



Review Article

Alzheimers disease: A review

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ABSTRACT

The relationship of oral contamination and oral aggravation to certain non-transmittable interminable infections (NCDs) has underlined the significance of the mouth to the body and shows how the strength of the oral hole can affect general wellbeing. Explicit connections have been distinguished for oral contamination/oral aggravation and certain fundamental illnesses and conditions, basically cardiovascular/cerebrovascular ailments, diabetes mellitus, pregnancy results, and respiratory infections. Another affiliation that has gotten a lot of intrigues is the relationship between oral contamination and psychological disability, explicitly the sort of dementia known as Alzheimer's disease. In this review we will tell you about the relation between oral and Alzheimer disease.

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1. Introduction

Once upon a time, individuals accepted that oral health isn't associated with any disease. From that point forward, a few investigations have been directed that demonstrate something different. Your oral health is related to a wide range of functions in your body. Science has connected oral hygiene to numerous frameworks, from psychological well-being to fertility in men and women, digestion, absorption, and the resistant framework are both emphatically affected by oral cleanliness, and even your blood might be contaminated because of obstruction on your teeth.¹ Could there be an association between poor oral cleanliness and Alzheimer's? As dental specialists we have to do something about oral wellbeing for all patients.² I have made it my central goal to teach people in general on how best to think about your friends and family when they get a finding of dementia or Alzheimer's. Because of individual experience, I see precisely how troublesome the determination can be for patients and families.³ There

is a need to facilitate that nervousness however much as could reasonably be expected, which is the reason I have committed an additional chance to consider the exceptional analysis of dental issues managing Alzheimer's from tolerant train patients to teach and join forces with families.⁴

Alzheimer's ailment (AD) patients show neuroinflammation predictable with contamination, including microglial enactment, inflammasome initiation, supplement actuation, and changed cytokine profiles.^{5,6} Irresistible operators have been found in the cerebrum and hypothesized to be associated with AD, however powerful proof of causation has not been set up.⁷ The ongoing portrayal of amyloid-beta as an antimicrobial peptide has reestablished enthusiasm for distinguishing a conceivable irresistible reason for AD.^{8–10} Interminable periodontitis (CP) and contamination with *Porphyromonas gingivalis*—a cornerstone pathogen in the improvement of CP¹¹ have been distinguished as noteworthy hazard factors for creating amyloid-beta plaques, dementia, and AD.^{12–16}

Mini-Mental State Examination scales) over 6 months contrasted with AD patients without dynamic CP, bringing

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up issues about potential components basic these discoveries.¹⁷ In Apo e mice, oral contamination with *P. gingivalis*, yet not with two other oral microscopic organisms, brings about mind contamination and enactment of the supplement pathway.¹⁸ In transgenic mice overexpressing transformed human amyloid forerunner protein (hAPP-J20), oral contamination with *P. gingivalis* impedes psychological capacity, expands the statement of AD-like plaques, and results in alveolar bone misfortune contrasted with control hAPP-J20 mice.¹⁹ *P. gingivalis* lipopolysaccharide has been distinguished in human AD minds,²⁰ advancing the speculation that *P. gingivalis* contamination of the mind assumes a job in AD pathogenesis.²¹

P. gingivalis is mostly found during gingival and periodontal diseases; notwithstanding, it can likewise be found at low levels in 25% of sound people with no oral illness.²² Transient bacteremia of *P. gingivalis* can happen during basic exercises, for example, brushing, flossing, and biting, just as during dental techniques,²³ bringing about recorded translocation to an assortment of issues including coronary corridors,²⁴ placenta,²⁵ and liver.²⁶ An ongoing report found that 100% of patients with cardiovascular ailment had *P. gingivalis* blood vessel colonization.²⁷ *P. gingivalis* is an asaccharolytic Gram-negative anaerobic bacterium that produces significant harmfulness factors known as gingipains, which are cysteine proteases comprising of lysine-gingipain (Kgp), arginine-gingipain A (RgpA), and arginine-gingipain B (RgpB). Gingipains are emitted, moved to external bacterial layer surfaces, and incompletely discharged into the extracellular milieu insolvent and external layer vesicle (OMV)–related structures.^{28,29} Kgp and RgpA/B are basic for *P. gingivalis* endurance and pathogenicity, assuming basic jobs in having colonization, inactivation of host protections, iron and supplement obtaining, and tissue annihilation.^{30,31}

2. Porphyromonas Gingivalis is seen in Adrenal Cortex

CSF is viewed as a "window" into cerebrum disease, giving knowledge into the neuropathogenesis of irresistible specialists.³² Subsequently, led an imminent pilot study utilizing CSF gathered from 10 patients determined to have likely AD who had gentle to direct intellectual impedance. CSF and coordinated spit tests were gathered and broke down for *P. gingivalis* DNA by qPCR location of the hmuY quality.³³ Positive and negative controls, like the standard of care for identification of other cerebrum contaminations in CSF, were utilized.^{34,35} We had the option to recognize and measure duplicates of the hmuY quality by qPCR in CSF in 7 of the 10 clinically analyzed AD patients, with *P. gingivalis* load running from 10 to 50 duplicates of CSF and the relative fluorescence force of the qPCR items on agarose gel was reliable with the qPCR information. Sequencing of the endpoint PCR items from CSF affirmed the nearness of the hmuY quality. We at that point measured the *P. gingivalis*

load in the coordinating salivation tests from every one of the 10 patients. Each of the 10 coordinating spit tests was certain for *P. gingivalis* by qPCR measure of the hmuY quality. Similarly, as with the cerebrum tests noted above, for a PCR-negative control, we examined CSF tests for the nearness of *H. pylori* utilizing techniques with a similar affectability concerning *P. gingivalis*. The entirety of the CSF tests was negative for *H. pylori*. The CSF information is given extra proof to *P. gingivalis* contamination in the mind of AD patients.

3. Oral Bacteria Associated with Alzheimers Disease

The oral cavity harbors a great scope of bacterial phylotypes.³⁶ Atomic recognizable proof strategies have distinguished near 900 diverse overwhelming bacterial types of which 35% can't yet be refined.³⁷ The oral microbiome profiles have all the earmarks of being individualized,³⁸ implying that bacterial microbiomes can fluctuate both subjectively and quantitatively between people, even though there are likewise huge covers. Every individual can harbor up to 200 distinctive bacterial taxa in their mouth and there is a huge variety in the microbiota in various oral destinations.^{39,40} Moreover, the creation of the oral microbiota independent of being indigenous or pathogenic in the oral hole continues changing taking into account significant oral ailments (caries, gum disease, forceful and constant periodontitis, periodontal-endodontic injuries, periimplantitis, and mucositis).⁴¹ Especially, plaque-induced oral maladies, for example, periodontitis are related to an adjustment in the oral microbiota. There is a transcendence of anaerobic microscopic organisms in the oral depression. Numerous of the major periodontal microorganisms are anaerobic, for example *Porphyromonas gingivalis*, *Treponema denticola*, and *Tannerella forsythia*. The wealth of anaerobes will in general increment with the improvement of plaque-actuated oral illnesses.

4. Oral Yeast Associated with Alzheimers Disease

Oral yeast disease speaks to an optional sharp disease especially including *Candida albicans*, yet progressively non-*albicans* species, for example *Candida glabrata*. With a developing populace of old, extreme foundational contagious contaminations have expanded significantly in this age bunch during the most recent 30 years.^{42,43} Oral yeasts can be found in periodontal pockets, in root channels, on the mucosae, and underneath false teeth (dental replacement stomatitis).^{44,45} Dental replacement stomatitis is predominant in older wearing false teeth that are vigorously debated with yeasts which can be a wellspring of fundamental mycosis. Spread mycoses have as of late been accounted for in AD patients.^{46,47} Parasitic atoms including proteins and polysaccharides [(1,3)- β -glucan] were distinguished infringe blood serum, and parasitic

proteins and DNA were shown by PCR in mind tissue of AD patients. Chitin-like parasitic structures have additionally been found in the Promotion cerebrum⁴⁸ and chitinase action has been proposed as an amazing biomarker of AD.⁴⁹ In AD cerebrums, cytoplasmic material in few cells was focused on antibodies with immunoreactivity to yeast cells.⁵⁰ These discoveries were steady with the possibility that neurons can be contaminated by parasites. Strangely, antifungal treatment turned around the clinical indications of some AD patients.^{51,52}

5. Periodontal Disease and Alziemers Disease

There is expanding proof for a relationship between constant periodontitis and LOAD.⁵³ Cross-sectional also, longitudinal investigations have shown that gingival dying, loss of periodontal connection, periodontal testing profundity, alveolar bone misfortune, and antibodies to periodontal pathogens are essentially connected with lower psychological capacity and decay after change for co-variables. Intense stage proteins, counting cytokines are conceivable backhanded connections between periodontal pathogens as well as their destructiveness factors.^{54,55} Old regularly show disregard of oral cleanliness which can invigorate repetitive incessant oral contamination.⁵⁶ This again advances aggravation which can prompt disarray and dementia.^{57–59} In 152 subjects 5070 years old enough who were followed for a long time, more prominent degrees of periodontal aggravation corresponded with lower intellectual levels.⁶⁰ Moreover, gingival draining and misfortune of periodontal connection were fundamentally related to psychological debilitation in an associate of 5,138 individuals matured 2059 years.⁶¹ In 144 nuns, those encoding APOE ϵ 4 and who had fewer teeth experienced progressively quick psychological decay than those with neither or both of these chance variables.⁶² Clinical and epidemiological investigations that went on the defensive are related to poor memory.^{63,64} In another investigation of 597 network abiding men followed for a long time, tooth misfortune, expanding periodontal pocket profundities, and movement of alveolar bone misfortune were related to weakened discernment especially in those more than 45 years old.⁶⁵ As of late, de Souza Rolim et al.⁶⁶ found that periodontal diseases were more successful in patients with gentle AD than in solid subjects. Another fascinating element related to the pathogenesis of AD is the low degree of disease by 'commensals unhindered'.⁶⁷ These 'immuno-endured' microorganisms may quietly increase in locales outside of their essential specialty and progressing contamination at their auxiliary area may have critical pernicious impacts upon the strength of the older or unbalanced host with a current immunocompromised status.^{68,69}

6. Conclusion

The relationship of oral contamination and oral aggravation to certain non-transmittable interminable infections (NCDs) has underlined the significance of the mouth to the body and shows how the strength of the oral hole can affect general wellbeing. Explicit connections have been distinguished for oral contamination/oral aggravation and certain fundamental illnesses and conditions, basically cardiovascular/cerebrovascular ailments, diabetes mellitus, pregnancy results, and respiratory infections. With the above information it is clear that oral infection or oral health can be a cause for Alzheimer's.

7. Source of Funding

None.

8. Conflict of Interest

None.

References

1. Kwon JH, Olsen MA, Dubberke ER. The Morbidity, Mortality, and Costs Associated with Clostridium difficile Infection. *Infect Dis Clin North Am*. 2015;29(1):123–34.
2. Narayan PJ, Kim SL, Lill C, Feng S, Faull RLM, Curtis MA, et al. Assessing fibrinogen extravasation into Alzheimer's disease brain using high-content screening of brain tissue microarrays. *J Neurosci Methods*. 2015;247:41–9.
3. Kolb HC, Finn MG, Sharpless KB. Click Chemistry: Diverse Chemical Function from a Few Good Reactions. *Angew Chem Int Edition*. 2001;40(11):2004–21.
4. Poirier JL, Čapek R, Koninck YD. Differential progression of Dark Neuron and Fluoro-Jade labelling in the rat hippocampus following pilocarpine-induced status epilepticus. *Neurosci*. 2000;97(1):59–68.
5. Wyss-Coray T, Rogers J. Inflammation in Alzheimer Disease—A Brief Review of the Basic Science and Clinical Literature. *Cold Spring Harb Perspect Med*. 2012;2(1):a006346.
6. Kaushal V, Dye R, Pakavathkumar P, Foveau B, Flores J, Hyman B, et al. Neuronal NLRP1 inflammasome activation of Caspase-1 coordinately regulates inflammatory interleukin-1-beta production and axonal degeneration-associated Caspase-6 activation. *Cell Death & Differentiation*. 2015;22(10):1676–1686. Available from: <https://dx.doi.org/10.1038/cdd.2015.16>. doi:10.1038/cdd.2015.16.
7. Mawanda F, Wallace R. Can Infections Cause Alzheimer's Disease? *Epidemiol Rev*. 2013;35(1):161–80.
8. Kumar DKV, Choi SH, Washicosky KJ, Eimer WA, Tucker S, Ghofrani J, et al. Amyloid- β peptide protects against microbial infection in mouse and worm models of Alzheimer's disease. *Sci Transl Med*. 2016;8(340):340ra72.
9. Soscia SJ, Kirby JE, Washicosky KJ, Tucker SM, Ingelsson M, Hyman B, et al. The Alzheimer's Disease-Associated Amyloid β -Protein Is an Antimicrobial Peptide. *PLoS ONE*. 2010;5(3):e9505.
10. Spitzer P, Condic M, Herrmann M, Oberstein TJ, Scharin-Mehlmann M, Gilbert DF, et al. Amyloidogenic amyloid- β -peptide variants induce microbial agglutination and exert antimicrobial activity. *Sci Rep*. 2016;6(1):32228.
11. Darveau RP, Hajishengallis G, Curtis MA. Porphyromonas gingivalis as a Potential Community Activist for Disease. *J Dent Res*. 2012;91(9):816–20.
12. Kaye EK, Valencia A, Baba N, Spiro A, Dietrich T, Garcia RI. Tooth Loss and Periodontal Disease Predict Poor Cognitive Function in Older Men. *J Am Geriatr Soc*. 2010;58(4):713–8.

13. Gatz M, Mortimer JA, Fratiglioni L, Johansson B, Berg S, Reynolds CA, et al. Potentially modifiable risk factors for dementia in identical twins. *Alzheimer's & Dement*. 2006;2(2):110–7.
14. Stein PS, Desrosiers M, Donegan SJ, Yepes JF, Kryscio RJ, et al. Tooth loss, dementia and neuropathology in the Nun Study. *J Am Dent Assoc*. 2007;138(10):1314–22.
15. Kamer AR, Pirraglia E, Tsui W, Rusinek H, Vallabhajosula S, Mosconi L, et al. Periodontal disease associates with higher brain amyloid load in normal elderly. *Neurobiol Aging*. 2015;36(2):627–33.
16. Noble JM, Borrell LN, Papapanou PN, Elkind MSV, Scarmeas N, Wright CB. Periodontitis is associated with cognitive impairment among older adults: analysis of NHANES-III. *J Neurol, Neurosurg Psychiatry*. 2009;80(11):1206–11.
17. Poole S, Singhrao SK, Chukkappalli S, Rivera M, Velsko I, Kesavalu L, et al. Active Invasion of Porphyromonas gingivalis and Infection-Induced Complement Activation in ApoE^{-/-} Mice Brains. *J Alzheimer's Dis*. 2014;43(1):67–80.
18. Ishida N, Ishihara Y, Ishida K, Tada H, Funaki-Kato Y, Hagiwara M, et al. Periodontitis induced by bacterial infection exacerbates features of Alzheimer's disease in transgenic mice. *NPJ Aging Mech Dis*. 2017;3(1):15.
19. Poole S, Singhrao SK, Kesavalu L, Curtis MA, Crean S. Determining the Presence of Periodontopathic Virulence Factors in Short-Term Postmortem Alzheimer's Disease Brain Tissue. *J Alzheimers Dis*. 2013;36(4):665–77.
20. Singhrao SK, Harding A, Poole S, Kesavalu L, Crean S. Porphyromonas gingivalis/Periodontal Infection and Its Putative Links with Alzheimer's Disease. *Mediators Inflamm*. 2015;2015:1–10.
21. Griffen AL, Becker MR, Lyons SR, Moeschberger ML, Leys EJ. Prevalence of Porphyromonas gingivalis and Periodontal Health Status. *J Clin Microbiol*. 1998;36(11):3239–42.
22. Forner L, Larsen T, Kilian M, Holmstrup P. Incidence of bacteremia after chewing, tooth brushing and scaling in individuals with periodontal inflammation. *J Clin Periodontol*. 2006;33(6):401–7.
23. Mahendra J, Mahendra L, Kurian VM, Jaishankar K, Mythilli R. Prevalence of periodontal pathogens in coronary atherosclerotic plaque of patients undergoing coronary artery bypass graft surgery. *J Maxillofac Oral Surg*. 2009;8(2):108–13.
24. Katz J, Chagini N, Shiverick KT, Lamont RJ. Localization of P. gingivalis in Preterm Delivery Placenta. *J Dent Res*. 2009;88(6):575–8.
25. Ishikawa M, Yoshida K, Okamura H, Ochiai K, Takamura H, Fujiwara N, et al. Oral Porphyromonas gingivalis translocates to the liver and regulates hepatic glycogen synthesis through the Akt/GSK-3 β signaling pathway. *Biochim Biophys Acta*. 2013;1832(12):2035–43.
26. Mougeot JLC, Stevens CB, Paster BJ, Brennan MT, Lockhart PB, Mougeot FKB. Porphyromonas gingivalis is the most abundant species detected in coronary and femoral arteries. *J Oral Microbiol*. 2017;9(1):1281562.
27. 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*. 2000;54:15–44.
28. Gui MJ, Dashper SG, Slakeski N, Chen YY, Reynolds EC. Spheres of influence: Porphyromonas gingivalis outer membrane vesicles. *Mol Oral Microbiol*. 2016;31(5):365–78.
29. Grenier D, Roy S, Chandad F, Plamondon P, Yoshioka M, Nakayama K, et al. Effect of Inactivation of the Arg- and/or Lys-Gingipain Gene on Selected Virulence and Physiological Properties of Porphyromonas gingivalis. *Infect Immun*. 2003;71(8):4742–8.
30. Spudich SS, Nilsson AC, Lollo ND, Liegler TJ, Petropoulos CJ, Deeks SG, et al. Cerebrospinal fluid HIV infection and pleocytosis: Relation to systemic infection and antiretroviral treatment. *BMC Infect Dis*. 2005;5(1):98.
31. Yamamoto Y. PCR in diagnosis of infection: Detection of bacteria in cerebrospinal fluids. *Clin Diagn Lab Immunol*. 2002;9:508–14.
32. Espy MJ, Uhl JR, Sloan LM, Buckwalter SP, Jones MF, Vetter EA, et al. Real-Time PCR in Clinical Microbiology: Applications for Routine Laboratory Testing. *Clin Microbiol Rev*. 2006;19(1):165–256.
33. Linz B, Balloux F, Moodley Y, Manica A, Liu H, Roumagnac P, et al. An African origin for the intimate association between humans and Helicobacter pylori. *Nat*. 2007;445(7130):915–8.
34. Potempa J, Pike R, Travis J. The multiple forms of trypsin-like activity present in various strains of Porphyromonas gingivalis are due to the presence of either Arg-gingipain or Lys-gingipain. *Infect Immun*. 1995;63(4):1176–82.
35. Potempa J, Nguyen KA. Purification and Characterization of Gingipains. *Curr Protoc Protein Sci*. 2007;49(1):20.
36. Aas JA, Paster BJ, Stokes LN, Olsen I, Dewhirst FE. Defining the Normal Bacterial Flora of the Oral Cavity. *J Clin Microbiol*. 2005;43(11):5721–32.
37. Dewhirst FE, Chen T, Izard J, Paster BJ, Tanner ACR, Yu WH, et al. The Human Oral Microbiome. *J Bacteriol*. 2010;192(19):5002–17.
38. Imangaliyev S, Keijsers B, Crielaard W, Tsvitvadze E. Personalized microbial network inference via co-regularized spectral clustering. *Methods*. 2015;83:28–35.
39. Segata N, Haake S, Mannon P, Lemon KP, Waldron L, Gevers D, et al. Composition of the adult digestive tract bacterial microbiome based on seven mouth surfaces, tonsils, throat and stool samples. *Genome Biol*. 2012;13(6):R42.
40. Axelsson P, Lindhe J, Nystrom B. On the prevention of caries and periodontal disease. Results of a 15-year longitudinal study in adults. *Journal of Clinical Periodontology*. 1991;18(3):182–189. Available from: <https://dx.doi.org/10.1111/j.1600-051x.1991.tb01131.x>. doi:10.1111/j.1600-051x.1991.tb01131.x.
41. Armitage GC. Development of a Classification System for Periodontal Diseases and Conditions. *Ann Periodontol*. 1999;4(1):1–6.
42. Lewis RE. Overview of the changing epidemiology of candidemia. *Curr Med Res Opin*. 2009;25:173240.
43. Miceli MH, Díaz JA, Lee SA. Emerging opportunistic yeast infections. *Lancet Infect Dis*. 2011;11(2):142–51.
44. Song X, Eribe ERK, Sun J, Hansen BF, Olsen I. Genetic relatedness of oral yeasts within and between patients with marginal periodontitis and subjects with oral health. *J Periodontol Res*. 2005;40(6):446–52.
45. Kumar J, Sharma R, Sharma M, Prabhavathi V, Paul J, Chowdary CD. Presence of Candida albicans in root canals of teeth with apical periodontitis and evaluation of their possible role in failure of endodontic treatment. *J Int Oral Health*. 2015;7:425.
46. Olsen I. Denture stomatitis, Occurrence and distribution of fungi. *Acta Odontol Scand*. 1974;32:32933.
47. Alonso R, Pisa D, Marina AI, Morato E, Rábano A, Carrasco L. Fungal Infection in Patients with Alzheimer's Disease. *J Alzheimer's Dis*. 2014;41(1):301–11.
48. Alonso R, Pisa D, Rábano A, Carrasco L. Alzheimer's disease and disseminated mycoses. *Eur J Clin Microbiol Infect Dis*. 2014;33(7):1125–32.
49. Castellani RJ, Perry G, Smith MA. The role of novel chitin-like polysaccharides in Alzheimer disease. *Neurotoxicity Res*. 2007;12(4):269–74.
50. Watabe-Rudolph M, Song Z, Lausser L, Schnack C, Begus-Nahrmann Y, Scheithauer MO, et al. Chitinase enzyme activity in CSF is a powerful biomarker of Alzheimer disease. *Neurol*. 2012;78(8):569–77.
51. Pisa D, Alonso R, Juarranz A, Rábano A, Carrasco L. Direct Visualization of Fungal Infection in Brains from Patients with Alzheimer's Disease. *J Alzheimer's Dis*. 2014;43(2):613–24.
52. Ala TA, Doss RC, Sullivan CJ. Reversible dementia: A case of cryptococcal meningitis masquerading as Alzheimer's disease. *J Alzheimer Dis*. 2004;6(5):503–8.
53. Cerajewska TL, Davies M, West NX. Periodontitis: a potential risk factor for Alzheimer's disease. *Br Dent J*. 2015;218(1):29–34.
54. Kamer AR, Dasanayake AP, Craig RG, Glodzik-Sobanska L, Bry M, de Leon MJ. Alzheimer's Disease and Peripheral Infections: The Possible Contribution from Periodontal Infections, Model and Hypothesis. *J Alzheimer's Dis*. 2008;13(4):437–49.
55. Watts A, Gatz M, Crimmins EM. Inflammation as a potential mediator for the association between periodontal disease and Alzheimer's disease. *Neuropsychiatr Dis Treat*. 2008;4:865.

56. Holmes C, El-Okl M, Williams AL, Cunningham C, Wilcockson D, Perry VH. Systemic infection, interleukin 1 beta, and cognitive decline in Alzheimer's disease. *J Neurol Neurosurg Psychiatry*. 2003;74:7889.
57. Dunn N, Mullee M, Perry VH, Holmes C. Association between Dementia and Infectious Disease. *Alzheimer Dis Assoc Disord*. 2005;19(2):91–4.
58. Holmes C, Cotterell D. Role of Infection in the Pathogenesis of Alzheimer's Disease. *CNS Drugs*. 2009;23(12):993–1002.
59. Stewart R, Sabbah W, Tsakos G, D'Aiuto F, Watt RG. Oral Health and Cognitive Function in the Third National Health and Nutrition Examination Survey (NHANES III). *Psychosom Med*. 2008;70(8):936–41.
60. Stein PS, Kryscio RJ, Desrosiers M, Donegan SJ, Gibbs MB. Tooth Loss, Apolipoprotein E, and Decline in Delayed Word Recall. *J Dent Res*. 2010;89(5):473–7.
61. Gatz M, Mortimer JA, Fratiglioni L, Johansson B, Berg S, Reynolds CA, et al. Potentially modifiable risk factors for dementia in identical twins. *Alzheimer's Dement*. 2006;2(2):110–7.
62. Kaye EK, Valencia A, Baba N, Spiro A, Dietrich T, Garcia RI. Tooth Loss and Periodontal Disease Predict Poor Cognitive Function in Older Men. *J Am Geriatr Soc*. 2010;58(4):713–8. Available from: <https://dx.doi.org/10.1111/j.1532-5415.2010.02788.x>.
63. Stein PS, Desrosiers M, Donegan SJ, Yepes JF, Kryscio RJ. Tooth loss, dementia and neuropathology in the Nun Study. *J Am Dent Assoc*. 2007;138(10):1314–22.
64. Noble JM, Borrell LN, Papapanou PN, Elkind MSV, Scarmeas N, Wright CB. Periodontitis is associated with cognitive impairment among older adults: analysis of NHANES-III. *Journal of Neurology, Neurosurgery & Psychiatry*. 2009;80(11):1206–1211. Available from: <https://dx.doi.org/10.1136/jnnp.2009.174029>. doi:10.1136/jnnp.2009.174029.
65. de Souza Rolim T, Fabri GMC, Nitrini R, Anghinah R, Teixeira MJ, de Siqueira JTT, et al. Oral Infections and Orofacial Pain in Alzheimer's Disease: A Case-Control Study. *J Alzheimer Dis*. 2013;38(4):823–9.
66. Corrada MM, Paganini-Hill A, Berlau DJ, Kawas CH. Apolipoprotein E genotype, dementia, and mortality in the oldest old: The 90+ Study. *Alzheimer's Dement*. 2013;9(1):12–8.
67. Shoemark DK, Allen SJ. The Microbiome and Disease: Reviewing the Links between the Oral Microbiome, Aging, and Alzheimer's Disease. *J Alzheimer's Dis*. 2014;43(3):725–38.
68. Selkoe DJ. Alzheimer's Disease. *Cold Spring Harb Perspect Biol*. 2011;3(7):a004457.
69. Yankner B, Dawes L, Fisher S, Villa-Komaroff L, Oster-Granite M, Neve R. Neurotoxicity of a fragment of the amyloid precursor associated with Alzheimer's disease. *Science*. 1989;245(4916):417–420. Available from: <https://dx.doi.org/10.1126/science.2474201>. doi:10.1126/science.2474201.

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