

A breakthrough in Alzheimer's Disease

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Dear Editor,

I am writing to you in order to highlight one of the causative factors of Alzheimer's disease which is among the most prevalent types of dementia worldwide. Australian researchers discovered one of the possible origins of Alzheimer's disease which is a "breakthrough" according to some. Scientists have gained a better grasp of why and how Alzheimer's disease arises by researching the blood-brain barrier. Their findings point to possible therapy and preventive strategies for the neurodegenerative disorder.¹

Alzheimer's disease is a neurodegenerative illness that affects areas of the brain involved in memory, thinking, and language. Its symptoms range from moderate memory loss to inability to hold conversations to disorientation and mood swings in the environment. Centers for Disease Control and Prevention (CDC) opines that, up to 7 million American residents have Alzheimer's disease. Previous literature has demonstrated that a person's risk of getting Alzheimer's disease is influenced by plethora of factors.² Nevertheless, scientists in Australia have now uncovered a new component that may be to blame for the onset of this neurodegenerative disease. Dr. John Mamo, head of

the Curtin Health Innovation Research Institute at Curtin University in Perth, Australia and lead study author described the new research's findings to Medical News Today.

"To identify new chances to prevent and cure Alzheimer's, we need to understand what truly causes the illness, which is currently unknown," he added.¹ "This work indicates that an excess of potentially hazardous fat-protein complexes in the blood can damage small brain blood vessels called capillaries and then seep into the brain, triggering inflammation and brain cell death," Changes in dietary habits and some medicines may possibly reduce blood concentrations of these hazardous fat-protein complexes, thus lowering the risk of Alzheimer's or slowing disease progression," he concluded.^{1,2,3}

Dr. Mamo and his colleagues are attempting to identify previously unknown origins of Alzheimer's disease. They believe that this may lead to new areas of research and unique possible therapies for the disease.⁴

The researchers utilized two mouse models in their latest study. They genetically changed the test animals' livers such that they produced human amyloid-beta. This is the protein component of the deadly protein-fat complex that scientists suspected was the cause of Alzheimer's disease. The control group showed no genetic changes. The researchers treated both groups to a fear-motivated memory test for cognitive skills over time and recorded the findings.

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The researchers discovered that when amyloid-beta proteins produced in the test mice's liver merged with lipids and went to the brain, they disrupted the correct functioning of the brain's tiny blood vessels, or capillaries. Because of the malfunction in the blood-brain barrier, protein-fat complexes leaked from the blood into the brain, causing inflammation. This inflammation occurred in both the test and control groups, although it began considerably earlier in the test group.⁶ The researchers also looked at a neurodegenerative marker and discovered that it was around two times higher in the test animals than in control mice of the same age. As a result, it was expected that during the cognitive function test, the test mice did around half as well as the control group in terms of learning retention. These findings provide answers to long-standing concerns concerning the function of amyloid-beta in the development of Alzheimer's disease. The importance of the study results was conveyed to MNT by Warren Harding, board chairman of Alzheimer's WA. He stated: "Without significant medical advances like the one made by Prof. Mamo's team, the number of Australians living with dementia is expected to exceed one million by 2058. These findings have the potential to have a large global impact on the millions of people living with Alzheimer's disease."^{1,6}

Previously, it was considered that genetic variables had a significant influence in the likelihood of getting Alzheimer's disease (AD). Early-onset familial Alzheimer's disease is caused by rare mutations in at least three genes. A frequent variation in the apolipoprotein E gene is the main risk factor among families with late-onset AD, and in the local population. However, advanced age remains the most well-established risk factor for Alzheimer's disease. Environmental factors may also play a role in disease manifestation.⁷ Oxidative damage and messenger RNA alterations are two pathogenic causes that are directly linked to aging. Other variables unrelated to aging may be susceptible to

therapeutic intervention in the future, such as estrogen replacement treatment for postmenopausal women, anti-inflammatory medication therapy, and lowering vascular risk factors. Older beliefs, such as aluminum's role in the pathogenesis of Alzheimer's disease, have mainly been abandoned as our understanding of the pathogenic pathways of Alzheimer's disease has evolved.⁸ With limited known risk factors associated with Alzheimer's disease, we have also very limited choice of allopathic treatment for AD patients such as Cholinesterase inhibitors (tacrine, donepezil, metrifonate etc.). Plant extracts such as *alpha-tocopherol*, *selegiline*, *gingko biloba* etc., are also being used widely to treat this neurodegenerative disorder.⁸

The fact that the researchers only used animal models is one of the study's limitations. This means that, despite promising results, more research, particularly in humans, is required. Nevertheless, acknowledging how the amyloid-beta-fat complex affects brain capillaries could start opening potential medical possibilities to either cure Alzheimer's disease or slow down the condition's development. Although, it is a long road from mouse studies to human treatments, but such laboratory research is critical to making the advances needed to combat this severe, and increasingly common, condition.^{2,4}

Keywords: Alzheimer Disease, Blood brain barrier, Beta-amyloid protein, Dementia.

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