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PUFAs and Endotoxins Drive Alzheimer's While Tau and Amyloid Beta Protect Brain

Analysis by Dr. Joseph Mercola | October 17, 2024

STORY AT-A-GLANCE

- Tau protein protects brain cells from oxidative stress and toxic lipids by forming protective lipid droplets in glial cells
- Amyloid beta accumulation may be a protective response in areas of high brain metabolic activity, not the primary cause of Alzheimer's
- Excessive consumption of polyunsaturated fats (PUFAs) in seed oils and exposure to endotoxins disrupt gut health, leading to inflammation and oxidative stress in your brain
- Leaky gut and disturbed microbiome contribute to chronic inflammation, metabolic dysfunction and neurodegenerative conditions like Alzheimer's disease
- Holistic approaches focusing on gut health, mitochondrial function and addressing root causes is more effective than targeting tau and amyloid beta directly

You've likely heard that the accumulation of tau and amyloid beta proteins in the brain cause Alzheimer's disease. However, emerging evidence suggests these proteins may be protective responses to underlying issues, rather than the root cause of the disease.

Research published in Nature Neuroscience adds additional insight to the role of tau protein in brain health, challenging long-held beliefs about its involvement in Alzheimer's disease.[1] The study revealed that tau protects your brain cells from oxidative stress and toxic lipids.

Tau protein, it turns out, is essential for the formation of lipid droplets in glial cells — the support cells that surround and protect your neurons. These lipid droplets act as a defense mechanism, helping to neutralize harmful oxidized lipids that damage your brain cells.

When researchers increased tau levels in glial cells, it disrupted this protective process, making the cells more vulnerable to toxic lipids produced by stressed neurons. In other words, the tau accumulations seen in Alzheimer's disease may be your brain's attempt to combat ongoing damage, rather than the primary culprit.

The Positive Role of Tau in Brain Health

Your brain's glial cells are important for maintaining your cognitive health and have developed a fascinating mechanism to protect your brain from oxidative stress. When your neurons experience high levels of reactive oxygen species (ROS), they produce and put out toxic peroxidated lipids (LPOs) as a self-preservation strategy.

LPOs are what happens to PUFAs once they are damaged. Your glial cells then come to the rescue, taking up these dangerous lipids and storing them in lipid droplets.[2]

This process is like a cellular recycling program, where your glial cells safely contain and break down the toxic lipids that could otherwise harm your neurons. The study found that tau protein is crucial for this protective mechanism to function properly.

Without sufficient tau, your glial cells struggle to form these protective lipid droplets, leaving your neurons more vulnerable to oxidative damage. This discovery highlights the importance of maintaining a delicate balance of tau in your brain — too little is just as problematic as too much.

Accumulation of Amyloid Beta Is a Protective Mechanism

A prevailing theory about Alzheimer's disease has focused on the accumulation of amyloid beta (Aβ) plaques as the primary culprit behind cognitive decline. However,

accumulating research suggests Aβ plays a protective role in the brain.[3]

One study, published in Alzheimer's & Dementia,[4] found a correlation between Aβ accumulation and glucose metabolism in the brains of Alzheimer's patients. Contrary to popular belief, regions with higher average glucose metabolism showed greater Aβ deposition. This suggests that Aβ may accumulate more readily in areas of the brain with higher metabolic activity as a protective measure.

Think of Aβ as your brain's attempt to shield itself from damage in its most active regions. However, this protection appears to have limits. In individual brain regions of Alzheimer's patients, higher Aβ levels corresponded with lower glucose metabolism, indicating that excessive Aβ accumulation may eventually impair normal brain function.

This dual nature of Aβ — protective at first but potentially harmful in excess — could explain why Alzheimer's treatments targeting Aβ removal have been largely unsuccessful.[5]

Despite this, the U.S. Food and Drug Administration continues to approve **risky Alzheimer's drugs**, including Leqembi, which bind to amyloid beta in the brain. Reliance on drugs to reduce amyloid beta is, at best, misguided and, at worst, exposing patients to potentially life-threatening adverse effects for no benefit. Alzheimer's disease is a complex disease that requires a holistic approach for prevention and treatment.

Rather than being the primary causes of neurodegeneration, tau protein and Aβ appear to be your brain's response to underlying damage. Tau, in particular, has been associated with stabilizing microtubules in neurons and may play a role in protecting against oxidative stress. Similarly, Aβ may act as an antioxidant and help seal damaged blood vessels.

Your brain produces these proteins as a defense mechanism, but when the underlying causes of damage persist, their accumulation becomes excessive and potentially harmful. This perspective shift emphasizes the importance of addressing root causes like metabolic dysfunction and inflammation rather than focusing on removing tau and Aβ.

How Seed Oils and Endotoxins Drive Alzheimer's Disease

A thriving gut ecosystem is essential for your overall health, including brain function. This diverse community of microorganisms plays a crucial role in protecting you against various diseases, including Alzheimer's, but diets rich in polyunsaturated fats (PUFAs), including **linoleic acid** found in seed oils, destroy your gut health, leading to a cascade of harmful effects. Bioenergetic researcher Georgi Dinkov explains:[6]

> *"The [Nature Neuroscience] study[7] opined that the ROS seen in AD [Alzheimer's disease] can be caused by excessive lipid accumulation (like diabetes), and such accumulation (even when localized by the brain) can itself easily be caused by a low-carb/high-fat diet."*

Fostering beneficial oxygen-intolerant bacteria in your gut, including important species like Akkermansia, strengthens your intestinal defenses and promotes overall wellness. These beneficial bacteria ferment dietary fibers to produce short-chain fatty acids (SCFAs), particularly butyrate.

Notably, butyrate-producing bacteria like Eubacterium and Eisenbergiella were associated with **lower Alzheimer's risk**.[8] Butyrate nourishes your colonic epithelial cells, reinforcing the intestinal barrier. SCFAs also stimulate mucin production, creating a protective shield against harmful bacteria.

The Dangers of Leaky Gut and Endotoxemia

A reduction in oxygen-intolerant bacteria leads to increased intestinal permeability, or leaky gut. This allows toxins, undigested food particles and harmful microbes to enter your bloodstream, triggering systemic inflammation and chronic health issues. This inflammation may contribute to the underlying damage that prompts the protective accumulation of Aβ and tau in the brain. Oxygen-intolerant bacteria are vital for converting indigestible plant fibers into beneficial fats.

They thrive in an oxygen-free environment, which requires adequate cellular energy to maintain. However, factors like seed oil consumption, exposure to endocrine-disrupting chemicals (EDCs) in plastics and electromagnetic fields (EMFs) impair this energy production, making it difficult to sustain the ideal no-oxygen gut environment.

Further, the toxic lipids that tau helps neutralize are often a byproduct of excessive consumption of PUFAs and exposure to endotoxins. These factors trigger chronic inflammation and oxidative stress in your brain, leading to the cognitive decline associated with Alzheimer's.

A leading cause of death is, if not the number one cause of death, in my view, endotoxemia resulting in septic shock. This occurs when you secrete endotoxin from facultative anaerobes, also known as oxygen-tolerant bacteria, which shouldn't be in your gut. These pathogenic bacteria produce a highly virulent form of endotoxin, or lipopolysaccharide (LPS), which cause inflammation if they cross your compromised gut barrier into systemic circulation.

I am about to introduce a new easy term into the vocabulary to identify this concept which is Mighty Shock. Mighty is short for mitochondria and describes the target of endotoxin, your mitochondria. The reason why endotoxin release is so dangerous is that it poisons and shuts down your mitochondria. I will discuss this topic much more in the future so be prepared for more articles on Mighty Shock.

This chronic low-grade inflammation contributes to the metabolic dysfunction that precedes Aβ accumulation in Alzheimer's disease, as discussed in the earlier study. Thus, leaky gut, or a disturbed microbiome, is one of the fundamental causes of all disease, including neurodegenerative conditions like Alzheimer's.

Improving mitochondrial function and maintaining a healthy gut ecosystem promote beneficial bacteria growth while reducing harmful endotoxin effects. This approach mitigates factors contributing to dementia and other chronic diseases by addressing the root causes of inflammation and metabolic dysfunction, rather than focusing on removing Aβ and tau proteins.

Holistic Approaches to Brain Health

Understanding the protective roles of Aβ and tau, along with the importance of gut health, opens up new avenues for Alzheimer's prevention and treatment. Instead of targeting these proteins directly, future therapies may focus on supporting your brain's natural protective mechanisms while addressing underlying causes of neurodegeneration.

This includes strategies to reduce inflammation and optimize brain metabolism through gut health interventions. By nurturing a healthy gut microbiome and addressing factors that disrupt the balance of oxygen-intolerant and oxygen-tolerant bacteria, you may be able to maintain the protective effects of Aβ and tau while preventing their excessive accumulation.

Armed with this new understanding, you can take proactive steps to support your brain health and reduce your risk of Alzheimer's disease. By adopting a holistic approach to brain health that addresses these underlying factors, you support your brain's natural protective mechanisms and maintain cognitive function as you age. In addition to optimizing your mitochondrial function, the strategies below may also help reduce your Alzheimer's risk.

Avoid gluten and casein (primarily wheat and pasteurized dairy, but not dairy fat, such as butter) — Research suggests there's a link between gluten and neurodegenerative disease.[9] Gluten also makes your gut more permeable, which allows proteins to get into your bloodstream, where they don't belong. This sensitizes your immune system and promotes inflammation and autoimmunity, both of which play a role in the development of Alzheimer's.

Optimize your gut flora by regularly eating fermented foods and lowering your LA intake, including from processed foods. High LA consumption impairs energy production, resulting in the proliferation of pathogenic gut bacteria that produce endotoxin.

Optimize your vitamin D level with safe sun exposure — Low levels of vitamin D in Alzheimer's patients are linked with poor outcomes on cognitive tests. In one study, **vitamin D reduced dementia risk by 40%**.[10]

Keep your fasting insulin levels below 3.

Eat a nutritious diet, rich in folate — Vegetables are your best form of folate, which you can get by eating plenty of vegetables every day. Avoid supplements like folic acid, which is the inferior synthetic version of folate.

Avoid and eliminate mercury and aluminum from your body — Dental amalgam fillings, which are 50% mercury by weight, are one of the major sources of **heavy metal toxicity**. Make sure you use a biological dentist to have your amalgams removed. Sources of aluminum include antiperspirants, cookware and vaccine adjuvants.

Make sure your iron level isn't elevated, and donate blood if it is — Iron accumulations in the brain tend to **concentrate in areas most affected by Alzheimer's**, namely the frontal cortex and hippocampus. Magnetic resonance imaging tests have revealed elevated iron in brains affected by Alzheimer's.

Eat blueberries and other antioxidant-rich foods — Wild blueberries, which have high anthocyanin and antioxidant content, are known to guard against neurological diseases.

Avoid anticholinergics and statin drugs — Drugs that block acetylcholine, a nervous system neurotransmitter, increase your risk of dementia. These drugs include certain nighttime pain relievers, antihistamines, sleep aids, certain antidepressants, medications to control incontinence and certain narcotic pain relievers.

Statin drugs are particularly problematic because they suppress the synthesis of cholesterol, deplete your brain of CoQ10 and neurotransmitter precursors, and prevent adequate delivery of essential fatty acids and fat-soluble antioxidants to your

brain by inhibiting the production of the indispensable carrier biomolecule known as low-density lipoprotein.

Sources and References

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