Main Cause of Alzheimer’s identified as toxic fat-protein complexes from Bad Processed Foods

New research in mice may help open up new treatment avenues for Alzheimer’s disease.

- **Worldwide, Alzheimer’s disease is one of the most common forms of dementia.**
- **Using mouse models, researchers in Australia have identified one of the likely causes of Alzheimer’s disease. Some have dubbed the finding a “breakthrough.”**
- **By studying the blood-brain barrier, the scientists have come away with a better understanding of why and how Alzheimer’s disease occurs.**
Their findings suggest potential treatment and prevention options for the neurodegenerative condition.

The Centers for Disease Control and Prevention (CDC) estimate that up to 5.8 million people in the United States live with Alzheimer’s disease.

Alzheimer’s disease is a neurodegenerative condition affecting parts of the brain associated with memory, thought, and language. Its symptoms range from mild memory loss to the inability to hold conversations to environmental disorientation and mood changes.

Previous research has suggested that various factors — such as age, family history, diet, and environmental factors — combine to influence a person’s risk of Alzheimer’s disease.

However, scientists in Australia have recently discovered an additional factor that may be responsible for the development of this neurodegenerative condition.

Lead study author Dr. John Mamo, Ph.D. — distinguished professor and director of the Curtin Health Innovation Research Institute at Curtin University in Perth, Australia — explained to Medical News Today the conclusion from the new research.

He said, “To find new opportunities to prevent and treat Alzheimer’s, we need to understand what actually causes the disease, and presently that is not established.”

“This study,” he added, “shows that exaggerated abundance in blood of potentially toxic fat-protein complexes can damage microscopic brain blood vessels called capillaries and, thereafter, leak into the brain, causing inflammation and brain cell death.”

“[Changes] in dietary behaviors and certain medications could potentially reduce blood concentration of these toxic fat-protein complexes,
[subsequently] reducing the risk for Alzheimer’s or [slowing] down the disease progression,” he concluded.

The findings appear in the journal *PLOS Biology*.

**Study design**

Dr. Mamo and his team are working to unearth previously undiscovered causes of Alzheimer’s disease. Their hope is that this may suggest new avenues of investigation and novel potential treatments for the condition.

In their recent study, the researchers used two mouse models. They genetically modified animals in the test group so that their livers would produce human amyloid-beta. This is the protein part of the toxic protein-fat complex that the scientists thought may cause Alzheimer’s disease. The control group had no genetic modifications.

Over time, the researchers subjected both groups to a fear-motivated memory test for cognitive functions and noted the corresponding results.

As well as this test of cognitive function, the scientists harvested various tissue samples from the mice, including samples from the liver, brain, lung, and duodenum. This was to study the impact of the human amyloid-beta on the structure and function of these tissues.

When examining the tissue samples or conducting the cognitive tests, the scientists did not know if the mouse in question was from the test or control group. This information was only revealed once they were ready to start the statistical analysis of the results. This process is called blinding, and it is a research practice that helps reduce the risk of unconscious bias.
What the results say

The researchers found that when the amyloid-beta proteins made in the liver of the test mice combined with fats and traveled to the brain, they interfered with the proper functioning of the brain’s microscopic blood vessels, or capillaries.

This dysfunction in the blood-brain barrier led to the protein-fat complexes leaking from the blood into the brain, resulting in inflammation. This inflammation occurred in both the test group and the control group, but it started at a much younger age in the test group.

Unlike in the control group, this inflammation was also associated with marked degeneration in the brain cells of the mice in the test group when examined under a microscope. The scientists only rarely saw this neurodegeneration in the control mice, and it was usually at a much older age.

The team also assessed a marker of neurodegeneration and found it to be approximately two times greater in the test mice than in control mice of the same age.
So, it was unsurprising that during the test for cognitive function, the test mice performed approximately half as well as the control group at retention of learning.

These findings suggest explanations to long standing questions about the role of amyloid-beta in Alzheimer’s disease development.

Warren Harding, board chairman of Alzheimer’s WA, revealed to MNT the significance of the study results. He said:

“Without significant medical advances like the breakthrough Prof. Mamo’s team has made, it is estimated that the number of Australians living with dementia will exceed 1 million by 2058. [...] These findings may have a significant global impact on the millions of people living with Alzheimer’s disease.”
Understanding how the amyloid-beta-fat complex affects brain capillaries may open up potential medical options to either treat Alzheimer’s disease or slow down the condition’s progression.