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Emerging Science: The True Precipitating Cause of Alzheimer's May be Much Simpler Than Previously Believed

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Theme: Science and Medicine

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While the mainstream medical community — to the extent it is interested in preventing/treating disease in the first place and not just capitalizing off of it — has long held that the buildup of amyloid and tau proteins in the brain is the precipitating cause of Alzheimer's disease, along with other degenerative conditions of the brain, the true cause may be much simpler and, critically, much more simply prevented/treated.

First, it's important to understand what amyloid proteins do and why they are found in the nervous system tissue in the first place.

The human immune system has two components: the innate immune system and the adaptive immune system. As the terms suggest, the former is the frontline of defense against pathogens and the latter is comprised of antibodies produced in response to prior encountered pathogens.

The brain, probably for evolutionary reasons, has limited adaptive immunity and relies heavily on innate immunity, of which amyloid plaques are an important part.

Via Science Translational Medicine:

> "Neurodegeneration in Alzheimer's disease (AD) is mediated by soluble oligomeric intermediates generated during fibrillization of the amyloid-β protein (Aβ)…
>
> Members of the evolutionarily ancient family of proteins, collectively known as antimicrobial peptides (AMPs), share many of Aβ's purportedly abnormal activities, including oligomerization and fibrillization (3, 4). For AMPs, these activities mediate key protective roles in innate immunity. AMPs are the first-line of defense against pathogens and act as potent broad-spectrum antibiotics and immunomodulators that

> target bacteria, mycobacteria, enveloped viruses, fungi, and protozoans, and in some cases, transformed or cancerous host cells (5). AMPs are widely expressed and are abundant in brain and other immunoprivileged tissues where actions of the adaptive immune system are constrained…
>
> Synthetic Aβ exhibits potent in vitro antimicrobial activity towards eight common and clinically relevant microbial pathogens."

To amyloid proteins are present in the brain when a pathogen is present. Their long-term presence indicates a long-term infection, which might be sub-clinical, meaning that it presents no obvious symptoms like fever, etc. that a clinician would notice and attempt to treat.

Via Experimental Biology (emphasis added):

> "Researchers are reporting new findings on how bacteria involved in gum disease can travel throughout the body, exuding toxins connected with Alzheimer's disease, rheumatoid arthritis and aspiration pneumonia. They detected evidence of the bacteria in brain samples from people with Alzheimer's and used mice to show that the bacterium can find its way from the mouth to the brain."

Via Journal of Alzheimer's Disease, 2015 (emphasis added):

> "We found over a ten-fold increased occurrence of AD when there is detectable evidence of spirochetal infection… and over a four-fold increased occurrence of AD in a conservative risk estimate (OR: 4.45; 95% CI: 2.33-8.52). We found over a five-fold increased occurrence of AD with Cpn infection. This study shows a strongly positive association between bacterial infection and AD."

The implications of this finding above are profound. If it is the case that Alzheimer's might be prevented or cured by mitigating subclinical infections, then super-cheap (unpatented) antibiotics, antifungals, or antivirals might be the whole or partial solution.

But, of course, how would the pharma vultures generate revenue, then? Simple and cost-effective isn't going to cut the mustard for their shareholders. They're in the business of making money, not treating disease. To the extent that diseases are treated by these companies, such results are incidental to the prime directive. That's what made the industry as lucrative as it is.

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