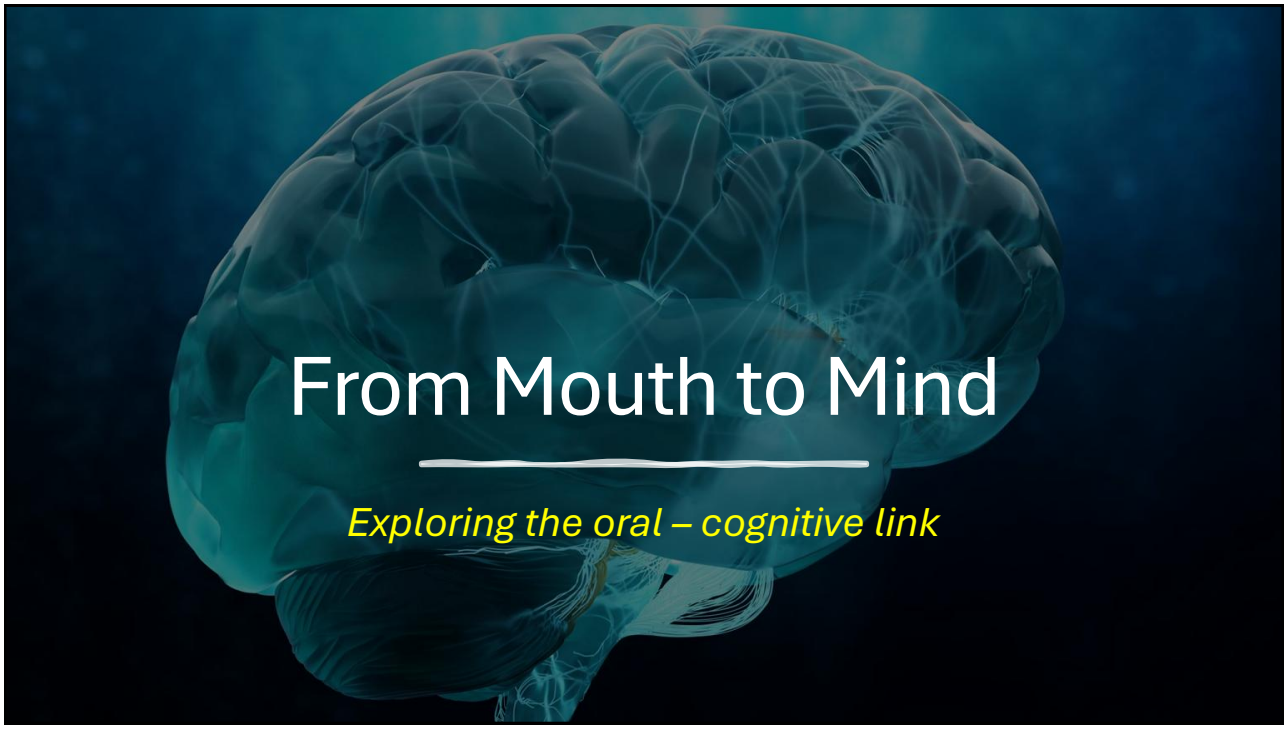
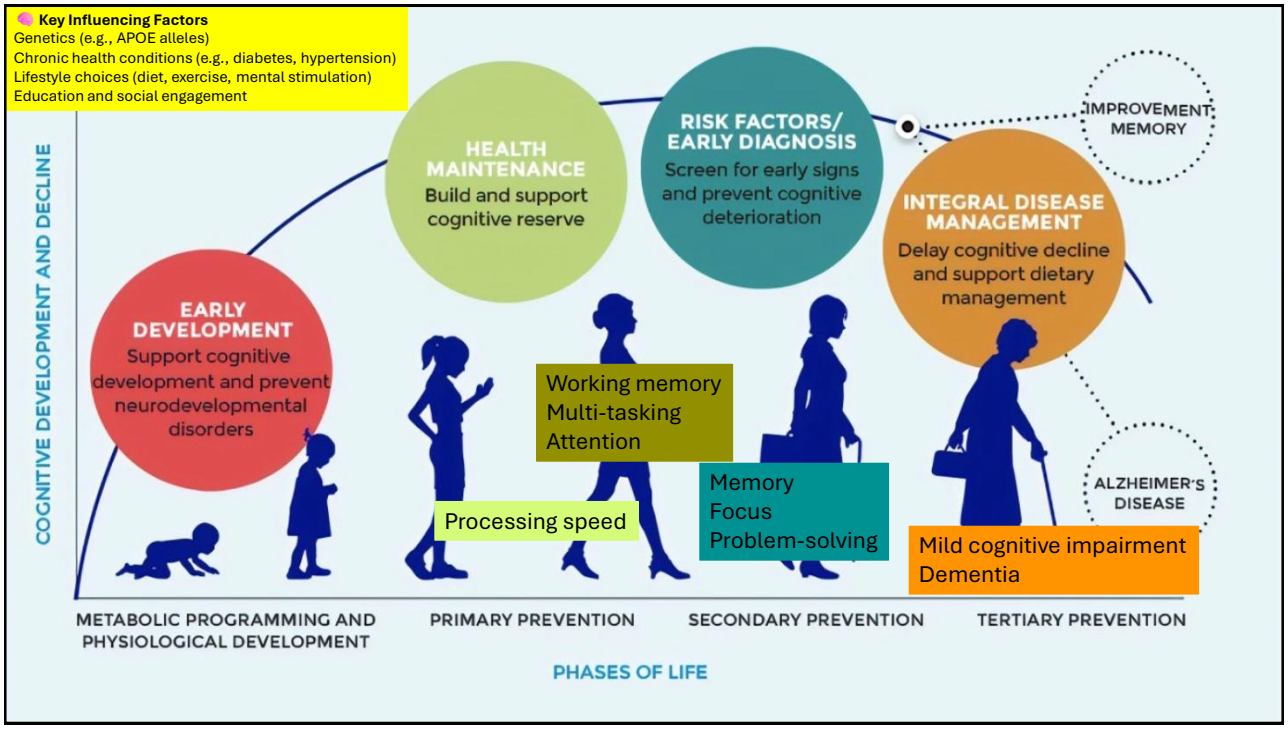

From Mouth to Mind
Exploring the oral – cognitive link

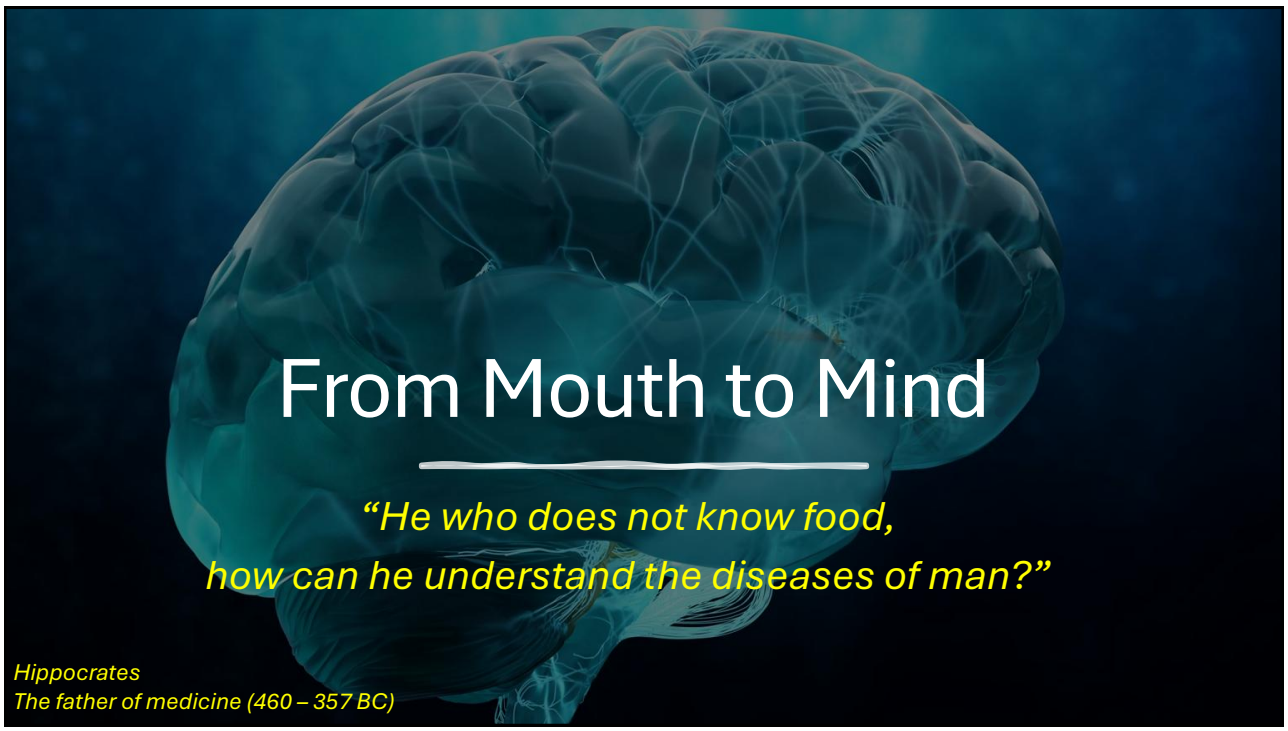

🧠 Key Influencing Factors
Genetics (e.g., APOE alleles)
Chronic health conditions (e.g., diabetes, hypertension)
Lifestyle choices (diet, exercise, mental stimulation)
Education and social engagement
COGNITIVE DEVELOPMENT AND DECLINE
EARLY DEVELOPMENT
Support cognitive development and prevent neurodevelopmental disorders
HEALTH MAINTENANCE
Build and support cognitive reserve
RISK FACTORS/ EARLY DIAGNOSIS
Screen for early signs and prevent cognitive deterioration
INTEGRAL DISEASE MANAGEMENT
Delay cognitive decline and support dietary management
IMPROVEMENT MEMORY
ALZHEIMER'S DISEASE
Working memory
Multi-tasking
Attention
Processing speed
Memory
Focus
Problem-solving
Mild cognitive impairment
Dementia
METABOLIC PROGRAMMING AND PHYSIOLOGICAL DEVELOPMENT
PRIMARY PREVENTION
SECONDARY PREVENTION
TERTIARY PREVENTION
PHASES OF LIFE

My aim for this presention
By the end of this presentation, participants will be able to:

1. ***Recognize the oral-cognitive link*** and its implications for aging and neurological health.

2. ***Understand the role of the oral microbiome*** in brain function, including:
• How beneficial bacteria like *Neisseria* and *Haemophilus* are linked to better cognitive performance.
• How harmful bacteria such as *Porphyromonas* and *Prevotella* may contribute to memory decline and Alzheimer's risk
• The oral-gut-brain axis and its role in neuroinflammation

3. ***Apply this knowledge in clinical practice*** *by:*
• Integrating oral microbiome awareness into patient assessments.
• Promote preventive oral care practices by
  • Promoting nitrate-rich diets (e.g., leafy greens, beetroot juice ☺) to support beneficial oral bacteria
  • Encouraging consistent oral hygiene to reduce both systemic and neuroinflammatory risks



Longitudinal Health Studies

The Blue Zones

The China Study

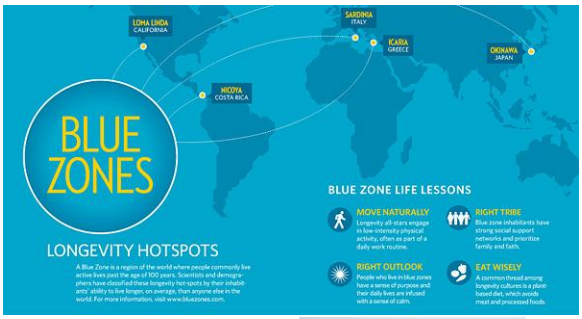
The Adventists Health Studies

Where people forgot to die!

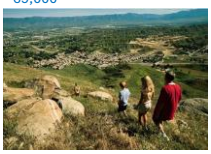
SARDINIA: WINE, GOAT'S MILK, MASTIC OIL
Shed stress, strong will, high self-esteem, great stubbornness, survive in unfavorable circumstances, walks many miles per day

ICARIA: HONEY, SIESTA, HERBAL TEAS (antioxidants and diuretic function), WILD GREENS, GOAT'S MILK, POTATOES
Lower rates of cardiovascular disease, depression and dementia (only 20% of people over 80 – US 50%)
Read about Stamatis!!

LOMA LINDA: NUTS, VEGETARIAN DIET, SABBATH DAY, EXERCISE
Seventh day adventists have a much lower rate of cancers, heart disease and diabetes compared to rest of US.
Ellen G. White started the Adventists Preventive Medicine Movement
The Adventists health study by Dr. Fraser: Life styles and Health outcomes of over 34,000 people over 12 years.
New study AD-2, over 69,000



OKINAWA: SWEET POTATO, TURMERIC, TOFU
5 per 10,000 inhabitants; Healthy centenarians living independent lives; lower levels of cancer (colon and breast) and cardiovascular disease, small plates. Preventive medicine has a higher rank than Medical treatments



NICOYA: BEANS, RICE, TORTILLA, FRUITS, "HARD" Calcium rich water; Use lime (CaOH) to cook the corn; nixtamalization. Lower rates of stomach cancer

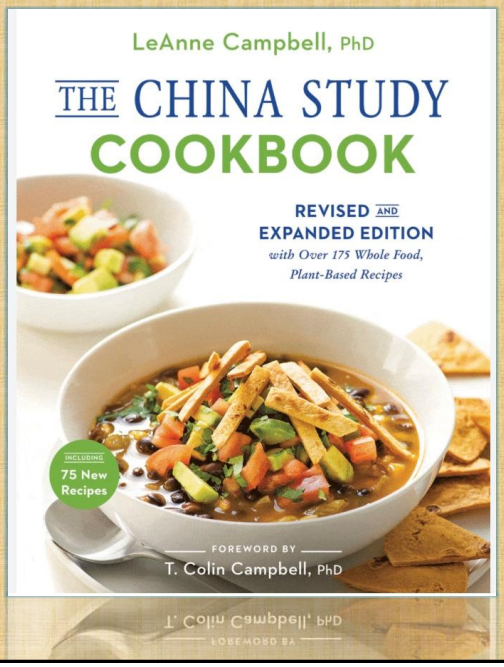
Image: Levine & associates (blog.levinedc.com/2009/...)

The China Study

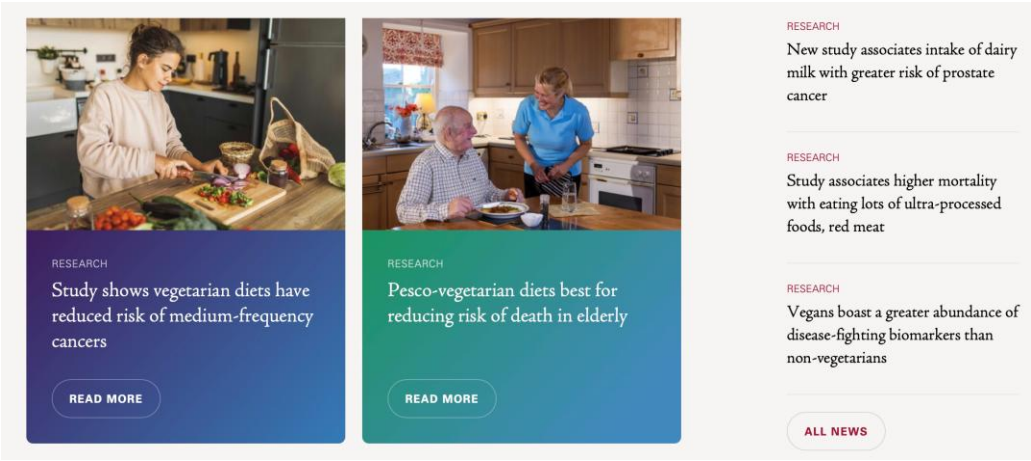
- ***Diet and chronic disease***
  - The study found a strong link between eating a lot of animal-based foods and higher rates of chronic illnesses, while a plant-based diet was associated with better health.
- ***Animal protein***
  - Even small amounts of animal protein were linked to adverse health effects, and researchers concluded that cutting down on animal protein is one of the most effective ways to lower blood cholesterol and reduce disease risk.
- ***Plant-based advantages***
  - The more plant-based foods people ate, the healthier they tended to be, and the lower their risk of chronic diseases.

The China Study – the China-Cornell-Oxford Project

- ***Dietary recommendations***
  - Based on the findings, the study recommends a diet based on whole, unrefined plant foods like fruits, vegetables, tubers, beans, and whole grains, with limited nuts and no meat, dairy, eggs, or processed foods.



Adventist's Health Studies (1 and 2)



Mediterranean diet - DASH & MIND

- *Mediterranean diet*
  - One of the healthiest eating patterns to reduce inflammation, promote longevity and prevent chronic disease

  - *DASH* (**Di**etary **A**pproaches to **S**top **H**ypertension)
    - Vegetables, fruits, whole grains, fat-free or low-fat dairy, fish, poultry, beans, nuts, seeds and vegetable oils. Limit or avoid food high in sodium, saturated fats, and sugar. This means fatty meats, full-fat dairy products and sugary drinks and sweets.

  - *MIND (***M**editerranean-DASH Diet **I**ntervention for **N**eurodegenerative **D**elay)
    - A hybrid of the Mediterranean and DASH diets designed to prevent cognitive decline and reduce the risk of Alzheimer's disease. Following this diet is linked to a lower risk of dementia and slower cognitive decline over time.

The MIND diet

- *COGNITIVE DECLINE (2024)*
  4% lower risk of cognitive decline
  women and African/American

- *DEMENTIA (2025)*
  9% lower risk of developing dementia
- Following the diet over 10 yrs had a 25% lower risk of dementia

- *ALZHEIMER'S DISEASE (2016)*
  Strictly following the MIND diet had up to a 53% reduced risk of developing AD

Morris et al. (2016); Sawyer et al. (2024); Park et al. (2025);

Why oral health matters to medicine

Oral bacteria ≠ harmless commensal bacteria

Our diet directly influences the oral biofilm and the oral microbiome

Oral Health among Centenarians

- Oral health of centenarians and supercentenarians (100-135 yoa) and their offsprings was better than that of their birth cohort controls
- *New England Centenarian study*
  - Oral microbiome
    - Age only had a marginal impact on the oral microbiome. Centenarians and their offspring consistently show better oral health, with more natural teeth, compared to people born in the same era.
    - *Centenarians had a more diverse oral microbiome than expected, which included a higher relative abundance of certain periodontopathogens and lower levels of cariogenic bacteria.*
  - Skin, gut
    - Significant variations were observed in microbiome from other bodily regions where centenarians displayed a separate cluster from the young and the (normal) elderly

Kaufman et al. (2014)

The Aging US population

• There are about ***35 million*** edentulous people in the US, and ***178 million*** people are missing at least one tooth

• Roughly 90% of the US edentulous population wears dentures

• ***51%*** of Americans, ages 55 to 64, wear full or partial dentures

• ***29%*** of Americans, ages 45 to 55, wear full or partial dentures

• ***16%*** of Americans, ages 35 to 44, wear full or partial dentures

• The number of partially edentulous people will continue to increase in the next 15 years to more than 200 million individuals. *Partial edentulism affects the majority of adult Americans.*

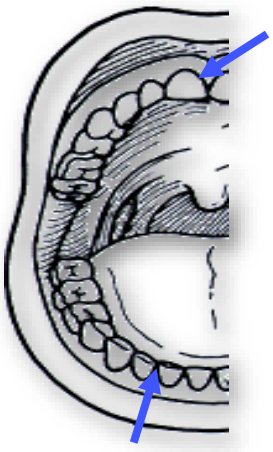
• *in 2030, 21% of the US population will be 65+ → 72 million*

If you compare these figures with the year of 2000, the number of 65+ individuals have doubled!!

Source: American College of Prosthodontics




Oral health is a lifelong concern and has implications for systemic and cognitive health, especially in aging populations



Caries

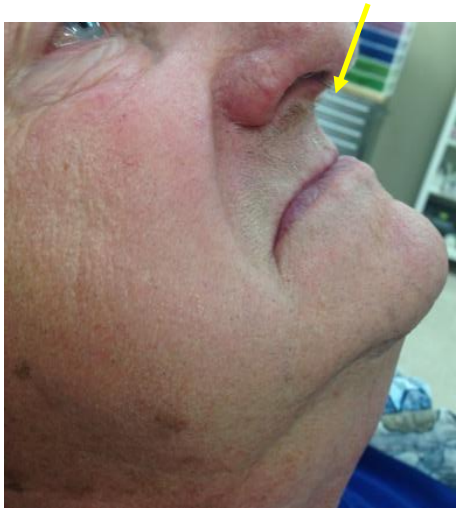
- The caries process develops during the first months following eruption, and we have three peaks:
  - 6 years
  - 25 years
  - *70 years*

- Ion exchanges between teeth and the oral cavity
  - Inorganic compounds: Ca, Mg and other ions from oral fluids
  - Organic compounds enter the enamel through micro-cracks, enamel tufts and lamellae
  - Ca/P ratio in hydroxyapatite is reduced (1.67 → 1.33)
  - *With F substitution the ratio can be increased to 2*

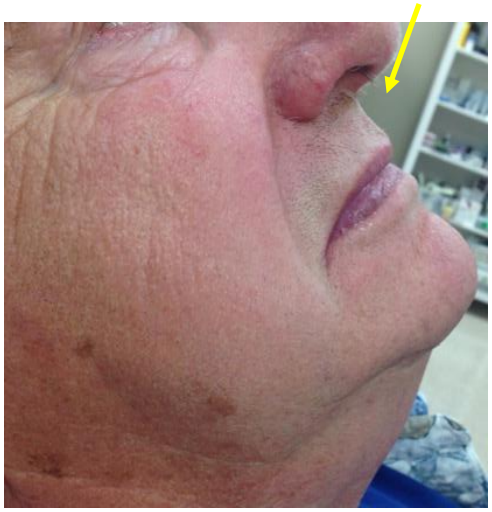
- *Most soluble enamel is at the gingival margin, and the proximal contact surfaces of the teeth*

Kunin et al., 2015

Loss of bone mass

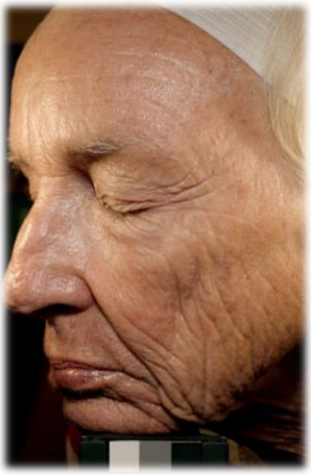


Full mouth extractions in UA



With newly inserted CD in UA

Loss of connective tissue "volume"

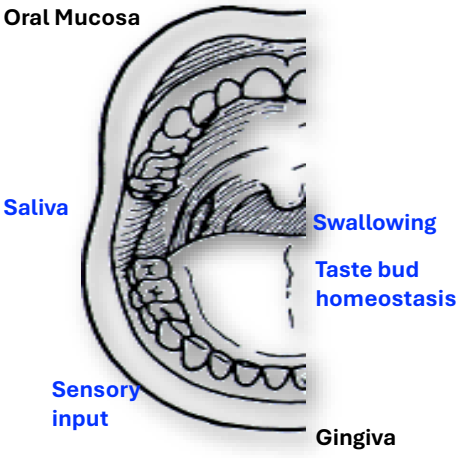


Thinning of collagen and elastic fibers

- Fat atrophy and thinning of lips

Beer and Beer, 2009

Loss of connective tissue "volume"



Oral mucosa
Atrophic, satin-like and friable
Thin, smooth and dry

Gingiva
Looses its stippling due to flattening of rete pegs
Clinically: Loss of attachment – *gingiva migrates to tooth apex; reduced resilience*

Epithelium
- Diminished keratinization, thinner
- Parakeratosis is frequent
- *Chronic inflammation*

Connective tissue
- Fewer connective tissue cells (fibroblasts)
- Slower turn-over of fibers
- Less elastic fibers – tissue loss of elasticity
- Thicker collagen bundles = Fibrosis
- *Capillaries* decrease in numbers

Andreescu et al., 2013

Aging of the Immune System

Adaptive

- Decrease in mature, naïve TLs
- Age dependent decreases in natural ab titers and increase in auto-ab titers
  - *Example: Defective responses to Candida infections in the 65+ group*
  - *3x as many women as men display this lack of response*

Innate

- *Macrophages*
  - Reduced chemotaxis
  - Reduced antigen presentation (due to reduced expression of MHC class II)
  - Reduced cytokine and chemokine secretion
  - Reduced wound repair
- Dendritic cells
  - Reduced chemotaxis
- *Neutrophils*
  - Reduced phagocytic capacity
  - Reduced intracellular killing efficiency
  - Reduced "infiltration" at sites of injury

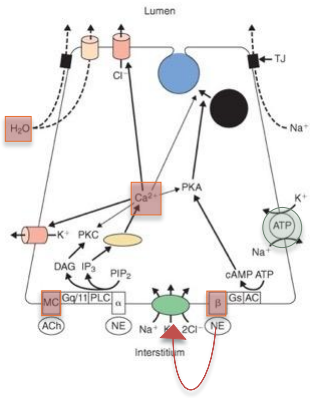
Gomez et al. (2012)

Clinical consequences of Immunological senescence

- *Reduced wound healing*
  - Extractions
  - Surgery
- Increasing rate of
  - *Infectious disease* (ex. Initiation of Respiratory disease; RSV vaccine in 2023)
  - *Autoimmune disease*
    - Females have a higher incidence
  - *Cancer* incidence
  - *Tissue graft tolerance*
- *Gingival immune defense is diminished*
  - *More rapid and severe development of gingivitis*
  - *Healing process is slower*
- *Ineffective protection against microbes at the muco-cutaneous junction*

Polypharmacy - Medications which affect salivary flow

- Anti-cholinergic medication
- Beta-blockers
- Ca^{2+} antagonists
- Diuretics



Summary of Aging changes in the Oral Cavity

- ***Enamel*** is stable, but prone to demineralization at the gingival margin and proximal surfaces. Dentin is only 60% mineral → caries processes develop 3x faster
- Biological changes for women at menopause are drastic. Loss of 25% of *bone mass* (750g/3000g), men loose less (450g/4000g). This loss of bone mass is esp. evident in the mandible. Loss of vertical dimension.
- *Gingiva, oral mucosa and PDL*: Decreased barrier function, more prone to gingivitis, slower collagen turn-over, chronic inflammation, slower healing following surgery and extractions
- Loss of *sensory input* from nerve endings and mechanoreceptors
- *Salivary glands*: Reduced flow rate with aging, decreased levels of mucins, histatins; increased oral dryness which is accompanied by decreased levels of antimicrobials
- Secondary oral aging as a result of *medications*: Anti-cholinergic, beta-blockers, Ca2+ antagonists, diuretics negatively affect *saliva secretion*

Overall biological changes

- Flattening of the *circadian rhythm* reduces protein synthesis in oral mucosa and salivary glands
- *Menopause* affects oral mucosa and salivary glands to a yet not fully known extent.
  - Women no longer have active estrogen circulating in plasma. Instead, they are dependent on local conversion of DHEA into bioactive estrogens and androgens for tissue metabolism.
- Loss of *stem cell* activity: In salivary glands a 20-40% decrease in number of acinar cells (serous and mucous) with aging.

Root Caries in Elderly

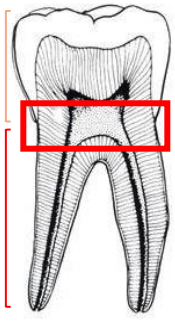
> 50% of older adult over 65 y/75 y have root caries

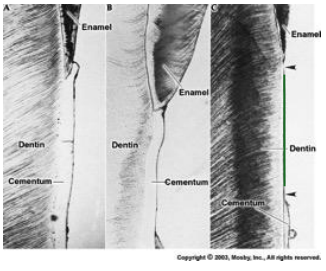
> Patients who have lived in fluoridated areas throughout most of their lives have a lower prevalence of root caries

> Demineralization

> Critical pH Enamel: pH 5.2 – 5.5

> Critical pH Cementum: pH 6.1 - 6.7



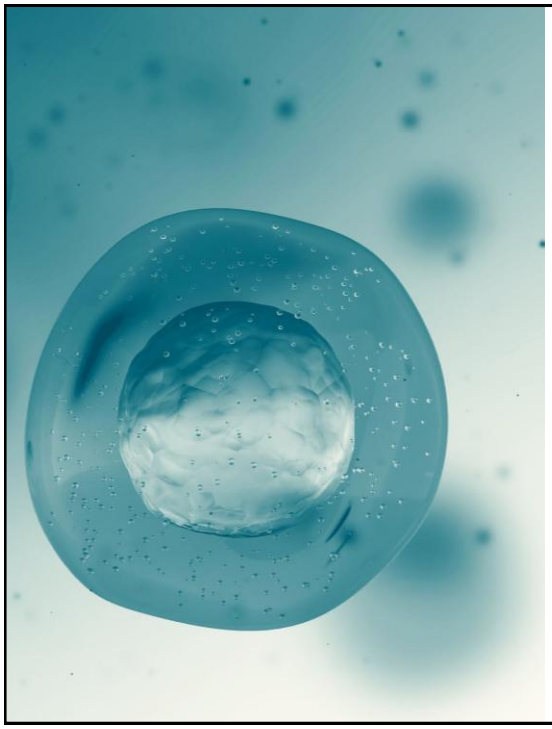


TenCate:
Afibrillar Acellular Cementum

Sources: Center for Disease Control and Prevention, 1992; Ten Cate: Oral Histology

So, what about the Oral Microbiome?

How does the Oral Microbiome influence systemic inflammation and chronic disease?

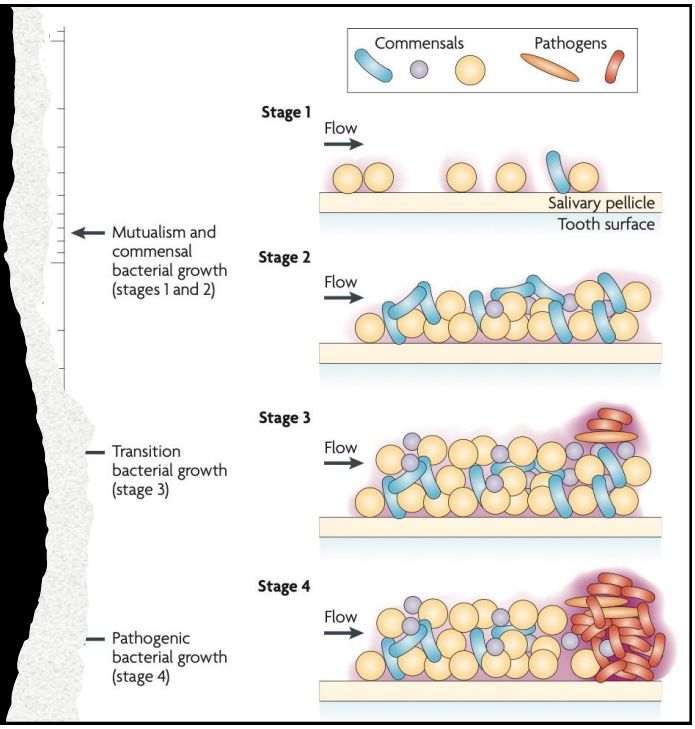


Oral bacteria ≠ harmless commensals

- Oral biofilm contain highly virulent, immune-evasive organisms
- Some bacteria produce proteases, endotoxins, amyloid-like proteins

- *P. gingivalis* (Porphyromonas)
- *S. mutans cnm* (Streptococcus)

Oral biofilm



Borsa et al. (2021)

Biofilm in the Oral Cavity

The oral microbiome is composed of ca *770 different microorganisms*, which form a biofilm on the surface of the teeth and the oral mucosa.
It is *dynamic* and the composition depends on the host environment; ie. our *salivary flow, diet and oral hygiene habits* ☺

Healthy conditions

• Gram-positive bacteria dominate the composition of the biofilm

Oral dysbiosis

• Dietary challenges – *sugars*, *acids*, *lack of tooth brushing* -→ the plaque is evolving to having a greater proportion of *Gram-negative anaerobic bacteria*.

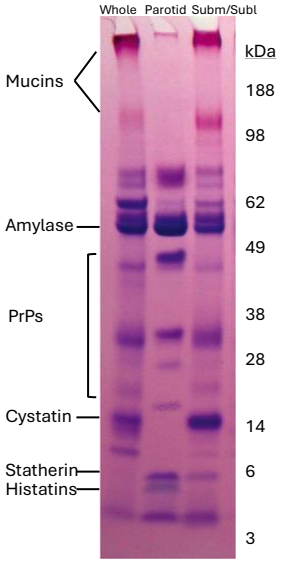
The inflammation in the oral cavity starts as *gingivitis* or *incipient caries* lesion.

The aggressiveness of the biofilm depends on the flow, pH and buffering capacity of the saliva

- The protein and fluid secretion occurs in response to stimulation from receptors in the oral cavity, mainly α/β adrenergic receptors and the cholinergic receptor.
- *The content of our saliva secretion responds to our diet*
  - Submandibular glands secrete a saliva rich in *anti-microbial peptides,* providing a broad-spectrum defense against pathogens in the oral cavity.

Luoro et al. (2021)

Contents of saliva



- *99.5% water and 0.5% dissolved substances:*

Salivary proteins and glycoproteins

- Amylase, Cystatins, *Mucins*, Proline-rich proteins (PrPs), Lysozyme, sIg's, Lactoferrin, Peroxidase, *Histatins*, Statherin, Defensins etc.
  - *Growth factors:* NGF, EGF, and other regulatory peptides
  - ***Antimicrobials***: *Lysozyme, Lactoferrin, Lactoperoxidase, Histatins, Defensins*
  - *Digestion of starch and lipids:* Amylase and Lipase

Inorganic ions

- Bicarbonate, potassium, *calcium and phosphate ions*

Gases

- Oxygen, carbon dioxide and nitrogen

Carpenter (2013)

Tooth loss

Due to inflammation

- As a result of *Caries* and *Periodontitis*
- Result of *Injury*, *Cancer, Polypharmacy* or simply *Wear*
- *Loss of teeth due to lack of care*
- *Edentulism affects our most vulnerable populations – the aging and the economically disadvantaged*
- *Consequences of missing teeth include significant nutritional challenges, obesity, diabetes, coronary artery disease, and some forms of cancer.*

Inflammatory diseases of the Oral Cavity

Both Caries and Periodontal disease are bacterial diseases

Caries

- *S mutans*
- 10-20% of the population has a collagen binding *S mutans cnm*

Periodontal disease

- 1- 3 (4)mm pockets
  - Aerobic bacteria
- 4+ mm pockets
  - Anaerobic bacteria

Inflammatory diseases of the oral cavity

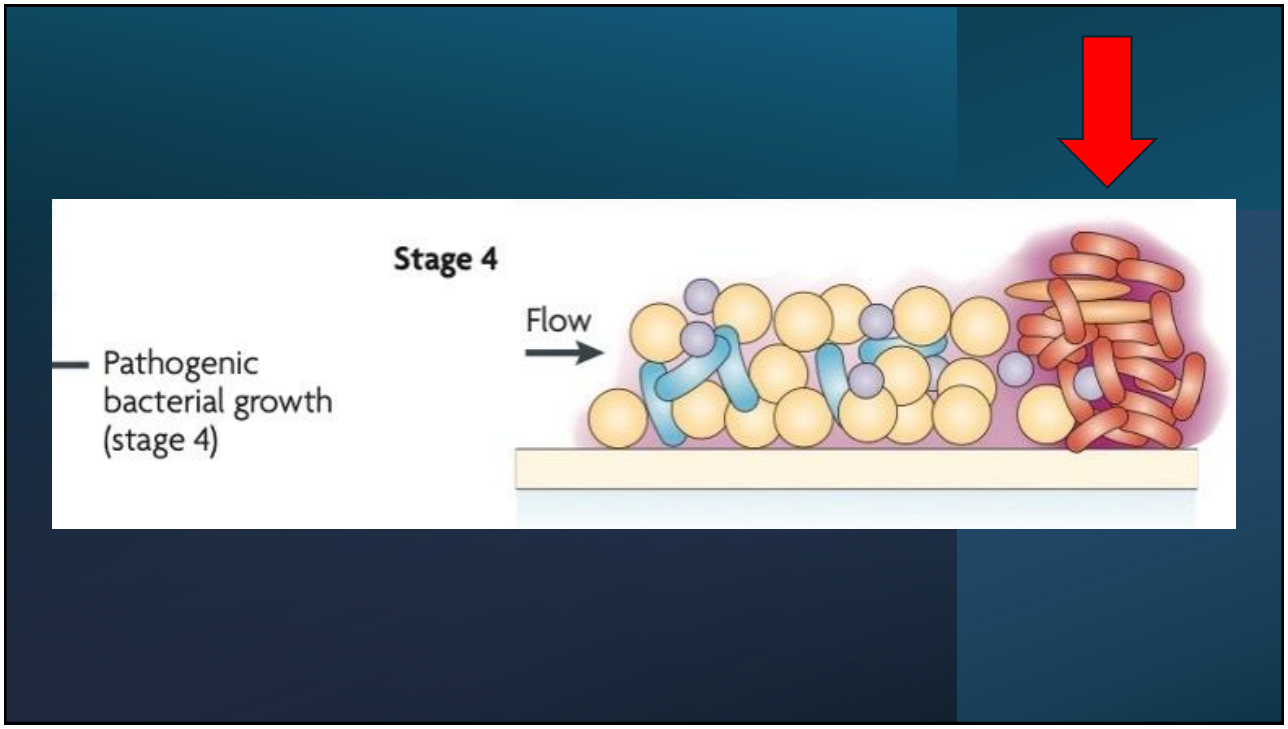
Both Caries and Periodontal disease are bacterial diseases

Caries

- *S mutans*
- 10-20% of the population has a collagen binding *S mutans cnm*

Periodontal disease

- 1- 3 (4)mm pockets
  - Aerobic bacteria
- 4+ mm pockets
  - *Anaerobic bacteria*



Caries

Caries active bacteria (S mutans cnm)
→
Cerebral microbleeds

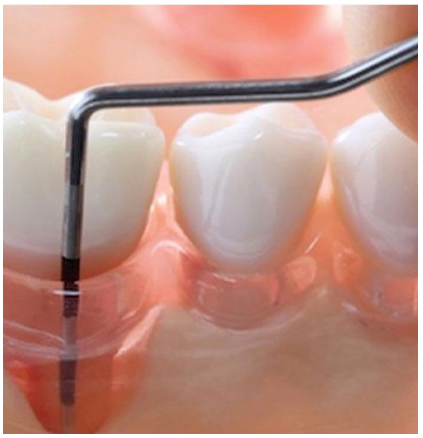
- Influences on overall health?
  - Infective endocarditis
  - Atherosclerosis
  - *Cerebral hemorrhage (CMB)*
  - IgA Neuropathy
  - Diabetes – A1c
  - Nonalcoholic steatohepatitis (NASH)

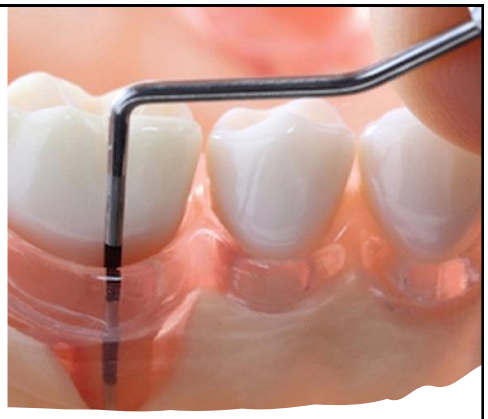
Periodontitis

Periodontal bacteria → Cognitive decline

- Diet
- Oral hygiene – Remaining soft plaque and calculus

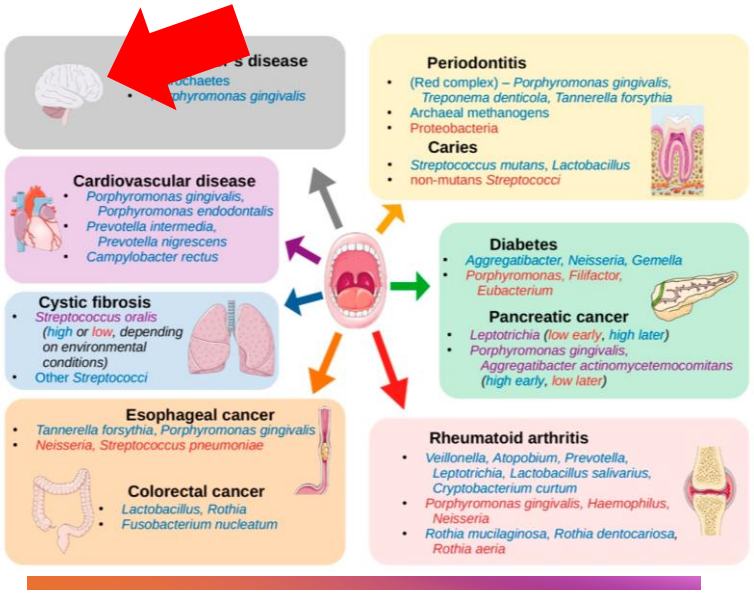
- Gingivitis
- Pockets down to 4mm – aerobic bacterial flora
- Pockets 4 – 12 mm can harbor between 10^7 to 10^9 bacteria/pocket
- *Pockets deeper than 4mm+ - anaerobic bacterial flora*
  - *In terms of systemic health → here lies the culprit to oral inflammation influencing systemic health*
  - Heart disease
  - Joints
  - *Brain health → inducing cognitive decline*





How do we currently prevent and treat Caries and Periodontitis?

... and could we do more or different?



Common participants in the oral dysbiotic biofilm which also influence systemic dysbiosis

Borsa et al. (2021); Willis and Gabaldon (2020)

Caries and Cerebral microbleeds

- *S. mutans*
  - Certain strains of *S mutans* express a collagen-binding protein called ***Cnm***
    - *Cnm* is present on the bacterial surface and helps the bacteria invade the lining of blood vessels by binding to collagen IV
  - Approx. 10 – 20% of us have *S mutans* with the *Cnm* gene (Finland and Japan)
  - *Cnm* - positive strains have been linked to
    - *BBB disruption:* These bacteria can bind to collagen IV in damaged blood vessels and also have the potential to invade endothelial cells → Injury to the BBB and neuroinflammation
    - *Cerebral microbleeds (CMBs):* Small hemorrhages in the brain associated with vascular cognitive impairment and increased stroke risk (Watanabe et al. 2016)
    - Apart from cognitive decline, this subgroup of *S mutans* has also been linked to *infective endocarditis* and *IgA neuropathy.*
      - *Studies that focus on infective endocarditis show a cnm-positive rate of 40%*
    - In children, there is a higher severity of dental caries in children having the cnm-positive *S mutans*

Watanabe et al. (2016) Oral Cnm-positive Strep Mutans expressing collagen binding activity is a risk factor for cerebral microbleeds and cognitive impairment. Nature Dec 9, 2016
Satoshi Hosoki (2020) Oral Carriage of S mutans harboring the cnm gene relates to an increased incidence of cerebral microbleeds. Stroke 1 (12): 3632-3639.

CMB study for correlation with Cnm-binding S mutans (1)

- 279 participants, 70 +/- 6.1 yoa
- Brain MRI exams and cognitive functional examinations (2012 – 2014)
  - 189 males
  - 90 females
- Systemic medical evaluations and dental evaluations of caries and periodontitis. Lab evaluations.

- *Cnm-binding S. mutans* were shown to interfere with the healing of blood vessels and to cause more extensive hemorrhage.

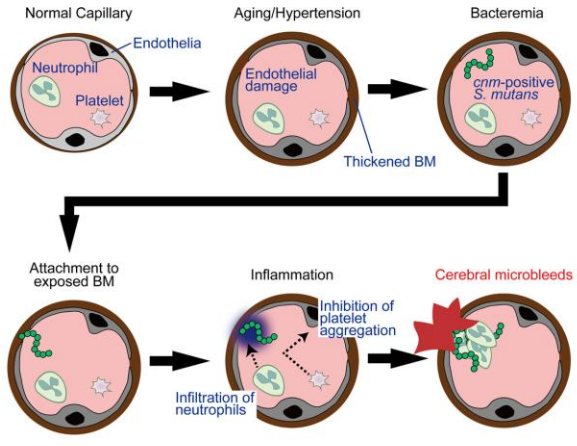
Watanabe et al. (2016) Oral Cnm-positive Strep Mutans expressing collagen binding activity is a risk factor for cerebral microbleeds and cognitive impairment. Nature Dec 9, 2016
Satoshi Hosoki (2020) Oral Carriage of S mutans harboring the cnm gene relates to an increased incidence of cerebral microbleeds. Stroke 1 (12): 3632-3639.

CMB study for correlation with Cnm-binding S mutans (2)

- Retrospective study of stroke patients that received an oral microbiological exam and head 3T magnetic resonance imaging twice between 2014 – 2019
  - 111 subjects with comparable clinical history
    - 21 with *cnm*-positive *S mutans*
    - 90 w/o

- New CMBs were more commonly observed in patients with Cnm-positive *S mutans* (52% vs. 23%; P=0.008)
- The incidence of CMB was significantly higher in the group with cnm-positive *S mutans*, especially in deep areas – incidence ratio (15) vs. in any brain region (5.1)
- *The study demonstrates that cnm-positive S mutans is associated with an increased incidence of CMB.*
- *Treatment of cnm-positive S mutans infection may be a novel microbiome-based therapeutic approach for stroke and cognitive impairment.*

Watanabe et al. (2016) Oral Cnm-positive Strep Mutans expressing collagen binding activity is a risk factor for cerebral microbleeds and cognitive impairment. Nature Dec 9, 2016
Satoshi Hosoki (2020) Oral Carriage of S mutans harboring the cnm gene relates to an increased incidence of cerebral microbleeds. Stroke 1 (12): 3632-3639.



Aging and hypertension results in endothelial damage and thickened basement membranes (BM). Unlike *cnm*-negative *S. mutans*, *cnm*-positive *S. mutans* can attach to the BM. Once *cnm*-positive *S. mutans* binds to the vessel wall, infiltration of neutrophils results in local inflammation. The negative charges on the surface of *cnm*-positive *S. mutans* inhibit the aggregation of platelets, which also possess negative charges on the surfaces. CMB are eventually induced.

Satoshi Hosoki (2020) Oral Carriage of S mutans harboring the cnm gene relates to an increased incidence of cerebral microbleeds. Stroke 1 (12): 3632-3639.

Environmental stressors

S mutans thrives under a range of harsh oral environmental conditions, and this stress can trigger the expression of virulence factors like ***Cnm***.

- ***pH fluctuations***
  The oral cavity experiences significant pH fluctuations due to the fermentation of carbohydrates. In order to survive, *S. mutans* activates stress tolerance mechanisms that may be linked to Cnm expression.
- ***Oxidative stress***
  The oral microbiome is a dynamic environment with varying oxygen levels and exposure to reactive oxygen species (ROS) from other bacteria and hygiene products. *S. mutans* has multiple systems to counter oxidative stress, and these response pathways are linked to the expression of virulence factors.
- ***Quorum sensing***
  S. mutans uses a peptide-based quorum-sensing system to coordinate gene expression based on cell density. The competence-stimulating peptide (CSP) can act as a stress-signaling molecule, inducing antibiotic persistence and activating other stress defense genes that could influence virulence.
- ***Carbohydrate availability***
  The consumption of fermentable carbohydrates like sucrose influences the expression of many virulence-related traits in *S. mutans*, and Cnm is involved in sucrose-independent adhesion mechanisms.

Environmental stressors

S mutans thrives under a range of harsh oral environmental conditions, and this stress can trigger the expression of virulence factors like ***Cnm***.

- ***pH fluctuations***
  The oral cavity experiences significant pH fluctuations due to the fermentation of carbohydrates. In order to survive, *S. mutans* activates stress tolerance mechanisms that may be linked to Cnm expression.
- ***Oxidative stress***
  The oral microbiome is a dynamic environment with varying oxygen levels and exposure to reactive oxygen species (ROS) from other bacteria and hygiene products. *S. mutans* has multiple systems to counter oxidative stress, and these response pathways are linked to the expression of virulence factors.
- ***Quorum sensing***
  S. mutans uses a peptide-based quorum-sensing system to coordinate gene expression based on cell density. The competence-stimulating peptide (CSP) can act as a stress-signaling molecule, inducing antibiotic persistence and activating other stress defense genes that could influence virulence.
- ***Carbohydrate availability***
  The consumption of fermentable carbohydrates like sucrose influences the expression of many virulence-related traits in *S. mutans*, and Cnm is involved in sucrose-independent adhesion mechanisms.

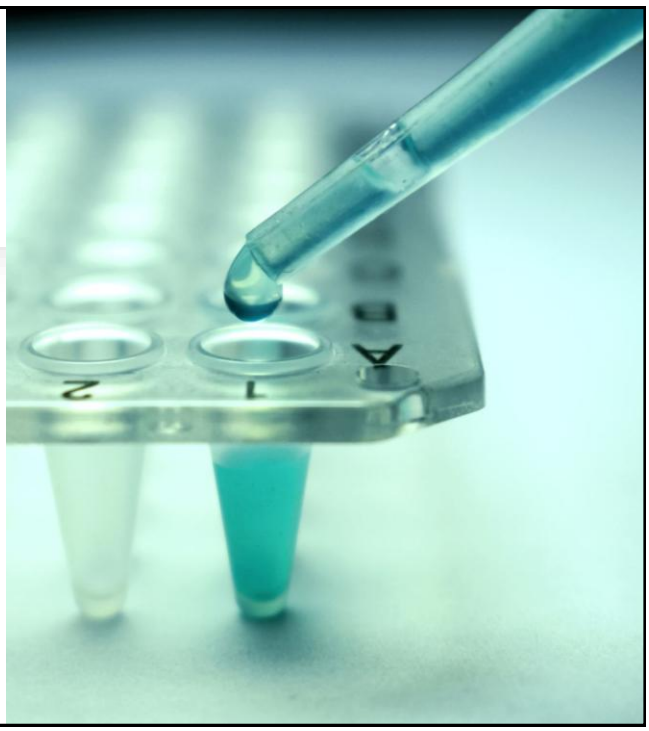
Environmental stressors

S mutans thrives under a range of harsh oral environmental conditions, and this stress can trigger the expression of virulence factors like ***Cnm***.

- ***pH fluctuations***
  The oral cavity experiences significant pH fluctuations due to the fermentation of carbohydrates. In order to survive, *S. mutans* activates stress tolerance mechanisms that may be linked to Cnm expression.
- ***Oxidative stress***
  The oral microbiome is a dynamic environment with varying oxygen levels and exposure to reactive oxygen species (ROS) from other bacteria and hygiene products. *S. mutans* has multiple systems to counter oxidative stress, and these response pathways are linked to the expression of virulence factors.
- ***Quorum sensing***
  S. mutans uses a peptide-based quorum-sensing system to coordinate gene expression based on cell density. The competence-stimulating peptide (CSP) can act as a stress-signaling molecule, inducing antibiotic persistence and activating other stress defense genes that could influence virulence.
- ***Carbohydrate availability***
  The consumption of fermentable carbohydrates like sucrose influences the expression of many virulence-related traits in *S. mutans*, and Cnm is involved in sucrose-independent adhesion mechanisms.

How is S. mutans cnm detected?

- ***Polymerase Chain Reaction (PCR)***
  - Extraction of DNA from saliva or plaque samples
- ***Real-Time PCR (qPCR)***
  - The most sensitive and quantitative method
  - Used to detect and measure the abundance of *S. mutans* and its virulence genes in dental plaque samples.



Periodontitis

- Gingival bleeding and attachment loss as the periodontal disease progresses, is usually *painless* for the individual, and thus fully ignored until probing depths are recorded.
- 50% of the population has *gingivitis* without bone loss and no pockets deeper than 3 mm around 3 or 4 teeth at any given time.
- *30% have periodontitis as defined by the presence of three or more teeth with pockets >= 4mm.*
- *Between 5 – 15% of periodontitis patients have an advanced forms with pockets >= 6mm.*

Loesche and Grossman (2001)

Traditional treatment options for reduction of periodontal pockets

- Scaling and Root Planing (SRP)
  - Disrupts biofilm and allow reattachment of gingival tissue
- Local Antimicrobial Therapy
  - Direct placement into periodontal pockets → Chx chips, minocycline microshpheres
    - Deliver high concentrations of antimicrobials to infected sites w/o systemic exposures
- Systemic Antibiotics (low dose)
  - Doxycycline 20mg BID
    - In cases of aggressive periodontitits
- Surgical interventions
  - Flap surgery – thorough debridement and pocket retention
    - Guided tissue regenerations
- Adjunct Therapies
  - Laser therapy for bacterial reduction
- Lifestyle modifications
  - Smoking cessation, *diet modification*

Periodontitis, SRP, probiotics and diet

Is diet and/or probiotics as an adjunct to SRP able to reduce periodontal pockets?

The most beneficial probiotic bacteria for reduction of periodontal pockets

Bifidobacterium lactis

- **Bifidobacterium lactis** is a Gram-positive anaerobic bacterium and
  - an initial colonizer of the human GI tract.
  - It produces **bacteriocins** (antimicrobial peptides)
- Probiotic treatment with *B. lactis HN019* increases expression of **β-defensin-3**, an antimicrobial peptide
- It inhibits the growth of *Porphyromonas gingivalis*—a major periodontal pathogen—possibly by competing for **vitamin K**, which *P gingivalis* needs for growth.
- Clinical trials show **larger clinical attachment gain** and **lower probing pocket depths (PPD)** in the test group compared to controls at 90 days

Matsubara et al. (2023); Invernici et al. (2018)

Lactobacillus species

- *Lactobacillus* have antibacterial activity via bacteriocins and production of hydrogen peroxide
- *Lactic acid lowers* pH and disrupts the outer membrane of Gram-negative bacteria

Ng et al. (2021);

Reduction of periodontal pocket (PD) and clinical attachment loss (CAL) through probiotics and diet

- Randomized clinical trial
- Probiotics (in capsules)
  - Lactobacillus rhamnosus
  - Bifidobacterium lactis
- 120 subjects
  - *all females w/o chronic disease*
  - SRP (40 subjects)
  - SRP + Probiotics (40 subjects)
  - SRP + Probiotics + Anti-inflammatory diet (40 subjects)

Yilmaz and Görgin (2025)

Reduction of periodontal pocket (PD) and clinical attachment loss (CAL) through probiotics and diet

- Results
  - PD
  - CAL
- Conclusion
  - *Systemic probiotics were found to support periodontal healing. This was enhanced by an anti-inflammatory diet.*

Table 3 PD and CAL measurements of participants at the beginning and at the end of the 6th week

Time	Measurement	Control ($n=40$)	Probiotic ($n=40$)	Diet + Probiotic ($n=40$)	p
Baseline	PD (mm)	5.3 ± 1.0	5.2 ± 0.9	5.3 ± 1.1	0.79
6 Weeks Later	PD (mm)	4.4 ± 0.9	3.6 ± 0.8	3.1 ± 0.7	< 0.001
Baseline	CAL (mm)	5.5 ± 1.1	5.6 ± 1.0	5.6 ± 1.0	0.91
6 Weeks Later	CAL (mm)	4.8 ± 1.0	3.9 ± 0.8	3.2 ± 0.7	< 0.001

p: Repeated measures ANOVA test

Yilmaz and Görgi (2025)

Periodontitis and Cognitive decline

- Periodontal bacteria is a significant risk factor in the development of cognitive decline.
  - *Periodontitis is linked to a six-fold increase in the rate of cognitive decline*
- Many studies show a *strong correlation* between gingivitis/periodontitis and cognitive decline, but to establish a direct cause-and-effect relationship is challenging due to the complexity of both conditions.

Periodontitis and Cognitive decline

- *Cognitive performance* of periodontitis patients were significantly worse than in periodontally healthy patients ($p<0.001$)
- *Dietary anti-inflammatory patterns* reduced the inflammatory burden in periodontal lesions;
- Adherence to *pro-inflammatory dietary patterns* are associated with greater cognitive decline ($p<0.001$)

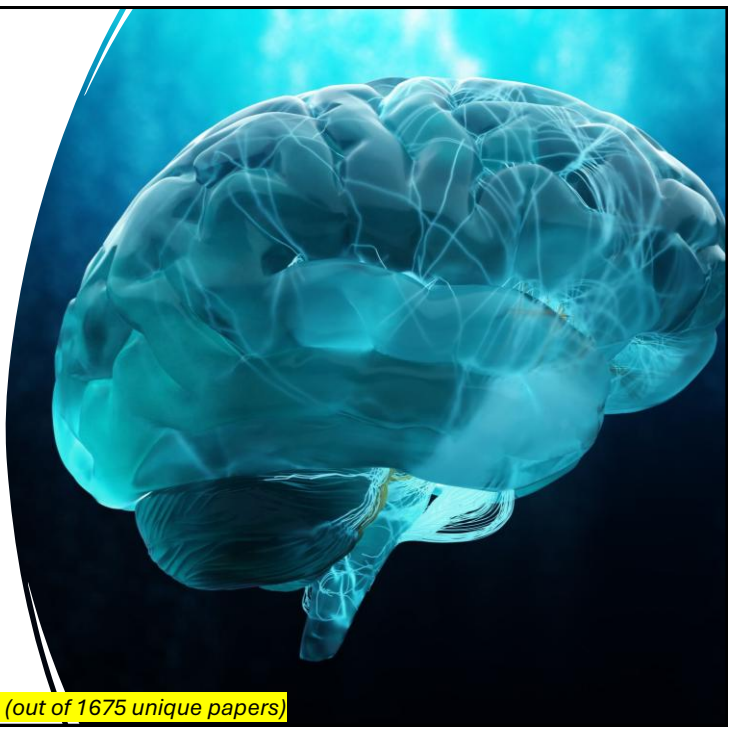
Botelho et al. (2021)

Periodontitis as a Risk Factor for AD

- *Fusobacterium nucleatum, P gingivalis* and *Prevotella intermedia* were found in higher percentages in AD patients than in non-AD patients.
- *Immunological response to these bacteria years before cognitive impairment suggests that the bacterial load of periodontal disease could contribute to the risk of developing AD.*
- Baseline assessment → development of AD was 9.6 yrs

Pruntel et al. (2024) – Systematic review of 17 studies (out of 1675 unique papers)

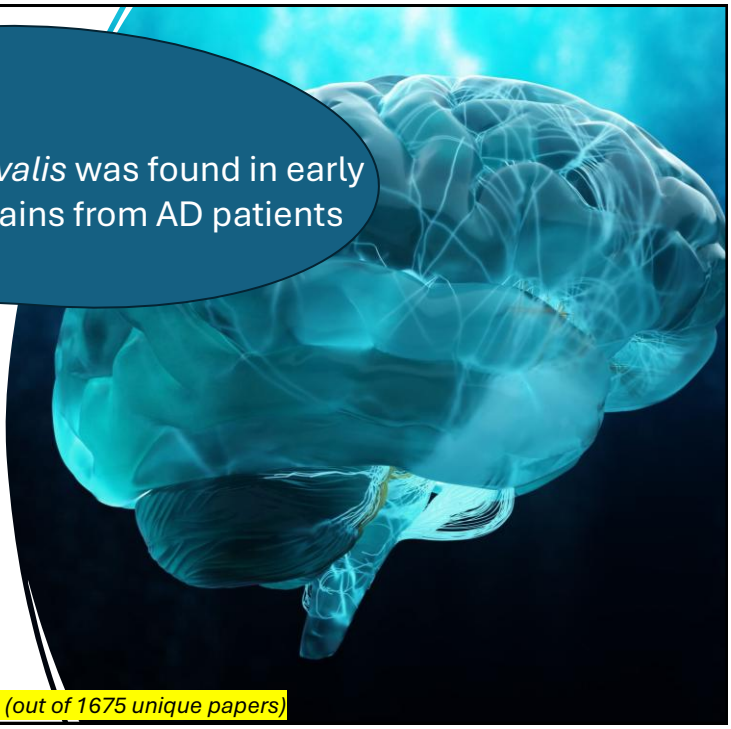
- LPS
- IgG
- Cytokines
- APP



Pruntel et al. (2024) – Systematic review of 17 studies (out of 1675 unique papers)

LPS from *P gingivalis* was found in early post-mortem brains from AD patients

- LPS
- IgG
- Cytokines
- APP



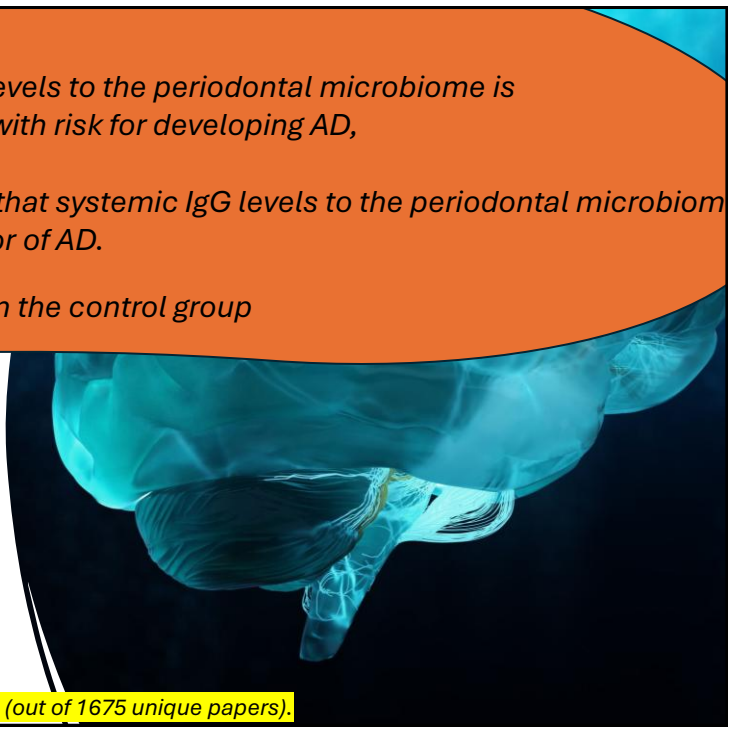
Pruntel et al. (2024) – Systematic review of 17 studies (out of 1675 unique papers)

Higher IgG levels to the periodontal microbiome is associated with risk for developing AD,

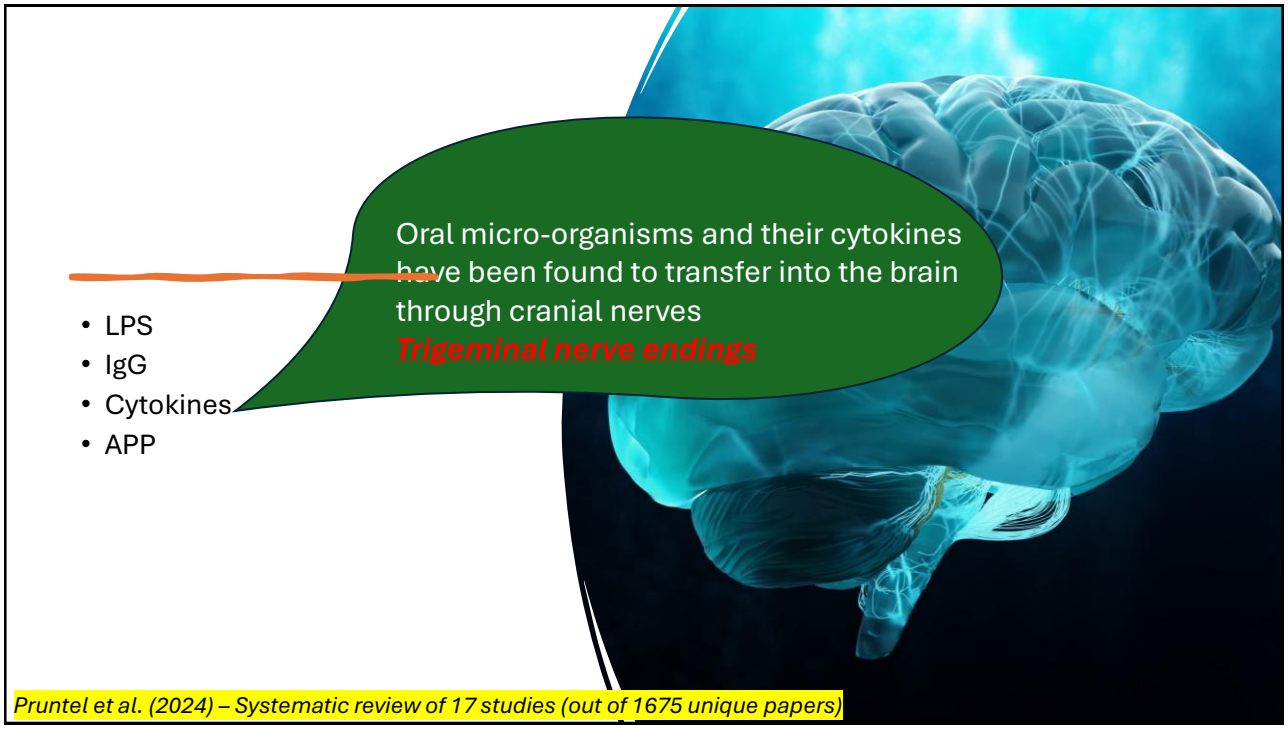
This means that systemic IgG levels to the periodontal microbiom is a predictor of AD.

***NOT** found in the control group*

- LPS
- IgG
- Cytokines
- APP

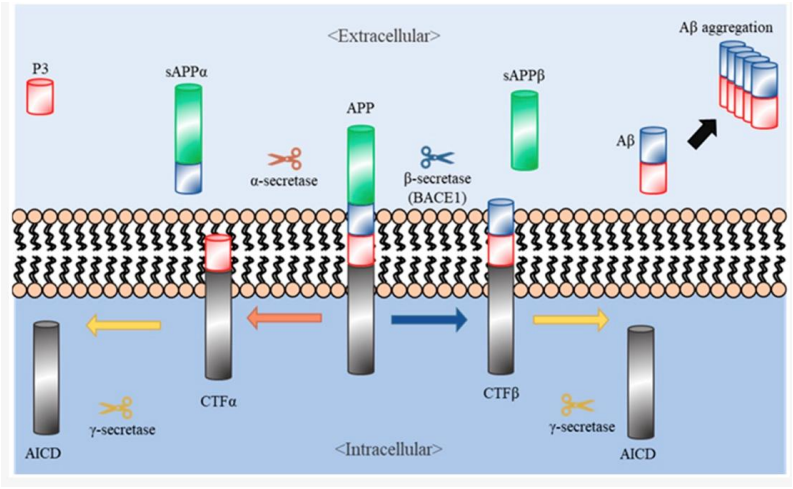


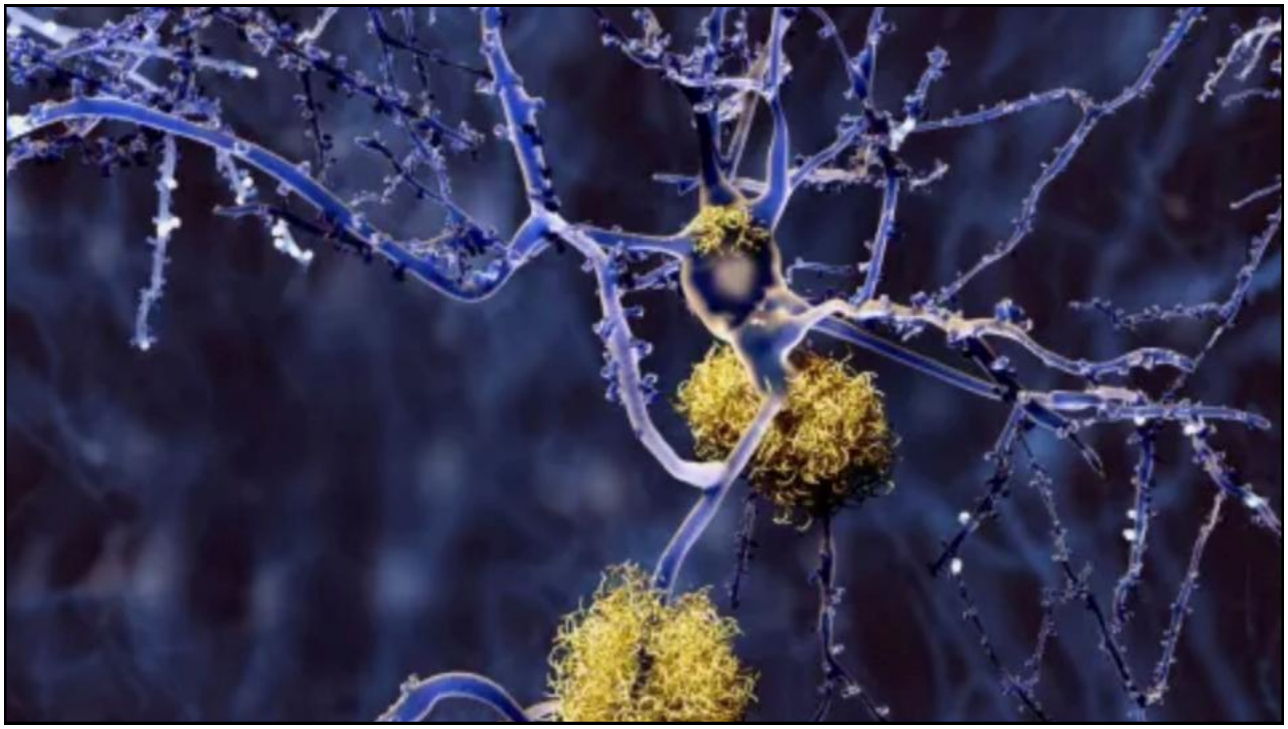
Pruntel et al. (2024) – Systematic review of 17 studies (out of 1675 unique papers).



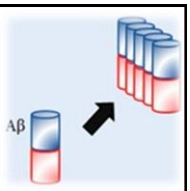
APP

- sAPPα → Neurotrophic and Neuroprotective factor in brain tissue
- sAPPβ →Aβ → Amyloid plaques



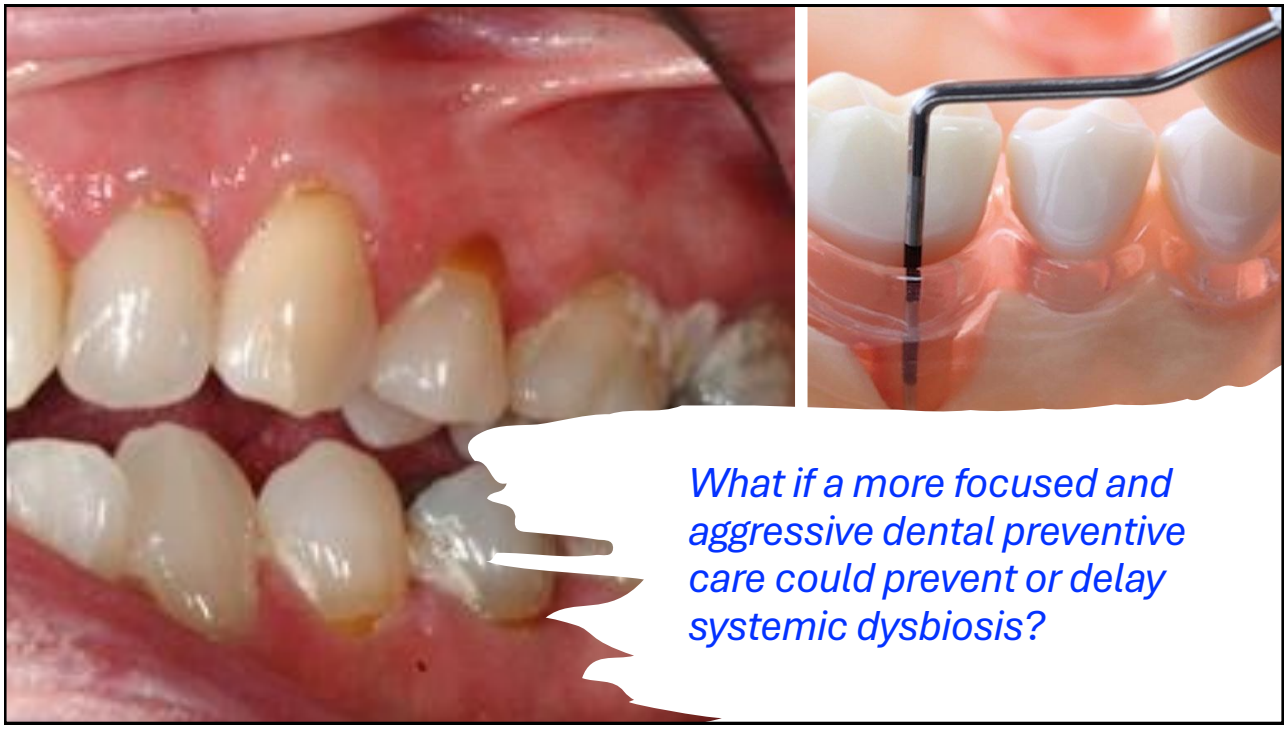


The Amyloid β protein (Aβ)



- Patients with moderate-to-severe periodontitis has a 3-5x higher risk of abnormal Aβ accumulation in the brain (amyloid PET scans)
- *Even cognitively healthy patients with periodontitis have higher levels of Aβ in the brain compared to those without periodontitis.*
- *P gingivalis*-induced periodontitis elevates Aβ deposition in the brain
- *All Aβ findings imply that poor oral health enhance and deepen periodontal pockets and lead to an increased risk of amyloid accumulation in the brain*

Pruntel et al. (2024) – Systematic review of 17 studies (out of 1675 unique papers); Kong et al. (2025); Gil-Montoya et al. (2025)



Dietary components influence on Biofilm dynamics

Positive influence

- ***Dairy products***
  - Decrease in cariogenic bacteria
  - Buffers pH
  - Balanced oral microbiome
- ***High fiber diets***
  - Stimulates salivary flow
  - Lower levels of pathogenic bacteria
- ***Probiotics***
  - Modulates oral microbiome
  - Reduce inflammation
  - Suppress pathogenic bacteria

Negative influence → Tissue inflammation

- ***Sugars and refined carbohydrates***
  - Bacterial adhesion
  - Acid production
  - Biofilm maturation
- ***Starchy foods***
  - Sticks to teeth
  - Enhance cariogenic potential
- ***Acidic foods and beverages***
  - Erosive tooth wear
  - Alters biofilm composition – favors acid-tolerant species

Foods that promote beneficial oral bacteria

- ***Nitrate-rich vegetables***
  - Nitrates are metabolized by oral bacteria into nitric oxide, which supports vascular health and promotes the growth of *Neisseria* and *Haemophilus*, bacteria linked to better cognitive performance
- ***High-fiber plant-based foods***
  - Supports microbial diversity
- ***Polyphenol-rich foods***
  - Anti-microbial and anti-inflammatory properties
- ***Fermented foods***
  - Restore microbial balance and reduce inflammation in Periodontal pockets
- ***Healthy fats***
  - Anti-inflammatory pathways and mucosal integrity
- ***Water, and Saliva-stimulating foods***
  - Stimulating salivary flow (anti-microbial peptides and buffering of the oral pH)

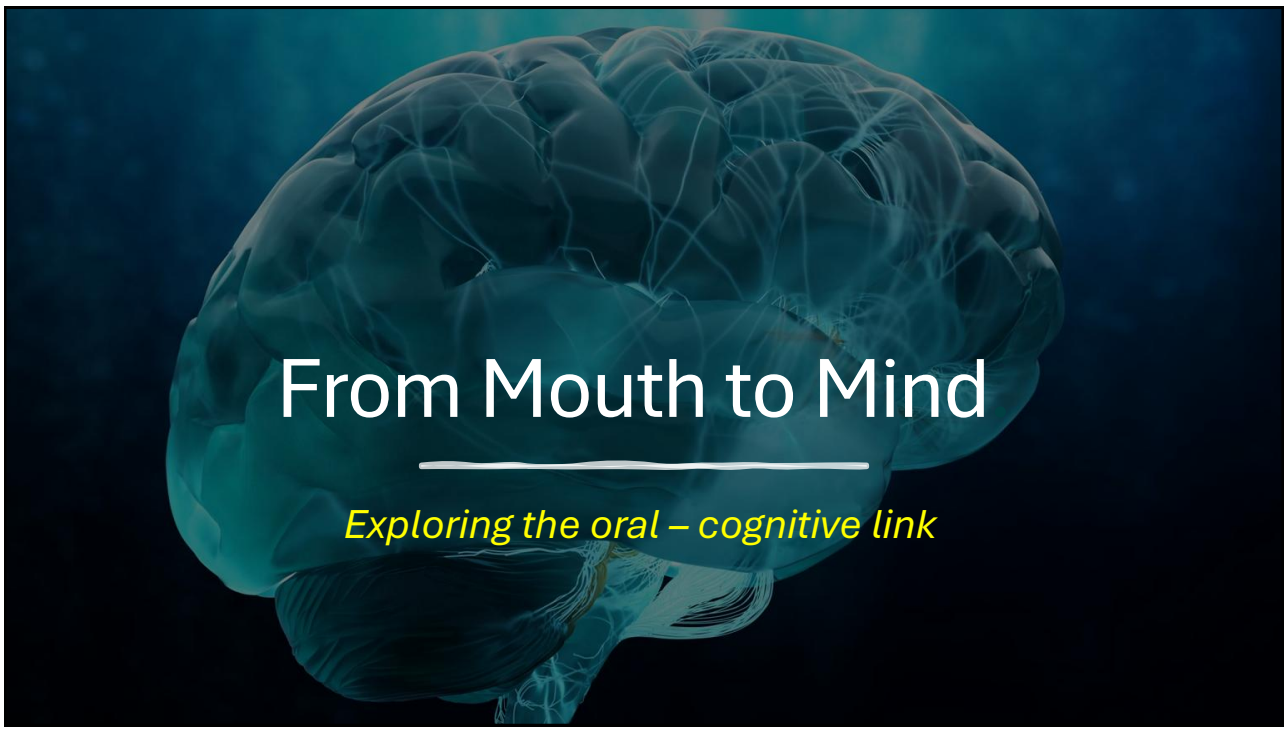
Consistency of Foods

- **The physical consistency** of foods is important due to their retention time in the mouth.
- Liquids are cleared rapidly, and *sticky foods* are cleared slowly.
- High retention rates are found in foods such as *sweet biscuits*, *crackers* and *potato chips*.

Necessary Dentistry

Role of interdisciplinary care

- *Oral health underscores a simple truth: all care must intersect*



Questions ☺