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Eat This Kind of Meat and You Could End Up With Alzheimer's

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STORY AT-A-GLANCE

- For a number of years now, researchers have theorized and found evidence suggesting Alzheimer's disease may in fact be a type of prion-based disease, capable of being contracted via prion-contaminated meat and transmitted via certain invasive medical procedures
- Research published in 2011 found a prion-like protein called TDP-43 in 25% to 50% of Alzheimer's patients, and research presented in 2014 revealed Alzheimer's patients with TDP-43 were 10 times more likely to have been cognitively impaired at death than those without it
- More recent research adds further weight to this hypothesis, finding the two hallmark proteins associated with Alzheimer's — amyloid beta and tau — act as prions, effectively making it a double-prion disease
- Higher levels of prion-like amyloid beta and tau were found in those with early onset of Alzheimer's who died at an earlier age, with tau buildup showing the strongest correlation
- Compared to a patient who died of Alzheimer's at the age of 90, a patient who died at 40 had on average 32 times higher amounts of tau prions in their brain
- Other recent studies suggest amyloid beta is an antimicrobial peptide, a primary effector protein of your innate immune system that target bacteria, viruses and fungi, which has led to the development of the antimicrobial protection hypothesis of Alzheimer's disease. The presence of beta amyloid may not be the actual cause of Alzheimer's but rather the result of an innate defense mechanism against prion infection

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Prions are abnormal and infectious forms of proteins that collect in brain tissue, causing cells to die. The sponge-like holes left in the brain are a hallmark of transmissible spongiform encephalopathies such as bovine spongiform encephalopathy (BSE, also known as mad cow disease in cows and Chronic Wasting Disease[1] in deer and elk) and Creutzfeldt-Jakob disease[2] (CJD), the human version of BSE.

Both BSE and CJD are the result of a prion infection; both are untreatable and always fatal. Sporadic CJD (sCJD), a form that appears without known risk factors, accounts for nearly 85% of diagnosed CJD cases:[3]

CJD is difficult to diagnose, as taking a brain biopsy to rule out a disease is impractical. However, in 2018, the National Institutes of Health published work from colleagues at the University of California San Diego and San Francisco, showing you can measure the distribution and level of prions in the human eye.[4]

According to Dr. Christina J. Sigurdson, professor of pathology at UC San Diego and Davis, who was on the team,[5] "Our findings have implications for both estimating the risk of sCJD transmission and for development of diagnostic tests for prion diseases before symptoms become apparent."

Alzheimer's Disease Linked to Prions

For a number of years now, researchers have theorized and found evidence suggesting Alzheimer's disease may in fact be a type of prion-based disease,[6,7,8] capable of being contracted via meat[9] and transmitted via certain invasive medical procedures.[10]

Researchers have noted that Alzheimer's behaves like a slow moving version of CJD,[11,12,13] and according to one paper,[14] "Prions are considered a subclass of amyloids in which protein aggregation becomes self-perpetuating and infectious." As reported by Scientific American:[15]

> *"Between 1958 and 1985, a number of individuals with short stature received shots of human growth hormone extracted from the pituitary glands of cadavers … Some of these samples were contaminated with prions that caused certain patients to develop Creutzfeldt-Jakob disease (CJD), a rare and fatal brain disorder.*
>
> *Treatments ceased once these reports came to light, but by that time an estimated 30,000 people had already received the injections. As of 2012, researchers have identified 450 cases of CJD worldwide that are the result of these growth hormone injections and other medical procedures, including neurosurgery and transplants."*

Previous animal research[16] has also found that when tiny amounts of amyloid-beta proteins — which are a hallmark of Alzheimer's — are injected into mice or monkeys, they act as self-propagating "seeds," unleashing a chain reaction of protein misfolding that results in pathology that is very reminiscent of that seen in Alzheimer's patients.

Up to Half of Alzheimer's Patients Have Prion-Like Proteins

Mounting research reveals a compelling link between a protein known as TDP-43 and neurodegenerative diseases such as Alzheimer's, Parkinson's and Lou Gehrig's disease.TDP-43 behaves like the prions responsible for the brain destruction seen in Mad Cow and Chronic Wasting Disease.[17]

According to research[18] published in 2011, TDP-43 pathology is detected in 25% to 50% of Alzheimer's patients, particularly in those with hippocampal sclerosis, characterized by selective loss of neurons in the hippocampus, which is associated with memory loss.

Research presented at the 2014 Alzheimer's Association International Conference also revealed Alzheimer's patients with TDP-43 were 10 times more likely to have been cognitively impaired at death than those without it.[19,20]

Alzheimer's Disease — A Double-Prion Disorder

More recent research by scientists at the University of California San Francisco (UCSF) adds further weight to the hypothesis that Alzheimer's disease is a prion-related disease. The study,[21,22] published in the May, 2019, issue of Science Translational Medicine, found that the two hallmark proteins associated with Alzheimer's — amyloid beta and tau — indeed act as prions, effectively making it a double-prion disease.

Prions, while being misfolded proteins and not viruses or bacteria, have the curious capacity to spread in a self-propagating manner by forcing normal proteins to misfold. The first prion, called PrP, was discovered in the 1980s, when it was identified as the cause of CJD and SBE.[23]

As noted by UCSF,[24] it was "long suspected that PrP was not the only protein capable of acting as a self-propagating prion, and that distinct types of prion could be responsible for other neurodegenerative diseases caused by the progressive toxic buildup of misfolded proteins."

Indeed, by applying recently developed laboratory tests, the UCSF research team was able to measure "self-propagating prion forms of the proteins amyloid beta and tau in postmortem brain tissue of 75 Alzheimer's patients,"[25] confirming previous findings that amyloid plaques and tau tangles spread in much the same way as PrP, causing similar damage but at a slower rate.[26]

Tau Prion Levels Strongly Correlate to Longevity

Importantly, higher levels of prion-like amyloid beta and tau were found in those with early onset of Alzheimer's who died at an earlier age, with tau buildup showing the strongest correlation. Compared to a patient who died of Alzheimer's at the age of 90, a patient who died at 40 had on average 32 times higher amounts of tau prions in their brain. As noted by UCSF:[27]

> *"Alzheimer's disease is currently defined based on the presence of toxic protein aggregations in the brain known as amyloid plaques and tau tangles, accompanied by cognitive decline and dementia.*

> *But attempts to treat the disease by clearing out these inert proteins have been unsuccessful. The new evidence that active Aß and tau prions could be driving the disease … could lead researchers to explore new therapies that focus on prions directly."*

Senior author Dr. Stanley Prusiner, director of the UCSF Institute for Neurodegenerative Diseases, commented on the results:[28]

> *"I believe this shows beyond a shadow of a doubt that amyloid beta and tau are both prions, and that Alzheimer's disease is a double-prion disorder in which these two rogue proteins together destroy the brain.*
>
> *The fact that prion levels also appear linked to patient longevity should change how we think about the way forward for developing treatments for the disease."*

One of the study's lead authors, Carlo Condello, Ph.D., assistant professor of neurology in the Institute for Neurodegenerative Diseases, added:[29]

> *"We have recently seen many seemingly promising Alzheimer's therapies fail in clinical trials, leading some to speculate that we have been targeting the wrong proteins. But what if we just haven't been designing drugs against the distinctive prion forms of these proteins that actually cause disease?*
>
> *Now that we can effectively measure the prion forms of Aß and tau, there's hope that we can develop drugs that either prevent them from forming or spreading, or help the brain clear them before they cause damage."*

What Makes Amyloid Infectious?

A study[30] published in the journal Prion in 2014 sought to determine why certain proteins prone to form amyloids have the capacity to infect their neighbors. Here, too, the author referred to Alzheimer's as a prion disease, specifically with reference to the amyloid plaques formed:

> *"The conformational diseases, linked to protein aggregation into amyloid conformations, range from non-infectious neurodegenerative disorders, such as Alzheimer's disease (AD), to highly infectious ones, such as human transmissible spongiform encephalopathies (TSEs). They are commonly known as prion diseases.*
>
> *However, since all amyloids could be considered prions … it is necessary to find an underlying cause of the different capacity to infect that each of the proteins prone to form amyloids has.*
>
> *As proposed here, both the intrinsic cytotoxicity and the number of nuclei of aggregation per cell could be key factors in this transmission capacity of each amyloid."*[31]

The author goes on to state that while amyloids are universal and share certain internal structural characteristics, "prions represent only a tiny drop in the amyloid ocean." In order for an amyloid to become a prion, something has to occur causing the aggregation process to become self-perpetuating and infectious.

He points out that the Alzheimer's disease process, while similar to that of CJD, is much slower, and doesn't follow the same pathway of transfer (from the spleen to the central nervous system). So, what causes amyloid in an Alzheimer's patient to become infectious? What turns it into a prion? To answer this question, the author turns to research on fungal and yeast prions.

> *"Recent findings in the field have shown that the number of nuclei of aggregation could be a factor that affects the infection capacity of amyloid-prone proteins, just as their intrinsic cytotoxicity does.*
>
> *In both fungal and yeast prions, the number of nuclei of aggregation per cell determines, following Poisson's law, the probability of prion infectivity. Thus, high numbers of nuclei of aggregation per cell result in an increase in infectivity,"* he writes.

He also speculates that cytotoxicity plays a big role, and that "the intrinsic cytotoxicity of each amyloid … could be a key factor in the differentiation between infectious and noninfectious amyloids in humans."

The following year, 2015, the same author, joined by several others, published a second paper[32] in the same journal, titled "Amyloids or Prions? That Is the Question." "Despite major efforts devoted to understanding the phenomenon of prion transmissibility, it is still poorly understood how this property is encoded in the amino acid sequence," they write.

According to this 2015 paper, experiments using yeast prions have demonstrated that in order for prions to form, there must exist "intrinsically disordered sequence regions enriched with a particularly high proportion of glutamine and asparagine."

The Antimicrobial Protection Hypothesis of Alzheimer's

Other recent studies,[33,34,35] meanwhile, suggest the amyloid beta found in Alzheimer's patients is also an antimicrobial peptide (AMP). AMPs are the primary effector proteins of your innate immune system that target bacteria, viruses and fungi. They also act as mediators of inflammation and play a role in cytokine release, angiogenesis and more.[36]

In one such study,[37] the authors suggest amyloid beta, as an AMP, "utilizes fibrillation to protect the host from a wide range of infectious agents." Another study[38] points out that "Ancient origins and widespread conservation suggest the human Aβ sequence is highly optimized for its immune role."

Findings such as these would support the hypothesis that amyloid beta protein might actually be targeting prions and trying to protect the host from infection. In other words, the presence of beta amyloid may not be the actual cause of Alzheimer's but rather the result of an innate defense mechanism against prion infection, perhaps acquired through consumption of prion-infected meat.

A lot of this is still speculative, but it's an intriguing idea. And, while slim, there's some evidence[39] (which has yet to be reproduced) that cross-species prion infections could in

fact occur. As noted in "The Antimicrobial Protection Hypothesis of Alzheimer's Disease," published in the December 2018 issue of Alzheimer's & Dementia:[40]

> *"We explore here a novel model for amyloidogenesis in Alzheimer's disease (AD). This new perspective on AD amyloidosis seeks to provide a rational framework for incorporating recent and seemingly independent findings on the antimicrobial role of β-amyloid and emerging experimental, genetic, and epidemiological data, suggesting innate immune-mediated inflammation propagates AD neurodegeneration …*
>
> *[E]emerging findings are increasingly inconsistent with characterization of Aβ oligomerization as a nonphysiological and exclusively pathological activity. Recent studies suggest Aβ is an ancient, highly conserved effector molecule of innate immunity.*
>
> *Moreover, Aβ oligomerization and β-amyloid generation appear to be important innate immune pathways that mediate pathogen entrapment and protect against infection.*
>
> *NEW AD AMYLOIDOGENESIS MODEL: Recent findings on inflammation-mediated neurodegeneration and the role of Aβ in immunity have led to emergence of the 'Antimicrobial Protection Hypothesis' of AD. In this model, β-amyloid deposition is an early innate immune response to genuine, or mistakenly perceived, immunochallenge.*
>
> *Aβ first entraps and neutralizes invading pathogens in β-amyloid. Aβ fibrillization drives neuroinflammatory pathways that help fight the infection and clear β-amyloid/pathogen deposits. In AD, chronic activation of this pathway leads to sustained inflammation and neurodegeneration.*
>
> *Mounting data link elevated brain microbe levels with AD. The Antimicrobial Protection Hypothesis reveals how increased brain microbial burden may directly exacerbate β-amyloid deposition, inflammation, and AD progression."*

Alzheimer's Is Largely Preventable

It is often believed dementia is a condition that can't be controlled, but there are many factors you can influence to greatly reduce your risk. It is important to address several factors, however, and not focus exclusively on only one or two.

That said, improving your cardiovascular fitness is an excellent place to start, when combined with other approaches to resolve mitochondrial dysfunction, it can be highly effective in preventing cognitive decline.

Other strategies to help you reduce your risk of Alzheimer's disease include eating a ketogenic diet, optimizing your vitamin D and omega-3 levels, eliminating gluten and processed foods, and cyclical (both intermittent and partial) fasting, as detailed in my latest book, "**KetoFast**."

Additionally, one of the most effective and simple strategies for increasing heat shock proteins, which are responsible for refolding the amyloid and tau proteins properly, is near infrared sauna. I personally believe this is a strategy that virtually everyone over 50 should regularly engage in. Please review my engaging interview with Brian Richards below for more details on this valuable therapy.

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