parkinson's Disease Dementia, Huntington's Disease, Creutzfeldt-Jakob Disease and other Prion Diseases, Dementia in HIV/AIDS, Traumatic Brain Injury, and Wernicke-Korsakoff Syndrome, which includes dementia from alcohol abuse.³⁸

Dementia generally does not have one distinct cause. It is well-established that dementias, especially the most common forms, have a variety of risk factors and that these risk factors may vary throughout life. Educational attainment in childhood and early adulthood, is protective against dementia, i.e., patients with higher levels of education demonstrated delayed dementia onset once they are of older age.³⁹ Additionally, hypertension, type 2 diabetes mellitus, hyperlipidemia, decreased cognitive activity, social isolation, lack of exercise, alcohol use, dietary factors, and smoking have also been associated with dementia development.^{39,40} Late-onset Alzheimer's is the most common form of dementia. This dementia is considered to be multifactorial with disease development due to genetic, lifestyle, and environmental factors. Several genes and genetic polymorphisms have been identified as playing a role in the development of Alzheimer's Disease. Such genes may convey an increased risk through mechanisms that include: increasing the production of presenilin and b-amyloid precursor protein (BAPP) and enhancing the deposition of b-amyloid protein in brain tissue.⁴¹ In addition to these genetic risk factors, environmental/lifestyle factors may

induce epigenetic alterations of the genome (modification of the genome to turn genes off), which could increase risk for dementia development in some patients.⁴² Lastly, destruction of the blood-brain barrier may result in increased permeability which can lead to introduction of neurotoxic substances, including bacteria and bacterial byproducts, into brain tissues which have the potential to contribute to neurodegenerative diseases.⁴³

Cognitive Impairment Classifications

Dementia symptoms generally vary in severity and progress over time from no impairment to very severe dementia. Due to the heterogeneity in early symptoms and the differing speed of disease progression among individuals, early detection of dementias can be challenging. Both the Global Deterioration Scale/Reisberg Scale and the Functional Assessment Staging Test (FAST) assign seven stages to the progression of dementia (Figure 1).⁴⁴ Familiarity with these stages and their associated symptoms may help dental healthcare providers identify at-risk individuals and refer them for cognitive screening at early timepoints in their dementia disease.

Epidemiology and Pathogenesis of Periodontal Disease

Periodontal diseases include inflammatory diseases of the supporting structures around the teeth--the gingiva, periodontal ligament, alveolar bone, and cementum.^{45,46} The two most common forms of periodontal disease are gingivitis and periodontitis.³¹ Gingivitis is a non-specific inflammatory disease associated with the accumulation of dysbiotic bacterial biofilm.⁴⁷ All patients are susceptible to gingivitis with cessation of oral hygiene measures. Further, for many patients, gingivitis is a precursor to more serious, irreversible forms of periodontal diseases.⁴⁷ Removal of biofilm and local etiologic factors results in the reversal of gingivitis symptoms and reduces local and systemic levels of inflammatory markers in patients with gingivitis.⁴⁷⁻⁴⁹

Periodontitis is generally believed to be a bacterially-initiated disease in which the host immune-inflammatory response results in destruction of the and soft tissues supporting the teeth.^{50,51} Periodontal disease progression is generally slow to moderate. Average clinical progression of periodontal disease is

Diagnosis	Stage	Signs and Symptoms
No Dementia	Stage 1: No Cognitive Decline	In this stage a person functions normally and has no memory loss and is mentally healthy. People without dementia would be in stage 1.
No Dementia	Stage 2: Very Mild Cognitive Decline	This stage is used to describe normal forgetfulness associated with aging. For example, forgetting names and where familiar objects were left. Symptoms of dementia are not evident to the individual or their physician.
No Dementia	Stage 3: Mild Cognitive Decline	This stage includes increased forgetfulness, slight difficulty concentrating, and decreased work performance. People may get lost more frequently or have difficulty finding the right words. At this stage, a person's loved ones will begin to notice a cognitive decline.
Early Stage	Stage 4: Moderate Cognitive Decline	This stage includes difficulty concentrating, decreased memory of recent events, and difficulties managing finances or traveling alone to new locations. People have trouble completing complex tasks efficiently or accurately and may be in denial about their symptoms. They may also start withdrawing from family or friends because socialization becomes difficult. At this stage, a physician can detect clear cognitive problems during a patient interview and exam.
Mid-Stage	Stage 5: Moderately Severe Cognitive Decline	People in this stage have major memory deficiencies and need some assistance to complete their daily living activities (dressing, bathing, preparing meals, etc.). Memory loss is more prominent and may include major relevant aspects of current lives. For example, people may not remember their address or phone number and may not know the time of day or where they are.
Mid-Stage	Stage 6: Severe Cognitive Decline (Middle Dementia)	People in Stage 6 require extensive assistance to carry out their Activities of Daily Living (ADLs). They start to forget names of close family members and have little memory of recent events. Many people can remember only some details of earlier life. Individuals also have difficulty counting down from 10 and finishing tasks. Incontinence (loss of bladder or bowel control) is a problem in this stage. Ability to speak declines. Personality / emotional changes, such as delusions (believing something to be true that is not), compulsions (repeating a simple behavior, such as cleaning), or anxiety and agitation may occur.
Late-Stage	Stage 7: Very Severe Cognitive Decline (Severe Dementia)	People in this stage have essentially no ability to speak or communicate. They require assistance with most activities (e.g., using the toilet, eating). They often lose psychomotor skills. For example, the ability to walk.

approximately 0.1mm of attachment loss and 0.2 teeth lost annually.⁵² In longitudinal investigations, groups with fastest and slowest disease progression differed considerably with regard to demographics and underlying health conditions.⁵² Increased rapidity of attachment loss is associated with decreased access to comprehensive dental care as well as certain local and/or systemic factors.⁵² In an updated classification system from the American Academy of Periodontology (AAP) and European Federation of Periodontology (EFP), individuals are classified with a Stage and Grade to characterize disease severity and risk of future disease progression.^{51,53} Periodontitis Stages I-IV are determined based upon a patient's current disease presentation, including attachment loss, alveolar bone levels, and tooth loss, and the Stage may be modified by case complexity.^{51,53} Periodontitis Grades A-C are selected based upon risk factors and direct or indirect evidence of the pace of disease progression.^{51,53} The prevalence of periodontitis has been estimated to be over 42% of U.S. adults over 30 years of age.³¹ Of those individuals, 7.8% had severe periodontitis.³¹ Further, severe periodontitis was most prevalent among adults 65 years or older, Mexican Americans, non-Hispanic blacks, and smokers.³¹ In fact, periodontitis prevalence among US adults is nearly 4-fold greater than that of diabetes mellitus⁵⁴ and over 6-fold greater than that of coronary artery disease.⁵⁵ Given the highly prevalent nature of periodontitis, identification of periodontal disease and consideration during assessment and therapy for systemic diseases is critical. Disease progression and tissue destruction occurs through host-mediated inflammatory pathways,⁵⁶ which may vary based upon genetic and other risk factors.⁵⁷⁻⁶⁰ The result is a chronic immune-inflammatory disease that may pose a significant systemic burden for individuals.⁶¹

Proposed Mechanisms of Interaction between Periodontal Disease and Dementia

Periodontitis has been linked to cognitive impairment and dementia after controlling for confounding factors such as age, sex, and educational attainment.^{4,18,20,62-65} Periodontal disease progression is characterized by dysbiosis and elevated chronic inflammation,

and dementia and Alzheimer's Disease have been linked to periodontal disease through both inflammatory and bacterial mechanisms.^{20,27,28,66-71} Periodontitis has also been linked to increased systemic and neurological markers of Alzheimer's disease.^{20,72,73}

Common Pathways of Immune Response and Inflammatory State

Chronic inflammation is correlated with the onset and progression of Alzheimer's Disease, and it has been postulated that chronic inflammation and neuronal aging induces stress and neuropathological changes.⁷⁴ In this model, chronic inflammation primes the microglia and induces a hyperreactive state, which then results in a failure to clear misfolded or damaged neuronal proteins and enhances the aggregation of neuronal proteins associated with dementia, such as A β 1-42.^{75,76} Similarly, periodontal tissue breakdown seen in periodontitis is a result of host inflammatory response to bacterial stimuli. Periodontal tissue breakdown is mediated by pro-inflammatory cytokines and mediators such as interleukin-1 β (IL-1 β), interleukin-6 (IL-6), tumor necrosis factor- α (TNF- α), prostaglandin E2 (PGE2), receptor activator of nuclear factor kappa B ligand (RANKL), and matrix metalloproteinases (MMPs). These pro-inflammatory mediators interact with bacteria and the surrounding tissues. The heterogeneity among individuals in this response can influence disease susceptibility and severity.⁷⁷ Additionally, periodontal disease severity is correlated to increased levels of pro-inflammatory mediators systemically.⁷⁸⁻⁸⁰ Because inflammation may influence the progression of disease in both periodontitis and dementia, one mechanism of interaction between periodontitis and dementia may include increased levels of inflammation and their influence on neuronal function.

Microbial Interactions between Periodontal Disease and Dementia

Oral bacteria and bacterial byproducts have been found in brain tissue of patients with dementia at higher rates than in healthy controls.^{4,5,8,71} Additionally, serum antibodies to periodontopathogenic bacteria have been found to be increased in patients with

Alzheimer's disease and dementia when compared to healthy individuals.^{23,27,69} The question remains: how do these bacteria enter the brain? It has been proposed that such transport of bacteria into human neural tissues could occur through one of several mechanisms: 1) bacteremias are seen in patients with gingival inflammation and/or periodontitis, which may put the bacteria in proximity to the brain and in cases of a weakened blood-brain barrier (due to age, chronic infection, or inflammation) bacteria may cross the blood-brain barrier, 2) direct access to the brain could be accessed by bacteria entering perivascular spaces, and 3) oral bacteria may translocate to the brain directly through olfactory and/or trigeminal nerve pathways.⁸¹ Further, bacterial byproducts, including virulence factors such as lipopolysacchride, capsular material, proteolytic enzymes, and gingipains, may cross the blood-brain barrier through similar pathways and contribute to demential progression.^{1,2,20,62,82,83}

A less-well investigated method for the interaction between periodontal disease and dementia may include alteration of gene expression caused by exposure to bacteria and their byproducts. In these cases, epigenetic alterations of intracellular DNA may change gene expression and up- or down-regulate the proteins that they code for. Bacteria and their byproducts increase DNA methylation and histone acetylation, which has been implicated in the development of many diseases.^{84,85}

Oral hygiene status in elderly dentate patients is inversely proportional to dementia severity and poor oral hygiene is associated with dementia development.⁸⁶ Reduction of the overall microbial burden, as well as a shift to a more eubiotic microbiome may be achieved through oral hygiene delivery.^{87,88} This decreased microbial burden could lead to reduced bacteremias and therefore a reduction in the likelihood of bacteria and their byproducts crossing the blood-brain barrier.^{4,5,89} Improved oral health, including advanced oral hygiene practices has been suggested as an intervention to reduce the direct and indirect influences of oral bacteria and their byproducts on the brain and their subsequent potential influence on cognitive decline.⁹⁰

Oral Hygiene in Patients with Dementia

Oral bacterial biofilms are living entities that can transform into inhospitable environments given the right circumstances.^{86,91,92} Coaggregation of periodontal pathogens allows for innocuous local commensal bacteria to transform in the presence of more virulent pathogens.⁹² This overall increase in virulence of the bacterial biofilm allows for a shift in the balance in the local and systemic environments from homeostasis to dysbiosis, which can then impair the host immune surveillance and cause dysfunction in the inflammatory repair mechanisms.⁹² A recent review of the literature concludes that the oral health and hygiene of elderly individuals with dementia is inadequate and should be improved through oral health education of formal and informal caregivers.^{93,94} Given the importance to oral and overall health of maintaining optimal oral hygiene, it is critical for individuals with dementia to have effective oral hygiene measures performed by them or for them by caregivers.

Delivery of Oral Home Care by Primary Caregivers

Common oral health problems in older individuals include: caries, periodontal disease, xerostomia, candidiasis, and mucosal lesions.^{95,96} All of these conditions show an increased prevalence associated with age, likely due to age-related aggregation of risk factors, polypharmacy, and decline in dexterity leading to decreased efficiency in plaque removal.⁹⁶⁻¹⁰⁰ These conditions are also more prevalent in individuals with cognitive impairment or dementia.^{88,89} Older patients are also more likely to experience dysphagia, difficulty swallowing, which can make performing dental procedures in these patients more complex.^{101,102}

Individuals with dementia who reside in nursing homes often have poor oral hygiene associated with gingivitis and periodontitis.^{101,102} and oral hygiene practices are likely to be omitted or provided with chemical anti-plaque agents rather than toothbrushes. A failure to mechanically remove dental plaque on a regular basis is significantly less effective in preventing gingival inflammation and caries than mechanical plaque removal.¹⁰³⁻¹⁰⁵ Because more than half of individuals with dementia

rely on caregivers for many activities of daily living, including oral hygiene delivery, they are dependent on the knowledge and skills of others for their preventative oral health care.¹⁰⁶

One obstacle often encountered by primary caregivers is care resistant behavior (CRB) to oral hygiene delivery in patients with dementia.¹⁰⁷ Care resistant behavior is defined as behaviors with which persons with dementia withstand or oppose the helping efforts of a caregiver and can be categorized as “uncooperative,” “disruptive,” or “agitation.”¹⁰⁸⁻¹¹³ Eighty percent of certified nursing assistants (CNAs) report CRBs in response to the delivery of oral hygiene,¹¹⁴ which have been shown to be reduced by increasing caregivers’ awareness of best practices for oral hygiene,¹¹⁵⁻¹¹⁷ recognition of CRBs,^{107,118} and strategies to reduce threat perception in patients with dementia.^{16,116,119,120} Some especially effective strategies for threat reduction during delivery of oral hygiene measures include: smiling, bridging (e.g., having the elderly patient hold a toothbrush while the caregiver is delivering oral hygiene), and the use of polite, one-step commands.^{16,119} Inclusion of these simple steps in the oral hygiene routine of caregivers can increase cooperation for elderly patients with dementia significantly and improve oral hygiene quality and quantity.^{16,119}

Adjunctive Therapies to Improve Oral Health in Patients with Dementia

Caries incidence increases in older adults and root caries are a particular problem due to gingival recession, xerostomia, and poor plaque removal.^{89,121} These problems are particularly pronounced in patients with dementia who are unable to care for their teeth themselves, may be taking multiple medications that cause xerostomia, and may not be able to receive treatment for caries in an outpatient dental office.^{121,122} Silver diamine fluoride (SDF) is an alkaline topical solution containing fluoride and silver that has been approved by the FDA for both caries arrest and root desensitization.^{123,124} SDF is effective in caries arrest through remineralization and inhibition of collagenases and cysteine cathepsins.¹²⁵ Yearly SDF application on exposed radicular surfaces has been shown to be effective in reducing caries progression when compared to fluoride varnish

and placebo with rare complaints about the dark staining that is seen with SDF.¹²⁶ Given the low cost of SDF, the infrequency of application, the relative non-invasiveness of treatment, and the simplicity of the therapy, SDF application should be considered as a preventive measure for oral health promotion in older adults with dementia.

Many patients with dementia require care for their activities of daily living, including oral hygiene.¹¹⁰ Coordination of care between oral healthcare providers, nursing staff, and family caregivers is critical to insure optimal oral health for those patients. Understanding the role oral bacteria and their byproducts may play in the development and progression of dementia allows for better care delivery to patients. Furthermore, utilization of mechanisms to reduce CRBs when oral hygiene is delivered, as well as coordination of preventative dental care within the residential nursing home environment, are integral components for maintenance of oral and overall health for patients.

Interdisciplinary Care for Individuals in Residential Nursing Facilities

A multi-disciplinary approach should be employed for effective oral care in patients with dementia. Dentists, dental hygienists, dental assistants, physicians, nurses, nursing assistants, and familial and other caregivers each have a unique role in providing oral health care to these patients.

Dental healthcare providers play a central role in creating a customized, long-term dental care plan for a patient that has been newly diagnosed with dementia.¹²⁵ The dentist should attempt to make a plan that includes patient-specific information and involve the individual with dementia and their family while he or she is in the early stages of dementia.¹²⁶ This may allow for increased buy-in and autonomy in treatment decisions from the patient who may then be more open to treatment and more engaged in making decisions. The plan should concentrate on eliminating pain, managing infection, and preventing new disease.¹²⁷

Education of nurses, nursing assistants, and caretakers is critical to ensure the success of proper oral care in patients with dementia.¹²⁸ Dental prostheses are growing increasingly

diverse, including natural teeth, fixed bridges, dental implants, and removable prostheses.^{11,129} Natural teeth require brushing and interdental cleaning,¹¹⁷ while dentures need to be removed to be cleaned.¹¹⁶ Dental implants also require thorough preventative care to prevent inflammation and bone loss.¹¹⁶ Many nurses, CNAs, and other caretakers lack training in the complex care required for different dental prostheses.¹²⁸ In addition, CRBs exhibited by patients with dementia are a major obstacle to providing effective oral care.¹⁶ Offering strategies to improve oral hygiene delivery to front-line care providers is crucial as they are the primary individuals delivering oral care.¹⁶ Strategies offered may include MOUTH interventions (Managing Oral Hygiene Using Threat Reduction).¹⁶ MOUTH intervention is a nonpharmacologic, relationship-based intervention.¹⁶ Some tactics include forming a connection by approaching the patient at or below eye level, using a friendly and calm attitude, and using brief, one-step commands.^{16,119} Care providers may have a better chance of completing mouth-care activities if they are aware of different approaches that reduce CRBs in patients with dementia.¹⁶

Due to the progressive nature of dementia, verbal communication between the patient and the healthcare provider or caretaker may decrease as the disease progresses.¹²⁷ Consequently, verbal communication may no longer be the best way to recognize health problems, such as tooth pain. Healthcare professionals and caretakers should be aware of non-verbal cues of dental discomfort.¹²⁹ These include avoiding meals and being disinterested in eating, chewing of the lip, tongue or hands, “pulling” at the face, not wearing dentures, and aggression (particularly during activities of daily living, including oral hygiene).¹²⁹

Healthcare professionals and caretakers should also consider their patient’s progression and stage of dementia. The different stages of dementia can affect how a patient will cope

with dental treatments and interventions.¹²⁹ Healthcare professionals and caretakers must be aware that strategies that are effective in one patient, may not be for others.¹²⁹ Furthermore, a strategy that may be effective for a patient one day, may not work the next week for that same patient.¹³³ Healthcare professionals and caretakers should recognize that treatment plans may need to change over time as the patient’s disease state changes.¹²⁹

Oral hygiene must be seen as an integral part of a patient with dementia’s overall wellness and healthcare. An interdisciplinary approach allows the benefits of effective oral health to be achieved through delivery of regular oral hygiene and regular evaluation of oral health. Benefits of an increased focus on oral health may include decreased tooth loss, improved oral cancer detection, and decreased oral pain and infections. Patients with dementia may also see improvements in nutrition, decreased agitation associated with discomfort, overall health, and appearance with improved oral health.

Summary

Both the inflammation and oral bacteria and their byproducts associated with periodontitis have been implicated as able to travel from the mouth to brain tissues and implicated in the development and disease progression of dementia and Alzheimer’s disease. Because these diseases may interact, it is critically important for older adults to proactively maintain their oral health. Oral care delivery for patients with dementia can be difficult due to lack of education about oral healthcare and inability to address care resistant behaviors in patients with dementia. Older individuals with dementia also experience higher rates of caries, xerostomia and multimorbidity associated with their dementia symptoms and/or pharmacotherapies. Delivery of preventative and minimally invasive care for periodontitis and caries within a nursing home environment is challenging, but simple interventions and coordination between interdisciplinary healthcare providers can improve outcomes for patients.

Course Test Preview

To receive Continuing Education credit for this course, you must complete the online test. Please go to: www.dentalcare.com/en-us/professional-education/ce-courses/ce566/test

- 1. A 2019 study examining brain tissue from elderly individuals found that gingipains from *P. gingivalis* were present at _____ levels in brains from individuals with Alzheimer's Disease than those who did not have Alzheimer's Disease.**
 - A. Higher
 - B. Lower
 - C. Similar
- 2. Approximately how many people are diagnosed with dementia every year worldwide?**
 - A. 2 million
 - B. 5 million
 - C. 7 million
 - D. 10 million
- 3. What was the economic impact of dementia in the United States in 2015?**
 - A. \$818 million
 - B. \$8.1 billion
 - C. \$81 billion
 - D. \$818 billion
- 4. Dementia is the _____ leading cause of years lived with a disability.**
 - A. second
 - B. third
 - C. fifth
 - D. tenth
- 5. All of the following have been associated with the development of dementia, EXCEPT:**
 - A. Smoking
 - B. Alcohol use
 - C. Hypertension
 - D. Educational attainment
 - E. All of the above have been associated with dementia development.
- 6. Average clinical progression of periodontal disease is approximately ___ mm of attachment loss annually.**
 - A. 0.1
 - B. 0.5
 - C. 1.0
 - D. 2.0
- 7. Approximately _____ of U.S. adults over 30 years old suffer from periodontal disease.**
 - A. 24%
 - B. 38%
 - C. 42%
 - D. 65%

8. **Chronic inflammation influences the progression of dementia. It has been hypothesized that inflammation primes the microglia and increases the production of neuronal proteins associated with dementia.**
- A. Both statements are true.
 - B. The first statement is true, the second statement is false.
 - C. The first statement is false, the second statement is true.
 - D. Both statements are false.
9. **Pathways by which oral bacteria are thought to enter the brain include _____.**
- A. Bacteremias allow for bacteria within the bloodstream and chronic infection, age, and inflammation increase the permeability of the blood-brain barrier.
 - B. Bacteria may gain direct access to the brain through perivascular spaces
 - C. Bacteria may pass into the brain through olfactory and/or trigeminal nerve pathways
 - D. All of the above.
10. **Both caries and periodontal disease increase in prevalence with age and are common in older individuals. Factors that influence this increase in disease prevalence include _____.**
- A. Polypharmacy
 - B. Decline in dexterity leading to decreased efficiency in plaque removal
 - C. Aggregation of risk factors
 - D. All of the above.
11. **Care resistant behavior is defined as _____.**
- A. Persons with dementia withstand or oppose the helping efforts of caregivers
 - B. Caregivers that induce agitation in persons with dementia
 - C. Patients with dementia related to preference for one caregiver over another
 - D. Caregivers that decrease the effectiveness of healthcare delivery
12. **What percentage of certified nursing assistants report care resistant behaviors in response to oral hygiene delivery?**
- A. 30%
 - B. 45%
 - C. 60%
 - D. 80%
13. **Which of the following are mechanisms caregivers can implement to reduce care resistant behavior?**
- A. Smiling
 - B. Bridging
 - C. Using polite, one-step commands
 - D. All of the above.
14. **All of the following are true about Silver Diamine Fluoride (SDF), EXCEPT:**
- A. SDF must be applied monthly to arrest radicular caries in adults
 - B. SDF is an alkaline topical solution containing fluoride and silver
 - C. SDF has been approved by the FDA for dentinal sensitivity and caries arrest
 - D. SDF may cause black staining on carious teeth

- 15. SDF causes caries arrest by increasing fluoride availability and remineralization. SDF also inhibits of collagenases and cysteine cathepsins.**
- A. Both statements are true.
 - B. The first statement is true, the second statement is false.
 - C. The first statement is false, the second statement is true.
 - D. Both statements are false.
- 16. Which of the following are non-verbal signs of oral discomfort that may be recognized in a non-verbal patient with dementia?**
- i. Chewing of lip or tongue**
 - ii. Pulling at face or lips**
 - iii. Refusing denture wear**
 - iv. Aggression in response to oral-related activities of daily living**
- A. i, iii
 - B. i, ii, iii
 - C. i, ii, iii, iv
 - D. iv only
- 17. Involvement of the interdisciplinary healthcare team in the evaluation and delivery of oral health can result in _____ in patients with dementia?**
- A. decreased tooth loss
 - B. decreased oral pain and infections
 - C. increased identification of oral cancer
 - D. All of the above.
- 18. In patients with dementia, improved oral health may lead to improvements in overall health as well. Which of the following is not seen with improved oral health in patients with dementia?**
- A. Improved nutritional intake
 - B. Worsening sleep patterns
 - C. Decreased agitation associated with discomfort/pain
 - D. Improved appearance

References

1. Ryder MI, Xenoudi P. Alzheimer disease and the periodontal patient: New insights, connections, and therapies. *Periodontol 2000* 2021; 87: 32-42.
2. Ryder MI. *Porphyromonas gingivalis* and Alzheimer disease: Recent findings and potential therapies. *J Periodontol* 2020; 91(Suppl): S45-S49.
3. Thomson WM, Barak Y. Tooth loss and dementia: A critical examination. *J Dent Res* 2021; 100(3): 226-231.
4. Dominy SS, Lynch C, Ermini F, et al. *Porphyromonas gingivalis* in Alzheimer's disease brains: Evidence for disease causation and treatment with small-molecule inhibitors. *Sci Adv.* 2019 Jan 23;5(1):eaau3333. doi: 10.1126/sciadv.aau3333.
5. Ilievski V, Zuchowska PK, Green SJ, et al. Chronic oral application of a periodontal pathogen results in brain inflammation, neurodegeneration, and amyloid beta production in wild type mice. *PLoS One.* 2018 Oct 3;13(10):e0204941. doi: 10.1371/journal.pone.0204941.
6. Chen, CK, Wu YT, Chang YC. Association between chronic periodontitis and the risk of Alzheimer's disease: a retrospective, population-based, matched cohort study. *Alzheimers Res Ther.* 2017 Aug 8;9(1):56. doi: 10.1186/s13195-017-0282-6.
7. Kamer AR, Dasanayake AP, Craig RG, et al. Alzheimer's disease and peripheral infections: the possible contribution from periodontal infections, model and hypothesis. *J Alzheimers Dis.* 2008 May;13(4):437-49.
8. Kamer AR, Craig RG, Dasanayake AP, et al. Inflammation and Alzheimer's disease: possible role of periodontal diseases. *Alzheimers Dement.* 2008 Jul;4(4):242-50. doi: 10.1016/j.jalz.2007.08.004.
9. World Health Organization. Fact Sheets. Dementia. Accessed August 2, 2022.
10. Xu J, Zhang Y, Qiu C, Cheng F. Global and regional economic costs of dementia: a systematic review. *Lancet.* 2017; 390: S47.
11. Plassman BL, Langa KM, Fisher GG, et al. Prevalence of Dementia in the United States: The Aging, Demographics, and Memory Study. *Neuroepidemiology.* 2007;29(1-2):125-32.
12. Slade GD, Akinkugbe AA, Sanders AE. Projections of U.S. Edentulism Following 5 Decades of Decline. *J Dent Res.* 2014 Oct;93(10):959-65. doi: 10.1177/0022034514546165.
13. Zenthöfer A, Baumgart D, Cabrera T, et al. Poor dental hygiene and periodontal health in nursing home residents with dementia: an observational study. *Odontology.* 2017 Apr;105(2):208-213. doi: 10.1007/s10266-016-0246-5.
14. Zimmerman S, Austin S, Cohen L, et al. Readily Identifiable Risk Factors of Nursing Home Residents' Oral Hygiene: Dementia, Hospice, and Length of Stay. *J Am Geriatr Soc.* 2017 Nov;65(11):2516-2521. doi: 10.1111/jgs.15061.
15. Jablonski RA, Kolanowski A, Therrien B, et al. Reducing care-resistant behaviors during oral hygiene in persons with dementia. *BMC Oral Health.* 2011 Nov 19;11:30. doi: 10.1186/1472-6831-11-30.
16. Jablonski RA, Kolanowski AM, Azuero A, et al. Randomised clinical trial: Efficacy of strategies to provide oral hygiene activities to nursing home residents with dementia who resist mouth care. *Gerodontology.* 2018 Dec;35(4):365-375. doi: 10.1111/ger.12357.
17. Jablonski R, Winstead V, Azuero A, et al. Feasibility of Providing Safe Mouth Care and Collecting Oral and Fecal Microbiome Samples from Nursing Home Residents with Dementia: Proof of Concept Study. *J Gerontol Nurs.* 2017 Sep 1;43(9):9-15. doi: 10.3928/00989134-20170811-04.
18. Noble JM, Scarmeas N, Papapanou PN. Poor oral health as a chronic, potentially modifiable dementia risk factor: review of the literature. *Curr Neurol Neurosci Rep.* 2013 Oct;13(10):384. doi: 10.1007/s11910-013-0384-x.
19. Nilsson H, Berglund J, Renvert S. Tooth loss and cognitive functions among older adults. *Acta Odontol Scand.* 2014 Nov;72(8):639-44. doi: 10.3109/00016357.2014.882983.
20. Gil-Montoya JA, Sanchez-Lara I, Carnero-Pardo C, et al. Is periodontitis a risk factor for cognitive impairment and dementia A case control study. *J Periodontol.* 2015 Feb;86(2):244-53. doi: 10.1902/jop.2014.140340.
21. Schmidt R, Schmidt H, Curb JD, et al. Early inflammation and dementia: a 25-year follow-up of

- the Honolulu-Asia aging study. *Ann Neurol*. 2002 Aug;52(2):168-74.
22. Englehart MJ, Gerrling HI, Meijer J, et al. Inflammatory proteins in plasma and the risk of dementia: the rotterdam study. *Arch Neurol*. 2004 May;61(5):668-72.
 23. Kravitz BA, Corrada MM, Kawas CH. Elevated C-reactive protein levels are associated with prevalent dementia in the oldest-old. *Alzheimers Dement*. 2009 Jul;5(4):318-23. doi: 10.1016/j.jalz.2009.04.
 24. Flemmig TF. Periodontitis. *Ann Periodontol*. 1999 Dec;4(1):32-8
 25. Page RC, Schroeder HE. Pathogenesis of inflammatory periodontal disease. A summary of current work.
 26. Feres M, Teles F, Teles R, Figueiredo LC, Faveri M. The subgingival periodontal microbiota of the aging mouth. *Periodontol 2000*. 2016 Oct;72(1):30-53. doi: 10.1111/prd.
 27. Noble JM, Scarmeas N, Celenti RS, et al. Serum IgG antibody levels to periodontal microbiota are associated with incident Alzheimer disease. *PLoS One*. 2014 Dec 18;9(12):e114959. doi: 10.1371/journal.pone.0114959.
 28. Laugisch O, Johnen A, Maldonado A, et al. Periodontal Pathogens and Associated Intrathecal Antibodies in Early Stages of Alzheimer's Disease. *J Alzheimers Dis*. 2018;66(1):105-114. doi: 10.3233/JAD-180620.
 29. Volicer L, van der Steen JT, Frijters DH. Modifiable factors related to abusive behaviors in nursing home residents with dementia. *J Am Med Dir Assoc*. 2009 Nov;10(9):617-22. doi: 10.1016/j.jamda.2009.06.004.
 30. Ishii S, Streim JE, Saliba D. Potentially reversible resident factors associated with rejection of care behaviors. *J Am Geriatr Soc*. 2010 Sep;58(9):1693-700. doi: 10.1111/j.1532-5415.2010.03020.x.
 31. Eke PI, Thornton-Evans GO, Wei L, et al. Periodontitis in US Adults: National Health and Nutrition Examination Survey 2009-2014. *J Am Dent Assoc*. 2018;149(7):576-588.e6.
 32. World Health Organization. International Statistical Classification of Diseases and Related Health Problems 10th Revision; 2016. Accessed November 22, 2022.
 33. Kalra RN, Maestre GE, Arizaga R, et al. Alzheimer's disease and vascular dementia in developing countries: prevalence, management, and risk factors. *Lancet Neurol*. 2008 Sep;7(9):812-26. doi: 10.1016/S1474-4422(08)70169-8.
 34. Horton R. GBD 2010: understanding disease, injury, and risk. *The Lancet*. 2012;380(9859):2053-2054.
 35. Ferri CP, Prince M, Brayne C, et al. Global prevalence of dementia: a Delphi consensus study. *Lancet*. 2005 Dec 17;366(9503):2112-7.
 36. Forette F, Boller F. Hypertension and the risk of dementia in the elderly. *Am J Med*. 1991 Mar;90(3A):145-195.
 37. Alzheimer's Research UK. Dementia Statistics Hub. Different types of dementia. May 7, 2018. Accessed November 22, 2022.
 38. National Institute on Aging. Dementia Care Central. Lesser Known and Rare Types of Dementia. August 22, 2018. Accessed November 22, 2022.
 39. Hughes TF, Ganguili M. Modifiable midlife risk factors for late-life cognitive impairment and dementia. *Curr Psychiatry Rev*. 2009 May 1;5(2):73-92.
 40. Alzheimer's Society. What is Alzheimer's Disease? 2015. Accessed November 22, 2022.
 41. Van Cauwenberghe C, Van Broeckhoven C, Sleeger K. The genetic landscape of Alzheimer disease: clinical implications and perspectives. *Genet Med*. 2016 May;18(5):421-30. doi: 10.1038/gim.2015.117.
 42. Daniilidou M, Koutroumani M, Tsolaki M. Epigenetic mechanisms in Alzheimer's disease. *Curr Med Chem*. 2011;18(12):1751-6.
 43. Sweeney MD, Sagare AP, Zlokovic BV. Blood-brain barrier breakdown in Alzheimer disease and other neurodegenerative disorders. *Nat Rev Neurol*. 2018 Mar;14(3):133-150. doi: 10.1038/nrneurol.2017.188.

44. Dementia Care Central. Stages of Alzheimer's & Dementia: Durations & Scales Used to Measure Progression: GDS, FAST & CDR. September 1, 2018. Accessed November 22, 2022.
45. Flemmig TF. Periodontitis. *Ann Periodontol*. 1999 Dec;4(1):32-8.
46. Page RC, Schroeder HE. Pathogenesis of inflammatory periodontal disease. A summary of current work. *Lab Invest*. 1976 Mar;34(3):235-49.
47. Chapple ILC, Mealey BL, Van Dyke TE, et al. Periodontal health and gingival disease and conditions on an intact and a reduced periodontium: Consensus report of workgroup 1 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Periodontol* 2018; 89(Suppl 1): S74-S84.
48. Chapple ILC, Van der Weijden F, Doerfer C, et al. Primary prevention of periodontitis: managing gingivitis. *Proceedings of the 11th European Workshop on Periodontology*. *J Clin Periodontol* 2015; 42(Spec Iss 16): S71-S76.
49. Mombelli A. Microbial colonization of the periodontal pocket and its significance for periodontal therapy. *Periodontol 2000*. 2018 Feb;76(1):85-96. doi: 10.1111/prd.12147. Epub 2017 Nov 30.
50. AAP Glossary of Terms. Periodontitis. Accessed August 2, 2022.
51. Papapanou PN, Sanz M, Buduneli N, et al. Periodontitis: Consensus report of workgroup 2 of the 2017 Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Periodontol* 2018; 89(Suppl 1): S173-S182.
52. Needleman I, Garcia R, Gkraniias N, et al. Mean annual attachment, bone level, and tooth loss: A systematic review. *J Periodontol* 2018; 89(Suppl): S120-S139.
53. Tonetti MS, Greenwell H, Kornman KS. Staging and grading of periodontitis: Framework and proposal of a new classification and case definition. *J Periodontol* 2018; 89(Suppl 1): S159-S172.
54. Centers for Disease Control and Prevention (CDC). National Diabetes Statistics Report 2020. Estimates of Diabetes and its Burden in the United States.
55. Centers for Disease Control and Prevention (CDC). Heart Disease Facts. Available at: Accessed August 2, 2022.
56. Kornman KS, Page RC, Tonetti MS. The host response to the microbial challenge in periodontitis: assembling the players. *Periodontol 2000*. 1997 Jun;14:33-53.
57. Löe H, Anerud A, Boysen H, et al. Natural history of periodontal disease in man. Rapid, moderate and no loss of attachment in Sri Lankan laborers 14 to 46 years of age. *J Clin Periodontol*. 1986 May;13(5):431-45.
58. Michalowicz BS, Aeppli DP, Kuba RK, et al. A twin study of genetic variation in proportional radiographic alveolar bone height. *J Dent Res*. 1991 Nov;70(11):1431-5.
59. Michalowicz BS, Aeppli DP, Virag JG et al. Periodontal findings in adult twins. *J Periodontol*. 1991 May;62(5):293-9.
60. Kornman KS, Page RC, Tonetti MS. The host response to the microbial challenge in periodontitis: assembling the players. *Periodontol 2000*. 1997 Jun;14:33-53.
61. Winning, L., Linden, G. Periodontitis and systemic disease. *BDJ Team 2*, 15163 (2015).
62. Holmer J, Eriksdotter M, Schultzberg M, Pussinen P, Buhlin K. Association between periodontitis and risk of Alzheimer's disease, mild cognitive impairment, and subjective cognitive decline: A case control study. *J Clin Periodontol* Oct 5 [Epub Ahead of Print].
63. Gusman DJR, Mello-Neto JM, Alvex BES, Matheus HR< Ervolina E, Theodoro LH, de Almeida JM. Periodontal disease severity in subjects with dementia. *Arch Gerontol Geriatr* 2018; 76: 147-159.
64. Maldonado A, Laugisch O, Bürgin W, Schulean A, Eick S. Clincial periodontal variables in patients with and without dementia – a systematic review and meta-analysis. *Clin Oral Invest* 2018; Jun 22
65. Tonsekar PP, Jiang SS, Yue G. Periodontal disease, tooth loss, and dementia. Is there a link? A systematic review. *Gerodontol* 2012; 29: 36-42.
66. Miklossy J. Alzheimer's disease – a neurospichetosis. Analysis of evidence following Koch's and

- Hill's criteria. *J Neuroinflamm* 2011; 8: 90.
67. Van Charante EPM, Richard E, Eurelings LS, et al., Effectiveness of a 6-year multidomain vascular care intervention to prevent dementia (preDIVA): a cluster-randomised controlled trial. *The Lancet* 2016; 388: 797-805.
 68. Rivierre GR Riviere KH, Smith KS. Molecular and immunological evidence of oral *Treponema* in the human brain and their association with Alzheimer's disease. *Oral Microbiol Immunol* 2002; 17: 113-118.
 69. Kamer AR, Craig RG, Pirraglia E, et al. TNF-alpha and antibodies to periodontal bacteria discriminate between Alzheimer's disease patients and normal subjects. *J Neuroimmunol* 2009; 216: 92-97.
 70. Noble JM, Borrell JN, Papapanou PN, Elkind MS, Scarmeas N, Wright CB. Periodontitis is associated with cognitive impairment among older adults: analysis of NHANES-III. *J Nuerol Neurosurg Psychiatry* 2009; 80: 1206-1211.
 71. Poole S, Singhrao SK, Kesavalu L, Curtis MA, Crean S. Determining the presence of periodontopathic virulence factors in short term post-mortem Alzheimer's disease brain tissue. *J Alzheimers Dis* 2013; 36: 665-677.
 72. Kamer AR, Pirraglia E, Tsui W, et al. Periodontal disease associated with higher brain amyloid load in normal elderly. *Neurobiol Aging* 2015; 36: 627-633.
 73. Singharao SK, Chukkapalli S, Poole S, Velsko I, Crean SJ, Kesavalu L. Chronic *Porphyromonas gingivalis* infection accelerates the occurrence of age-related granules in ApoE-/- mice brains. *J Oral Microbiol* 2017; 9(1): 1270602. doi: 10.1080/20002297.2016.1270602.eCollection 2017.
 74. Krstic D, Knuesel I. Deciphering the mechanism underlying late-onset Alzheimer disease. *Nat Rev Neurol* 2013; 9: 25-34.
 75. Lim SL, Rodriguez-Ortiz C, Kitazawa M. Infection, systemic inflammation, and Alzheimer's disease. *Microbes Infect* 2015; 17: 549-556.
 76. Venegas C, Kumar S, Franklin BS, et al., Microglia-derived ASC specks cross-seed amyloid-beta in Alzheimer's disease. *Nature* 2017; 552: 355-361.
 77. Kinane DF, Preshaw PM, Loos BG. Host-response: understanding the cellular and molecular mechanisms of host-microbial interactions-consensus of the Seventh European Workshop on Periodontology. *J Clin Periodontol* 2011; 38(Suppl 11): 44-8.
 78. Loos BG. Systeic markers of inflammation in periodontitis. *J Periodontol* 2005; 76: 2106-2115.
 79. Noack B, Genco RJ, Tevisan M, Grossi S, Zambon JJ, De Nardin E. Periodontal infections contribute to elevated C-reactive protein level. *J Periodontol* 2005; 76: 2075-2084.
 80. Loos BG, Craandijj J, Hoek FJ, Weterhin-van Dillen PME, van der Velden W. C-reactive protein and other markers of systemic inflammation in relation to cardiovascular diseases are elevated in periodontitis. *J Periodontol* 2000; 71: 1528-1534.
 81. Olsen I, Singhrao SK. Can oral infection be a risk factor for Alzheimer's disease? *J Oral Microbiol* 2015; Sept 17; 7: 29143.
 82. Holt SC, Ebersole JL, *Prophyromonas gignivalis*, *Treponema denticola*, and *Tannerella forsythia*: the "red complex", a prototype polybacterial pathogenic consortium in periodontitis. *Periodontol* 2000 2005; 38: 72-122.
 83. Guo Y, Nguyen KA, Potempa J. Dichotomy of gingipains action as virulence factors: from cleaving substrates with the precision of a surgeon's knife to a meat chopper-like brutal degradation of proteins. *Periodontol* 2010; 54: 15-44.
 84. Martins MS, Jiao Y, Larsson L, et al. Epigenetic modifications of histones in periodontal disease. *J Dent Res* 2016; 95(2): 215-222.
 85. Kang MK, Mehrazarin S, Park NH, Wang CY. Epigenetic gene regulation by histone demethylases: emerging role in oncogenesis and inflammation. *Oral Dis* 2017; 23(6): 709-720.
 86. Paganini-Hill A, White SC, Atchison KA. Dentition, dental health habits, and dementia: the Leisure World Cohort Study. *J Am Geriatr Soc* 2012; 60: 1556-1563.
 87. Rozas NS, Sadowsky JM, Jeter CB. Strategies to improve dental health in elderly patients with cognitive impairment. A systematic review. *J Am Dent Assoc* 2017; 148(4): 236-245.
 88. Ellefsen B, Holm-Pedersen P, Morse DE, et al. Assessing caries increments in elderly patients

- with and without dementia. A one-year follow-up study. *J Am Dent Assoc* 2009; 140: 1392-1400.
89. Kato T, Yamazaki K, Nakajima M, et al. Oral administration of *Porphyromonas gingivalis* alters the gut microbiome and serum metabolome. *mSphere* 2018; 3(5). pii: e00460-18. doi: 10.1128/mSphere.00460-18.
 90. Harding A, Robinson S, Crean S, Singhrao SK. Can better management of periodontal disease delay the onset and progression of Alzheimer's disease? *J Alzheim Dis* 2017; 58: 337-348.
 91. Hajishengallis G, Darveau RP, Curtis MA. "The keystone pathogen hypothesis" *Nat Rev Microbiol* 2012; 10: 717-725.
 92. Delwel S, Binnekade TT, Perez RSGM, Hertogh CMPM, Scherder EJA, Lobbezzoo F. Oral hygiene and oral health in older people with dementia: a comprehensive review with focus on oral soft tissues. *Clin Oral Invest* 2018; 22: 93-108.
 93. Hajishengallis G, Lamont RJ. Beyond the red complex and into more complexity: the polymicrobial synergy and dysbiosis (PSD) model of periodontal disease etiology. *Mol Oral Microbiol* 2012; 27: 409-419.
 94. Zenthöfer A, Meyer-Kühling, Hufeland A-L, et al. Carers' education improves oral health of older people suffering from dementia – results of an interventional study. *Clin Intervent in Aging* 2016; 11: 1755-1762.
 95. Gonsalves WC, Wrightson AS, Henry RG. Common oral conditions in older persons. *Am Fam Physician* 2008; 78: 845-852.
 96. Petersen PE, Yamamoto T. Improving the oral health of older people: the approach of the WHO Global Oral Health Programme. *Community Dent Oral Epidemiol* 2005; 33: 81-92.
 97. Petersen PE. The World Oral Health Report 2003: continuous improvement of oral health in the 21st century – the approach of the WHO Global Oral Health Programme. *Community Dent Oral Epidemiol* 2003; 31: 3-24.
 98. Thomson WM. Dental caries experience in older people over time: what can the large cohort studies tell us? *Br Dent J* 2004; 196: 89-92.
 99. Boehm TK, Scannapieco FA. The epidemiology, consequences, and management of periodontal disease in older adults. *J Am Dent Assoc* 2007; 138(Supl) 26S-33S.
 100. Affoo RH, Foley N, Garrick R et al. Meta-analysis of salivary flow rates in young and older adults. *J Am Geriatr Soc* 2015; 63: 2142-2151.
 101. Morely JE. Dysphagia and aspiration. *J Am Med Assoc* 2015; 16: 631-634.
 102. Ortega O, Parra C, Zarcero S, Nart J, Sakwinska O, Clave P. Oral health in older patients with oropharyngeal dysphagia. *Age and Ageing* 2014; 43: 132-137.
 103. Eisenstadt ES. Dysphagia and aspiration pneumonia in older adults. *J Amer Acad Nurs Practit* 2010; 22: 17-22.
 104. Maeda K, Akagi J. Oral care may reduce pneumonia in the tube-fed elderly: a preliminary study. *Dysphagia* 2014; 29: 616-621.
 105. Booker S, Murff S, Kitko L, Jablonski R. Mouth care to reduce ventilator-associated pneumonia. *Am J Nursing* 2013; 113: 24-30.
 106. Jones AL, Dwyer LL, Bercovitz AR, Strahan GW. The National Nursing Home Survey: 2004 Overview. National Center for Health Statistics, Vital Health Statistics, 2009.
 107. Mahoney EK, Hurley AC, Volicer L, et al. Development and testing of the Resistiveness to Care Scale. *Res Nurs Health* 1999; 22: 27-38.
 108. Kambhu PP, Levy SM. Oral hygiene care levels in Iowa intermediate care facilities. *Spec Care Dentist* 1993; 13: 209-214
 109. Adams R. Qualified nurses lack adequate knowledge related to oral health, resulting in inadequate oral care of patients on medical wards. *J Adv Nurs* 1996; 24: 552-560
 110. Pule MA, Jasinevicius TR, Sawyer DR, Madsen J. Nursing home executive directors' perception of oral care in long-term care facilities. *Spec Care Dentist* 2005; 25: 111-117.
 111. Algase DL, Beck C, Kolanowski A, Whall A, Berent S, Richards K, Beattie E. Need-driven

- dementia compromised behavior: An alternative view of disruptive behavior. *Am J Alzheimers Dis Other Demen* 1996; 11: 10-19.
112. Kolanowski AM, Whall AL. Toward holistic theory-based intervention for dementia behavior. *Holist Nurs Pract* 2000; 14: 67-76.
 113. Whall A. Developing needed interventions from the need-driven dementia-compromised behavior model. *J Gerontol Nurs* 2002; 28: 5.
 114. Frenkel HF. Behind the scenes: care staff observations on the delivery of oral health care in nursing homes. *Gerodontology* 1999; 16: 75-80.
 115. Chalmers J Pearson A. Oral hygiene care for residents with dementia: a literature review. *J Adv Nurs* 2005; 52: 410-419.
 116. American Dental Association. Oral Health Topics: Dentures. Accessed November 22, 2022.
 117. American Dental Association. MouthHealthy. Healthy Teeth and Gums Adults over 60. Accessed November 22, 2022.
 118. Mahoney EK, Hurley AC, Volicer L. Instruction manual for the resistiveness to care scale (RTC-DAT). Boston, MA: Boston College School of Nursing; 1999.
 119. Jablonski RA, Therrien B, Mahoney EK, Kolanowski A, Gabello M, Brock A. An intervention to reduce care-resistant behavior in persons with dementia during oral hygiene: a pilot study. *Spec Care Dentist* 2011; 31: 77-87.
 120. Chalmers JM. Behavior management and communication strategies for dental professionals when caring for patients with dementia. *Spec Care Dentist* 2000; 20: 147-154.
 121. Lopez R, Smith PC, Gostenmeyer G, Schwendicke F. Ageing, dental caries, and periodontal disease. *J Clin Periodontol* 2017; 44(Suppl 18): S145-S152.
 122. Kassenbaum NJ, Bernabe E, Dahiya M, et al. Global burden of untreated caries: a systematic review and metaregression. *J Dent Res* 2015; 94: 650-658.
 123. Mei ML, Chin-Man Lo E, Chu CH. Clinical use of silver diamine fluoride in dental treatment. *Compend Contin Educ Dent* 2016; 37: 93-98.
 124. Horst JA, Ellenikiotis H, Milgrom PL. USCF protocol for caries arrest using silver diamine fluoride: rationale, indications, and consent. *J Calif Dent Assoc* 2016; 44: 16-28.
 125. Zhao IS, Goa SS, Hiraishi N, et al. Mechanisms of silver diamine fluoride on arresting caries: a literature review. *Int Dent J* 2018; 68: 67-76.
 126. Oliveira BH, Cunha-Cruz J, Rajendra A, Niederman R. Controlling caries in exposed root surfaces with silver diamine fluoride. *J Am Dent Assoc* 2018; 149: 671-679.
 127. Fiske J, Frenkel H, Griffiths J et al. Oral health of people with dementia. *Gerodontology* 2016; 23:3-32.
 128. Hoben M, Kent A, Kobagi N, Huynh KT, Clarke A, Yoon MN. Effective strategies to motivate nursing home residents in oral care and to prevent or reduce responsive behaviors to oral care: A systematic review. *PloS one*. 2017 Jun 13;12(6):e0178913.
 129. Chalmers J, Pearson A. Oral hygiene care for residents with dementia: a literature review. *Journal of Advanced Nursing*. 2005 Nov;52(4):410-9.

Additional Resources

- No Additional Resources Available

About the Authors

Maria L. Geisinger, DDS, MS



Mia L. Geisinger, DDS, MS is a Professor and Director of Advanced Education in Periodontology in the Department of Periodontology in the University of Alabama at Birmingham (UAB) School of Dentistry. Dr. Geisinger received her BS in Biology from Duke University, her DDS from Columbia University School of Dental Medicine, and her MS and Certificate in Periodontology and Implantology from the University of Texas Health Science Center at San Antonio. Dr. Geisinger is a Diplomate in the American Board of Periodontology and a Fellow in the International Team for Implantology. She has served as the President of the American Academy of Periodontology Foundation, as the Chair of the American Dental Association's Council on Scientific Affairs, and on multiple national and regional organized dentistry committees. She currently serves as the Vice President of the AAP, as a Board member for the ADA Science and Research Institute, and on numerous AAP and ADA committees and task forces. She has authored over 60 peer-reviewed publications and her research interests include periodontal and systemic disease interaction, implant dentistry in the periodontally compromised dentition, and novel treatment strategies for oral soft and hard tissue regeneration. She lectures nationally and internationally on topics in periodontology and oral healthcare.

Email: miagdds@uab.edu

Maggie A. Misch, BS



Dr. Maggie Misch-Haring is a practicing periodontist at Misch Implant Dentistry in Sarasota, Florida. She received her Doctor of Dental Medicine degree from the University of Alabama at Birmingham in 2019, graduating Omicron Kappa Upsilon and valedictorian of her class. Dr. Misch-Haring went on to a three-year specialty residency in Periodontics at the University of Alabama at Birmingham. She earned a Masters degree in Dentistry with a focus on growth factors used to enhance wound healing for treating advanced bone defects. Dr. Misch-Haring has received a number of awards including the American Academy of Periodontology Award for Achievement in Periodontics and the Academy of Osseointegration Outstanding Student in Implant Dentistry Award.

Email: mmisch@uab.edu