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Various Infections and Alzheimer's Disease

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Abstract

Alzheimer's disease (AD) is a major contributor to the worldwide prevalence of dementia. Extracellular-amyloid (A) senile plaques (SP) and intracellular neurofibrillary tangles (NFT) are the neuropathological hallmarks of Alzheimer's disease. Currently, it is believed that both hereditary and environmental variables interact to contribute to the pathophysiology of AD. Despite significant investments in neurological research, the precise molecular basis of AD pathogenesis remains unknown. Multiple studies point to the possibility that pathogenic microorganisms contribute to the development of AD. Microbes were formerly thought to have no connection to Alzheimer's disease, but a rising body of research suggests otherwise. Evidence that these microbes cause AD-specific cognitive and neuropathological deficits and changes is lacking, casting doubt on the hypothesis that AD is an infectious neurological illness. In addition, the gut flora may have a role in AD progression in humans.

Keywords: Microbial Infections; Gut Microbiota; Alzheimer's Disease; Pathophysiology

Introduction

In many parts of the world, AD is the most common form of dementia. Amyloid beta peptides accumulate extracellularly, and neurofibrillary tangles form inside, as hallmarks of its pathophysiology. Despite advanced research in the neurological field, the molecular basis of AD pathogenesis remains unknown. Evidence from multiple research projects suggests that harmful pathogenesis in the gut and elsewhere in the body may play a major role in the progression of AD [1].

Viral Infection and AD

It is still unknown what causes the majority of instances of Alzheimer's disease, but there is mounting evidence that microorganisms, and specifically herpes simplex virus type 1 (HSV-1), the so-called cold sore virus, play a role. After infection, this virus is known to remain dormant in the peripheral nervous system for life, where it can be triggered by factors like stress and immune-mediated mechanisms. High levels of human herpesvirus 6 and 7 have been detected in various brain areas of people with AD. As with primary human neural stem cells, HSV-1 infection has been shown to create amyloid fibrillar plaque-like formations in human-induced neural stem cells and human-like 3D brain tissue preparations. While certain herpesviruses have been linked to AD pathology and found in the brains of AD patients, other viruses (and other infections) may possibly play a role in the development of AD. An important risk factor for AD may be chronic inflammation triggered by viral infection. In recent years, neuroinflammation and neuro-immune interactions have emerged as possible causes of neurodegeneration. Due to the negative outcomes associated with elevated neuroinflammation and its capacity to block amyloid clearance, it is possible that viruses (or other pathogens) can

do both of these things to amyloid [1-3].

Bacterial Infection and AD

Infectious bacteria have also been connected to AD. In addition to the viruses already described. There is growing evidence between periodontal disease to Alzheimer's disease. Gum disease, also known as periodontitis, is an infection of the gums that spreads to the bone supporting the teeth, triggering the release of proinflammatory cytokines into the bloodstream and an increase in C-reactive protein. Porphyromonas gingivalis, a gram-negative anaerobic bacterium, is responsible for this condition. Postmortem examination of AD patients' brain tissue has revealed the presence of P. gingivalis and its accompanying toxins, known collectively as gingipains, which are expected to accelerate AD pathology in 96% of cases. P. gingivalis-mediated AD exacerbation is mostly attributable to gingipains. The P. gingivalis-secreted cysteine proteases known as gingipains have been linked to neuronal injury, increased tau synthesis, and the generation of neurotoxic APOE fragments. In addition to contributing to dementia and A plaque formation, P. gingivalis has been found to induce neuroinflammation, activate inflammasomes, and trigger the formation of various multiprotein complexes within the brain's immune system [2,4,5].

Fungal Infection and AD

The pathogenesis of AD may potentially be affected by fungal infection. Researchers have discovered fungus DNA and proteins in the brains of AD patients. More research has shown fungi's genetic material and protein can also be identified in the periphery, suggesting they could serve as useful biomarkers [2,3].

Invertebrate Parasites and AD

Parasite infection has been linked by scientists to neurological disorders including Alzheimer's disease. *Toxoplasma gondii* (*T. gondii*) is a protozoan parasite that can causes encephalitis and other neurological diseases, affecting as much as 50% of the world's population. Neuroinflammation and olfactory dysfunction, both of which have been linked to T. gondii, play a role in the aetiology of AD [2,3].

Gut Microbiota and AD

Via the gut-brain-microbiome axis, which includes cognitive behaviour, gut microbes can influence host brain function and behaviour. Alterations in gut microbiota and physiology, as well as host cognitive behaviour, can be induced by antimicrobials, probiotic supplements, and foodstuffs can either raise or lower the risk of AD. A disturbance in the gut microbiota leads to an increase in intestinal and blood-brain barrier (BBB) permeability, which in turn increases the prevalence of neurodegenerative diseases. The risk of AD may be raised or lowered depending on the metabolites produced by gut microbes and their effects on neurochemical alterations in the host. There is evidence to support the "hygiene hypothesis" for the development of AD, and infections with pathogenic bacteria are known to raise the risk of the disease. All these findings point to a possible gastrointestinal origin for AD, which is linked to an imbalance in the gut flora. It is likely that dietary modification or positive microbiota intervention may emerge as a new treatment for AD by moderating the gut microbiota [3,6,7].

Conclusion

Even though several studies lend credence to the idea that AD has an infectious cause, the mechanisms by which it can be caused are still unclear. Therefore, it is important to focus on detecting the mechanisms by which microbial infection and/or gut microbiota can lead to AD.

References

1. Uwishema O, Mahmoud A, Sun J, Correia IFS, Bejjani N, et al. (2022) Is Alzheimer's disease an infectious neurological disease? A review of the literature. Brain and Behavior 12(8): e2728.

2. Naughton SX, Raval U, Pasinetti GM (2020) The viral hypothesis in Alzheimer's disease: novel insights and pathogen-based biomarkers. Journal of Personalized Medicine 10(3): 74.

3. Alonso R, Pisa D, Fernandez-Fernandez AM, Carrasco L (2018) Infection of fungi and bacteria in brain tissue from elderly persons and patients with Alzheimer's disease. Frontiers in aging neuroscience 10: 159.

4. Sun J, Ludvigsson JF, Ingre C, Piehl F, Wirdefeldt K, et al. (2022) Hospital-treated infections in early- and mid-life and risk of Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis: A nationwide nested case-control study in Sweden. PLoS Med 19(9): e1004092.

5. Hu X, Wang T, Jin F (2016) Alzheimer's disease and gut microbiota. Sci China Life Sci 59(10): 1006-1023.

6. Chandra S, Sisodia SS, Vassar RJ (2023) The gut microbiome in Alzheimer's disease: what we know and what remains to be explored. Molecular Neurodegeneration 18(9): 1-21.

7. Cairns DM, Itzhaki RF, Kaplan DL (2022) Potential involvement of varicella zoster virus in alzheimer's disease via reactivation of quiescent herpes simplex virus type 1. Journal of Alzheimer's Disease 88(3): 1189-1200.