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Analysis of Evidence Suggesting Alzheimer's Disease is Type 3 Diabetes Mellitus

     Alzheimer's Disease (AD) is a form of dementia that has warranted years of research to understand its pathophysiology as well as attempting to find a successful treatment and cure. AD is degenerative, causing a decrease in cognitive function and memory, eventually lead to death. As of 2015, there were 5.1 million Americans suffering from the disease, with a new case diagnosed every 67 seconds (Alzheimer's Association, 2015). These numbers are expected to expand by 2050, with the prevalence of the disease increasing by 10 million, with another case developing every 33 seconds (Alzheimer's Association, 2015). AD is the sixth leading cause of death in the United States, taking more lives than breast and prostate cancer combined (Alzheimer's Association, 2017). With these numbers, it has become increasingly likely that any given person will at least know someone struggling with the disease.

     The toll that Alzheimer's disease takes on the population has many calling for a cure for the disease. Unfortunately, no such cure exists because even the causes of the disease are not fully known. Due to the fact that AD can only be definitively diagnosed after death, the pathophysiology of the disease has yet to be effectively studied, leading researchers to have to theorize about the cause. One theory that exists is that Alzheimer's is actually a form of diabetes, and some researchers have referred to it as Type 3 diabetes mellitus (DM). The aim of this research project was to extrapolate the development of a systematic mechanism connecting the two conditions using existing data on AD and DM.

     A thorough literature review was performed of previous studies observing the pathophysiological similarities between AD and DM. Observable characteristics found in affected human brain tissue with AD include tangles of neural fibers and accumulation of plaques due to an increase of a protein, tau, and an amyloid protein precursor known as Amyloid-β. While these characteristics can be seen after death, the cause of the increases in tau and Amyloid-β remain unknown. However, early in the progression of AD, it has been noted that the brain's ability to utilize glucose decreases, and a deficiency in energy metabolism is observed (Steen, 2005). Both indicate an impairment in insulin signaling within the body. The pathophysiology of DM also involves an impaired insulin response, causing improper absorption and usage of glucose. The inability to metabolize glucose in the brain is due to several abnormalities in insulin, specifically in the signaling mechanisms of insulin-like growth factors-I and II (Steen, 2005). Additionally, when studying a rat model simulating brain-based DM symptoms, researchers noted Alzheimer's-like deficits. Treating these rats with insulin improved

these deficits (de la Monte, 2008).   When synthesizing these data, I hypothesize that when insulin function is impacted, it induces the overproduction of tau leading to Alzheimer's development. If this mechanism is supported by further research, it will lead to prevention or delay in onset of AD symptoms through management of glucose and insulin utilization.

Sources

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