

DMSO AND DEMENTIA

Over 7 million Americans have Alzheimer's — equating to hundreds of billions in potential (Medicare funded) sales each year. Almost all Alzheimer's research for decades has been directed toward eliminating amyloid that destroys brain tissue, even after the basis for much of this work was shown to stem from fraudulent research. Chronic inflammation plays a much larger role in the disease.

Last year, Alzheimer's was estimated to cost the United States 360 billion dollars! The billions spent on amyloid Alzheimer's research have only produced three drugs, all of which offer minuscule benefits and severe side effects. Other affordable remedies are available. DMSO, for example, has incredible neuroprotective qualities that have spared many stroke and spinal cord injury victims from a life of "incurable" disability. Decades of forgotten research also show it treats cognitive impairment and dementia.

[Note: The Need To Know News does not give medical advice but reports the news; please consult with your own health experts before using any treatment].

STORY AT-A-GLANCE

- **Alzheimer's disease is commonly thought to result from abnormal plaque buildup in the brain that gradually destroys brain tissue. Almost all Alzheimer's research for decades has been directed toward eliminating amyloid, even after the basis for much of this work was shown to stem from fraudulent research**
- **The billions spent on amyloid Alzheimer's research have only produced three drugs, all of which offer minuscule benefits and severe side effects**
- **In contrast, affordable and straightforward treatments that reduce dementia or the preceding cognitive impairment have been maligned and buried by the medical industry**
- **DMSO for example, has incredible neuroprotective qualities that have spared many stroke and spinal cord injury victims from a life of "incurable" disability. Decades of forgotten research also show it treats cognitive impairment and dementia**
- **This article will review the great amyloid scam and the simple therapies for cognitive decline we're never told about**

Medicine is strongly biased towards adopting biochemical models of disease as this facilitates costly therapeutics being developed for each disease and hence sustains the medical industry. Unfortunately, in many cases, the biochemical approach to disease, at best, can manage symptoms, and as a result, many conditions remain "incurable" while non-patentable natural therapies that can cure them languish in obscurity.

That's why, despite spending an ever increasing amount of money on Alzheimer's research (e.g., the NIH spent 2.9 billion in 2020 and 3.9 billion in 2024), we've still failed to make any real progress on the disease. This is particularly remarkable given the vast costs to the country (e.g., last year Alzheimer's was estimated to cost the United States 360 billion dollars) and the even greater social costs that accompany it.

THE AMYLOID JUGGERNAUT

In 1906, plaques (of amyloid) in the brain were identified as the cause of Alzheimer's disease. As the years have gone by, the majority of research for treating Alzheimer's disease has been targeted at eliminating these plaques. Unfortunately, to quote a 2022 article:

“Hundreds of clinical trials of amyloid-targeted therapies have yielded few glimmers of promise, however; only the underwhelming Aduhelm has gained FDA approval. Yet A β still dominates research and drug development. NIH spent about \$1.6 billion on projects that mention amyloids in this fiscal year, about half its overall Alzheimer’s funding.

Scientists who advance other potential Alzheimer’s causes, such as immune dysfunction or inflammation, complain they have been sidelined by the ‘amyloid mafia.’ Forsayeth says the amyloid hypothesis became ‘the scientific equivalent of the Ptolemaic model of the Solar System,’ in which the Sun and planets rotate around Earth.”

Note: Frequently, when a faulty paradigm fails to explain the disease it claims to address, rather than admit the paradigm is flawed, its adherents will label each conflicting piece of evidence as a paradox (e.g., the French “paradox” disproves the notion cholesterol causes heart disease) and dig deeper and deeper until they can find something to continue propping up their ideology (e.g., cholesterol reducing statins provide almost no benefit for heart disease while having significant side effects yet continue being pushed on patients).

The consistent failure of the amyloid model to cure Alzheimer’s gradually invited increasing skepticism towards it, which resulted in more and more scientists studying alternative models of the disease. Before long, they found other factors played a far more significant role in causing the disease (e.g., chronic inflammation), and by 2006, this perspective appeared poised to change the direction of Alzheimer’s research.

In response, the amyloid proponents pivoted to defending their failed hypothesis was due not to amyloid clumps, but rather toxic parts of it (oligomers) and a Nature 2006 paper appeared which identified a previously unknown toxic oligomer, A β *56, and provided proof that it caused dementia in rats. This paper cemented both the amyloid beta and toxic oligomer hypotheses (as it provided the proof many adherents to the theory had been waiting for) and rapidly became one of the most cited works in the field of Alzheimer’s research. Its authors rose to academic stardom, produced further papers validating their initial hypothesis, and billions more were invested by both the NIH and the pharmaceutical industry in research of the amyloid and toxic oligomer hypothesis.

It should be noted that some were skeptical of their findings and likewise were unable to replicate this data, but rarely had a voice in the debate:

“The spotty evidence that A β *56 plays a role in Alzheimer’s had [long] raised eyebrows. Wilcock has long doubted studies that claim to use ‘purified’ A β *56. Such oligomers are notoriously unstable, converting to other oligomer types spontaneously. Multiple types can be present in a sample even after purification efforts, making it hard to say any cognitive effects are due to A β *56 alone, she notes — assuming it exists. In fact, Wilcock and others say, several labs have tried and failed to find A β *56, although few have published those findings. Journals are often uninterested in negative results, and researchers can be reluctant to contradict a famous investigator.”

THE AMYLOID SCANDAL

At the end of 2021, a neuroscientist physician was hired by investors to evaluate an experimental Alzheimer’s drug and discovered signs that its data consisted of doctored Western Blots (and therefore erroneous assessments of what oligomers were present within research subjects’ brains). As he explored the topic further, he discovered other papers within the Alzheimer’s literature had been flagged for containing doctored Western Blots.

Note: Western blots, used to test for proteins, are one of the few easily detectable forms of research fraud (e.g., we discovered Pfizer submitted fake Western blots to regulators to “prove” their vaccine worked). **Regrettably, far more undetectable fraud exists throughout the scientific literature** (e.g., independent researchers comparing regulatory submissions discovered Pfizer also submitted doctored data on where the COVID vaccine is distributed in the body).

Before long, the neuroscientist noticed three of those suspect papers had been published by the same author and decided to investigate the author’s other publications. This led him to the seminal 2006 Alzheimer’s publication, which contained clear signs of fraud.

As investigation then uncovered 20 doctored papers written by the author, 10 of which pertained to A β *56 (along with a co-researcher attesting to earlier scientific misconduct by the author).

THE AMYLOID INDUSTRY

One of the remarkable things about this monumental fraud was how little was done about it. For example, the NIH was notified in January 2022, yet in May 2022, beyond nothing being done, the NIH gave the suspect researcher a coveted \$764,792 research grant (signed off by another one of the authors of the 2006 paper). In July 2022, Science published an article exposing the incident and the clear fraud that had occurred. Despite this, the researcher was allowed to remain in his position as a tenured medical school professor. It was not until June 2024 that the 2006 article was retracted at the request of the authors — all of whom denied being at fault and insisted the doctored images had not affected the article’s conclusions.

Eventually, on January 29, 2025, during his confirmation hearing, RFK cited the paper as an example of the institutional fraud and wasted tax dollars within the NIH, and a few days later, the suspect researcher announced his resignation from the medical school professorship (while still maintaining his innocence). This odd behavior (e.g., the medical field continues to insist the proven fraud has not disproven the Amyloid hypothesis) likely results from how much money is at stake — beyond the research dollars, roughly 7 million adults have Alzheimer’s — equating to hundreds of billions in potential (Medicare funded) sales each year.

THE FAILED AMYLOID DRUGS

Recently, a monoclonal antibody that made immune cells target amyloid demonstrated limited success in treating Alzheimer’s — which was embraced as revolutionary by the medical community, the pharmaceutical industry, and drug regulators. In turn, the first new drug received accelerated approval (which the FDA proudly announced). The second then received a quiet backdoor approval (due to the immense controversy surrounding the first), and the third was partially approved a year and a half later.

Each year, JP Morgan (Chase Bank) hosts a private conference for pharmaceutical investors that sets the tone for the entire industry. In 2023, its focus was on the incredible profitability of the new Alzheimer’s drugs and the GLP-1s like Ozempic (which the FDA has also relentlessly promoted). Most remarkably, the corrupt FDA commissioner was a keynote speaker, and a few days before the conference, had enacted the second backdoor approval.

However, despite the rosy pictures painted around the drugs (which each attacked different aspects of amyloids), they were highly controversial as:

- The FDA’s independent advisory panel, in a very unusual move, voted 10-0 (with one abstaining) against approving Aduhelm, the first amyloid drug (which targeted amyloid plaques), but the FDA approved it anyways. In a highly unprecedented move, three of the advisors then resigned, calling it “probably the worst drug approval decision in recent U.S. history.”

- That drug was priced at \$56,000 a year — making it sufficient to bankrupt Medicare, (which attracted a Congressional investigation).
- Brain swelling or brain bleeding was found in 41% of patients enrolled in its studies. Additionally, headaches (including migraines and occipital neuralgia), falls, diarrhea, confusion, and delirium were also notably elevated compared to placebo.
- No improvement in Alzheimer's was noted; rather one analysis found it slowed the progression of Alzheimer's by 20% (although this could have been a protocol artifact rather than a real effect).

The second monoclonal antibody (which targeted amyloid precursors) had a somewhat better risk benefit profile (only 21% experienced brain bleeding and swelling due to reduced targeting of stable amyloid plaques), and 26.4% reduction in the progression of Alzheimer's was detected in the trial (which for context, translated to a 0.45 reduction on a scale where a reduction of at least 1 to 2 points is needed to create an impact which is in any way meaningful for a patient).

The third monoclonal (which targeted amyloid plaques thought to be more pathologic) was also contested as it caused 36.8% of recipients to develop brain bleeding or swelling, like the other amyloid medications, frequently caused headaches and infusion reactions (e.g., nausea, vomiting, changes in blood pressure, hypersensitive reactions or anaphylaxis) and there were reasons to suspect the trial had greatly overstated its minimal benefits.

Remarkably, despite widespread protest against the third drug, the FDA's new advisory panel voted unanimously in favor of it, even though it had a very similar mechanism, efficacy, and toxicity to the previously unanimously rejected amyloid drug.

It should therefore come as no surprise that, when the British Medical Journal conducted an independent investigation, it found that, within publicly available databases, 9 out of 9 (assessable) members of the advisory committee had significant financial conflicts of interest.

Fortunately, despite the aggressive promotion of amyloid drugs and the industry's best attempts to promote the sector, the market somewhat recognized how bad they were. The first drug had its price halved (then was withdrawn as no one wanted it — making around 5 million dollars total), while the other two have had very modest sales (e.g., 290 million for the most popular one).

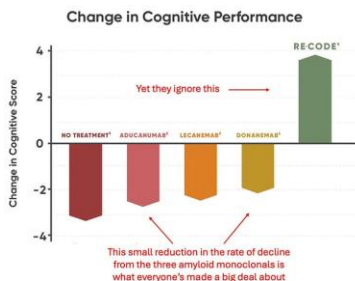
WHAT AMYLOIDS DRUGS SHOW US

From this, four things stand out:

- These drugs consistently damage brain tissue, indicating that their mechanism of action was inherently dangerous (e.g., it creates brain swelling by causing immune cells attacking amyloid also to attack brain tissue, or it creates brain bleeding by removing amyloid plaque that patches vessel walls and stabilizes brain tissue). Remarkably, despite this issue being recognized, it has not deterred the usage of these class drugs.
- Removing amyloid offers minimal benefit and may be counterproductive. In fact, one of the only protocols that has had proven success in treating Alzheimer's instead views amyloid as a protective mechanism the brain uses to prevent further damage.
- An absolutely absurd amount of money and time has been wasted on this endeavor due to the medical field's need to find a patentable drug.
- The focus on these lucrative drugs has diverted attention from other (off-patent) treatments that are more likely to help Alzheimer's patients.

For example, a randomized controlled trial which gave MCTs derived from coconut found that over 6 months, 27 80% remained stable or improved — which for context, is better than what any of the amyloid drug trials showed, and more importantly, does not cause brain bleeds (and costs a lot less than the annual rough \$30,000 cost for those drugs).

Note: Numerous readers have shared that coconut oil improved their relative's dementia. Likewise, very few are aware of a 2022 study that should have revolutionized the entire Alzheimer's field:



Note: The RECODE protocol was based around identifying the underlying cause of a patient's cognitive impairment (as five different things can cause dementia), and then providing appropriate natural therapies to address the applicable cause. Since then, many others have replicated its success in their patients.

DMSO AND DEMENTIA

Dimethyl Sulfoxide (DMSO) is a naturally occurring compound that has a variety of unique healing properties that allow it to rescue tissues from dying and revive those damaged from previous injuries — best demonstrated by decades of evidence showing DMSO can heal strokes, brain bleeds, severe concussions, and spinal cord injuries and save patients from a lifetime of paralysis.

MD
 Lance Grindle ↓ 42 mins ago **Pinned**

Dmso is indeed marvelous. We gave 50 grams of Dmso i.v. daily for five months to a person who severed her lumbar spinal column. No organ damage noted from the Dmso and she can now drive and walk albeit slowly. Thank you IMMENSELY for your article(s) on Dmso.

As many of DMSO's mechanisms directly counteract the processes that trigger dementia, I have received many accounts like these from readers:

"My uncle's wife has dementia and has been unable to speak for over a year. My mom recently visited them and told them about DMSO. He began to give his wife DMSO orally. After two weeks she began to talk again. I read the article and began giving it to my 93 year old mother in her juice every morning at the end of November. She has had some form of dementia for over 15 years. Since taking the DMSO, she no longer suffers with severe sundowners. She is more 'with it' and can communicate and laugh with us. Her personality is back. She is crossing her legs again and lifting her pinky finger while drinking her coffee. It's a lot of little things that make a difference.

She is able to understand when I am asking her to use the bathroom. She is more cognitive and has started coloring in her coloring books again.

I deeply appreciate your posts on DMSO. You helped bring spontaneous interaction back into the life of my father with Alzheimer's."

NUMEROUS STUDIES SUPPORT THESE EXPERIENCES:

- When rats had their carotid arteries surgically modified to reduce the blood going to the brain, DMSO prevented both the neuronal damage and the significant loss of spatial memory and learning that otherwise occurred.
- In a similar study, rats who developed persistent and severe memory impairment from reduced brain blood flow received DMSO and FDP for 7 days, which improved their memory by 54%, nearly reaching the cognitive function rats whose blood flow was never cut off.
- In rats, daily DMSO counteracted memory impairment induced by intracerebroventricular STZ infusions, while in a similar study, DMSO and Ginkgo biloba improved learning and memory in rats given Alzheimer's disease.
- Drinking minute amounts of DMSO prevented the visual degeneration otherwise seen in rats engineered to have early Alzheimer's disease. In another study of those rats, it protected key brain cells from disappearing and enhanced both their spatial memory and smell (while decreasing their anxiety). Likewise, in rats bred to develop cerebellar disorders, DMSO prevented age-related deterioration of certain cognitive functions (e.g., memory and spatial learning).

THESE RESULTS HAVE ALSO BEEN REPLICATED IN HUMANS:

- In 18 patients with probable Alzheimer's after three months, DMSO greatly improved memory, concentration, and communication, alongside a significant decrease in disorientation in time and space.
- In 104 elderly adults with dementia due to cerebrovascular diseases, concussions, or Parkinson's, DMSO combined with amino acids significantly improved their cognition and motor function.
- In 100 patients with cerebrovascular diseases (many of whom had dementia), DMSO caused almost all to have their cardiovascular parameters improve and:

"Recovery from the general symptoms was positive; there were favorable changes which were reflected in a feeling of well-being, the recovery of agility, changes of mood from depressed to gay, improvement of sleeping, and clearer speech. As regards the 'focal' results, accelerated recovery from hemiplegia and hemiparesia was registered. A speedier recovery of speech in cases of defined or indicated aphasia took place."

CONCLUSION

The Alzheimer's story illustrates how medical science's relentless focus on commercializable products has failed the country. This must be replaced with prioritizing understanding the root causes of the chronic illnesses we face.

Fortunately, now that MAHA can set national health policy and independent media has broken the media's monopoly over the truth due to the lies we saw throughout COVID-19, more and more are stepping outside the medical orthodoxy to pursue therapies that can actually heal them. An opportunity like this has never existed before, and it is critical that each of us brings attention to the need for real medicine before the window to fundamentally change the practice of medicine closes.