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A Short Note on the Alzheimer's Disease Pathology

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About the Study

In 1906, Alois Alzheimer speculated that the disease affecting 51-year old Auguste Deter, making her paranoid and suspicious, might have an infectious origin. Pressed by his director supervisor, he ignored certain findings that suggested infection and christened his patients' condition with his own name, calling it Alzheimer's disease. Alzheimer's peers shared his initial skepticism and observed that his patient had all the characteristic signs of tuberculosis. Unfortunately, without the aid of sophisticated laboratory equipment, his conjecture has remained speculation for more than one hundred years and conditions of senile dementia have come to be known as Alzheimer's disease.

After the death of Deter at age 56, Alzheimer studied her brain and identified the hallmark brain changes that characterize Alzheimer's disease "the amyloid beta plaques and neurofibrillary tau protein tangles. Other researchers-notably Oskar Fischer, Alzheimer's main rival-observed the presence of bacteria and commented that amyloid plaque deposits are seen in the brains patients with infections. Later studies showed that Deter had none of the genetic markers for Alzheimer's disease.

In the 1980s, infection was again considered as the causative agent in Alzheimer's disease. However, with the identification of amyloid beta plaque and its derivation from Amyloid Precursor Protein (APP), therapies for Alzheimer's disease have focused on reducing, blocking or eliminating these amyloid plaque deposits. To date, these therapies have ultimately met with virtually no success. This result has prompted researchers to realize that an underlying cause responsible for the neuronal changes needed to be identified.

In recent years, through studying frozen brain tissue from Alzheimer's disease patients stored in "brain banks", researchers have found that these specimens commonly contain microbes, particularly viruses, bacteria, fungi and protozoa. To a lesser extent, some microorganisms have also been found in the brains of age-matched control subjects. It appears that reactivation of viruses and persistent infection with bacteria, fungi and protozoa can initiate a chronic immune system response that leads to an inflammatory process in the brain known as neuroinflammation, which results in neurodegenerative changes. In addition, genome-wide association studies show that this neuroinflammation is the driving force rather than a consequence of the disease process in Alzheimer's disease.

Technological advances have also provided greater insight into the role that the immune system plays in Alzheimer's disease development. Immune system changes such as increased microglial brain cell activation and activation of complement seen in patients with Alzheimer's disease are consistent with changes seen in infection. These advances have also shown that the human body is inhabited by trillions of bacteria and other microbes that are necessary for life. Typically confined to the gut, skin, nose, mouth and vagina, they digest food; produce hormones, essential vitamins and neurotransmitters and help eliminate waste. However, when conditions of leaky gut syndrome, defects in the blood-brain barrier or mitochondrial dysfunction occur, these organisms easily make their way into the brain, setting the stage for neurodegeneration.

Amyloid beta protein has recently been found to be an antimicrobial agent that the body produces in response to infection as an early step in the immune response. Amyloid beta protein offers early benefits in fighting infection, but subsequently, when the infection spirals out of control, this protection directly leads to the production of the destructive amyloid beta oligomers, neuroinflammation and neuronal destruction. Amyloid beta oligomers have been found to be more toxic to neurons and their synaptic connections than the dreaded senile plaque deposits. To the surprise of researchers, many amyloid plaque deposits have also been found in the postmortem brains of elderly individuals with no symptoms of dementia. Researchers have likewise discovered that the specific pathway that the amyloid precursor protein takes directly influences whether the toxic form of amyloid beta protein is produced and senile plaques are formed.

While most studies have focused on herpes viruses as causative agents, many other viruses, bacteria, spirochetes, protozoa and fungi have also been implicated. While senile dementia has been previously been known to occur in cases of syphilis and meningitis, the ability to study the brain in greater detail has led to the discovery that microorganisms particularly herpes viruses and bacteria found in the oral cavity are very common in the brain of individuals with Alzheimer's disease and are also present in individuals with normal cognitive function.

Mycobacterium species are difficult to detect without specific laboratory tests that are ordered if *Mycobacterium* is suspected. These microbes are not detected in a routine culture and sensitivity test. With more than 190 *Mycobacterium* species in existence and *Mycobacterium tuberculosis*, the world's most prevalent infection, along with the fact that a persistent infection can linger for years without apparent symptoms, the role of *Mycobacterium* in association with secondary infectious organisms needs to be considered and its association with Alzheimer's disease should be described in the future research.

While less than 1% of the cases of Alzheimer's disease are directly caused by genes, the other cases can be associated with environmental causes such as high blood glucose levels, vascular disease, heavy metal toxicity, damage to the blood-brain barrier, mitochondrial dysfunction, lack of exercise, nutrient deficiencies, environmental toxins and stress. The increased susceptibility to environmental assault

seen in individuals with predisposing genes for late-onset Alzheimer's disease is described above. Studies likewise suggest that through treatment for the causative microorganism, the development and progression of Alzheimer's disease can be halted.