Review of Research Linking Infectious Agents and Alzheimer's Disease

By:

**Abstract:**

 Alzheimer’s Disease (AD) is one of the world’s most leading causes of dementia, it affects the individuals magnitude for critical thinking, understanding, communication and memory. The molecular makeup of AD includes the formation of extracellular β amyloid (Aβ) aggregates, neurofibrillary tangles of hyperphosphorylated tau protein, excessive oxidative damage, the imbalance of biothiols, dysregulated methylation, and a disproportionate inflammatory response. It is known that Microorganisms can trigger pathological changes in the brain that can resemble or induce the buildup of Aβ peptides and promote tau hyperphosphorylation and the small presence of infectious agents are also known to possibly induce both local and systemic inflammatory responses which in return promote cellular damage and neuronal loss. In this microreview we will be discussing the possible link between infectious agents such as viruses (Herpes simplex complex 1, 2, Hepatitis C, influenza), bacteria (*Chlamydia pneumoniae,Aggregatibacter actinomycetemcomitans,Helicobacter pylori*) and eukaryotic unicellular parasites (*Toxoplasma gondii*) and the effect they have on cognitive decline within the context of AD.

**Keywords:** Alzheimer’s Disease, Infectious Agents, SARS-CoV2, Cognitive Decline

**Introduction:**

It is predicted by epidemiologists that by the year 2040 the number of individuals that suffer from cognitive decline will surpass 80 million people worldwide. The makeup of Alzheimer’s Disease (AD) consists of the extracellular buildup of amyloid-beta peptide and the intracellular build up of hyperphosphorylated tau proteins that cause neurological cytoskeleton decline. These pathways can also lead to the death of certain neurons and maybe inflammation.

Discussion: