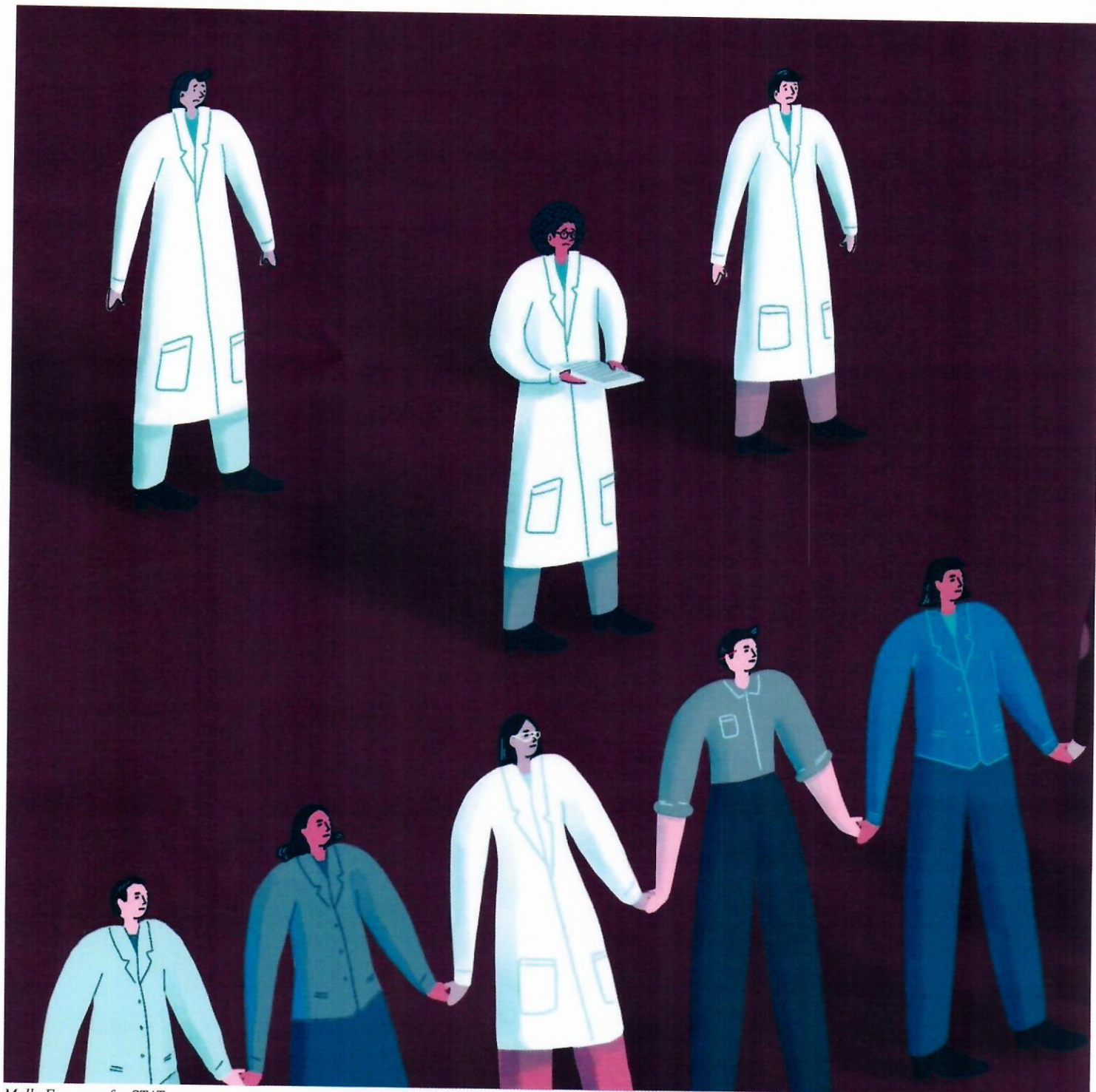


The maddening saga of how an Alzheimer's 'cabal' thwarted progress toward a cure for decades

By [Sharon Begley](#)³ [@sxbegle](#)⁴

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Molly Ferguson for STAT

In the 30 years that biomedical researchers have worked determinedly to find a cure for Alzheimer's disease, their counterparts have developed drugs that helped cut deaths from cardiovascular disease by more than half, and cancer drugs able to eliminate tumors that had been incurable. But for Alzheimer's, not only is there [no cure](#)⁵, there is not even a disease-slowng treatment.

The brain, Alzheimer's researchers patiently explain, is hard — harder than the heart, harder even than cancer. While that may be true, it is increasingly apparent that there is another, more disturbing reason for the tragic lack of progress: The most influential researchers have long believed so dogmatically in one theory of Alzheimer's that they systematically thwarted alternative approaches. Several scientists described those who controlled the Alzheimer's agenda as "a cabal."

In more than two dozen interviews, scientists whose ideas fell outside the dogma recounted how, for decades, believers in the dominant hypothesis suppressed research on alternative ideas: They influenced what studies got published in top journals, which scientists got funded, who got tenure, and who got speaking slots at reputation-buffing scientific conferences.

This stifling of competing ideas, say a growing number of scholars, is a big reason why there is no treatment for Alzheimer's. (The four approved drugs have no effect on the disease, providing only a temporary memory boost.)

The scientists described the frustrating, even career-ending, obstacles that they confronted in pursuing their research. A top journal told one that it would not publish her paper because others hadn't. Another got whispered advice to at least pretend that the research for which she was seeking funding was related to the leading idea — that a protein fragment called beta-amyloid accumulates in the brain, creating neuron-killing clumps that are both the cause of Alzheimer's and the key to treating it. Others could not get speaking slots at important meetings, a key showcase for research results. Several who tried to start companies to develop Alzheimer's cures were told again and again by venture capital firms and major biopharma companies that they would back only an amyloid approach.

"The amyloid hypothesis has been one of the most tragic stories [in] disease research," said neurobiologist Rachael Neve of Massachusetts General Hospital.

[Related:](#) ⁶

Watch: How did the amyloid hypothesis go from promising to perilous in the search for Alzheimer's treatments? ⁶

Despite being described as a "cabal," the amyloid camp was neither organized nor nefarious. Those who championed the amyloid hypothesis truly believed it, and thought that focusing money and attention on it rather than competing ideas was the surest way to an effective drug.

It has not worked out that way. Research focused on amyloid, and the development and testing of experimental drugs targeting it, have sucked up billions of dollars in government, foundation, and pharma funding with nothing to show for it. While targeting amyloid may or may not be necessary to treat Alzheimer's, it is not sufficient, and the additional steps almost certainly include those that were ignored, even censored. Probably the most shattering turn came in March, when Biogen [halted](#)⁷ the study of what proponents called the most promising Alzheimer's drug in years — an amyloid-targeting antibody.

For all her regrets about the amyloid hegemony, Neve is an unlikely critic: She co-led the 1987 [discovery](#)⁸ of mutations in a gene called APP that increases amyloid levels and causes Alzheimer's in middle age, supporting

the then-emerging orthodoxy. Yet she believes that one reason Alzheimer's remains incurable and untreatable is that the amyloid camp "dominated the field," she said. Its followers were influential "to the extent that they persuaded the National Institute of Neurological Disorders and Stroke [part of the National Institutes of Health] that it was a waste of money to fund any Alzheimer's-related grants that didn't center around amyloid."

To be sure, NIH did fund some Alzheimer's research that did not focus on amyloid. In a sea of amyloid-focused grants, there are tiny islands of research on oxidative stress, neuroinflammation, and, especially, a protein called tau. But Neve's NINDS program officer, she said, "told me that I should at least collaborate with the amyloid people or I wouldn't get any more NINDS grants." (She hoped to study how neurons die.)

A decade after her APP discovery, a disillusioned Neve left Alzheimer's research, building a distinguished career in gene editing. Today, she said, she is "sick about the millions of people who have needlessly died from" the disease.

Dr. Daniel Alkon, a longtime NIH neuroscientist who started a company to develop an Alzheimer's treatment, is even more emphatic: "If it weren't for the near-total dominance of the idea that amyloid is the only appropriate drug target," he said, "we would be 10 or 15 years ahead of where we are now."

Making it worse is that the empirical support for the amyloid hypothesis has always been shaky. There were numerous red flags over the decades that targeting amyloid might not slow or reverse Alzheimer's. That might be necessary, but it was almost certainly not sufficient.

"Even at the time the amyloid hypothesis emerged, 30 years ago, there was concern about putting all our eggs into one basket, especially the idea that ridding the brain of amyloid would lead to a successful treatment," said neurobiologist Susan Fitzpatrick, president of the James S. McDonnell Foundation. But research pointing out shortcomings of the hypothesis was relegated to second-tier journals, at best, a signal to other scientists and drug companies that the criticisms needn't be taken too seriously.

Zaven Khachaturian spent years at NIH overseeing its early Alzheimer's funding. Amyloid partisans, he said, "came to permeate drug companies, journals, and NIH study sections," the groups of mostly outside academics who decide what research NIH should fund. "Things shifted from a scientific inquiry into an almost religious belief system, where people stopped being skeptical or even questioning."

That would be tragic enough in any area of biomedical research, but it's especially so in Alzheimer's. Today, 5.8 million people in the U.S. have the disease, including 1 in 10 of those 65 and over, estimates the Alzheimer's Association. It is the fifth leading cause of death in that age group. For many patients and their families, that's a small mercy: Robbed of their memories, unable to recognize those they loved, often suffering from psychosis, they lose their mind and their identity long before their life.

Scientists closely associated with the amyloid model argue that if alternative ideas received little funding support, it was because NIH's Alzheimer's budget was woefully insufficient (\$425 million in 2012, \$2.4 billion in 2019). "It's our responsibility to choose studies that are the most promising, and I think we have been doing that," said Dr. Paul Aisen of the University of Southern California, a leading amyloid proponent. "I would reject the idea that we would have been further along if there had been more openness to other ideas."

Dr. Dennis Selkoe of Harvard Medical School, also a prominent amyloid researcher, isn't so sure. He, too, says low NIH funding for Alzheimer's from the 1980s through the 2000s is to blame for alternative ideas languishing. "But society has the right to ask, why haven't we made more progress?" he said. "I have no doubt that if we had done broader research we would be more advanced now."

“I don’t think there was a purposeful attempt to scuttle other approaches,” Selkoe added. Or as Aisen put it last week on the sidelines of the Aspen Ideas Festival, “I don’t think I’m part of a cabal.”



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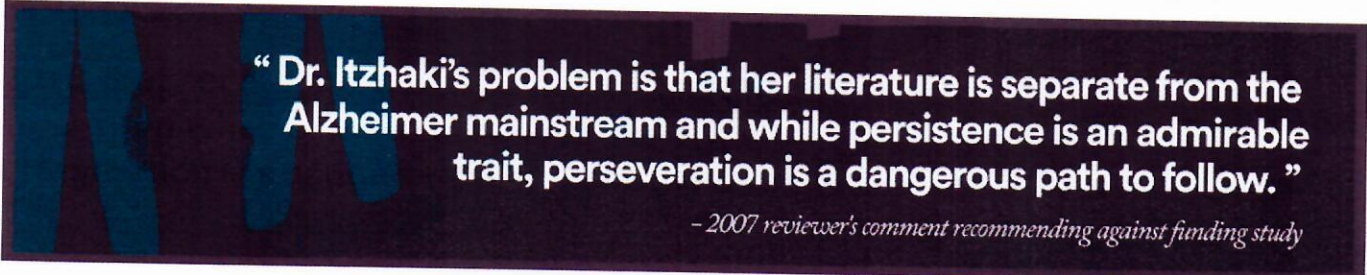
Ruth Itzhaki *Courtesy University of Manchester*

Ruth Itzhaki often felt like she was in a house of mirrors. A molecular neurobiologist at England’s University of Manchester, in 1991 she discovered pathogens — herpes simplex virus type 1 — in the brains of elderly people who had died with Alzheimer’s and carried the most common gene for the disease. It was the first indication that infectious agents might play a role in Alzheimer’s, raising the possibility that eliminating them (and the resulting immune response, including inflammation) might stop or even reverse it.

Nearly half a dozen journals rejected Itzhaki’s [paper](#)¹² before it was accepted by the Journal of Medical Virology, not a bad journal but not a leading one. A frequent reason top journals declined to publish her papers, as they did those of other amyloid skeptics, was previous rejections. As one peer reviewer wrote about a funding proposal Itzhaki submitted in 2010, “very few [of your] papers have appeared in the most highly regarded journals.”

“And here I thought research should be judged on its own merits,” Itzhaki said.

Like other doubters, Itzhaki wasn’t dismissing the idea that amyloid has a role in Alzheimer’s; she was questioning whether it was the cause, and therefore a good drug target. She saw it as a consequence of the true cause — making amyloid the gravestones of brain neurons killed by something else and not their assassins. In that case, targeting amyloid would no more revive dead neurons than removing headstones would resurrect bodies in a cemetery.



“Dr. Itzhaki’s problem is that her literature is separate from the Alzheimer mainstream and while persistence is an admirable trait, perseveration is a dangerous path to follow.”

– 2007 reviewer’s comment recommending against funding study

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Funders did not beat a path to her laboratory door. When Itzhaki was an advisor on a proposed clinical trial of an antiviral drug for Alzheimer’s, one scientist who assessed it for a private foundation wrote, “The novelty of this approach appears to be quite lacking,” according to documents she shared with STAT. To which Itzhaki wondered, the thousands of clinical trials based on eliminating amyloid, which keep getting funded, are novel?

The Alzheimer’s Association awards its Zenith Fellowships to scientists “on the cutting edge” of research, acknowledging that their studies “may not conform to current conventional scientific wisdom or may challenge the prevailing orthodoxy.” Itzhaki thought that described her work to a T, so in 2004 she applied for funding for a study on the role of herpes simplex virus in Alzheimer’s.

The experience was that of an impala asking a pride of lions for support. One of the four reviewers gave her scores of “poor” (3 on a 10-point scale) on key criteria, arguing that because “there is no conclusive evidence for a major role of this pathogen in Alzheimer’s disease,” the research “will not have an impact on advancing the field of dementia research.” A second reviewer called the role of pathogens in Alzheimer’s “a fringe topic.” Although one gave Itzhaki scores of 10 (“outstanding”), the two dismissive reviews sank her chances.



“Unlikely to be of major significance to AD research.”

– A peer review recommending against publication of Itzhaki’s paper

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It was another demonstration of the power of the amyloid camp. Even when some reviewers were open to alternative ideas, funding and publishing are so competitive, a single slam meant failure.

Itzhaki fared little better getting speaking slots at the field’s most important annual gathering. In 2004, a colleague, neuroscientist George Perry of the University of Texas at San Antonio, talked the organizers of the Alzheimer’s Association International Conference into giving Itzhaki 10 minutes, something, he recalled, that “took a lot of persuading.”

That was the only time she “was allowed to give a talk,” Itzhaki said, “though I applied every year. They never included viruses in the list of meeting topics, and I was allowed only a poster. No one influential ever even came to see it.”

For all the obstacles thrown in her way, in 2009 Itzhaki [showed](#)¹⁵ that herpes simplex virus type 1 is a strong risk factor for Alzheimer’s, and in 2007 that beta-amyloid accumulates in mouse brains that are infected with it. Studying mouse brains and patients’ brains, she found evidence “that this virus is a major cause of amyloid plaques and hence probably a significant [causative] factor in Alzheimer’s disease.”

“Very few [of your] papers have appeared in the most highly regarded journals.”

– 2007 reviewer's comment recommending against funding study

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Last year, two [studies](#)¹⁷ by teams at Mount Sinai and Harvard tied infectious agents to Alzheimer's more strongly than any previous research had, supporting the idea that targeting pathogens and not the response to pathogens (amyloid plaques) might prevent or slow Alzheimer's. In fact, a little-noticed study in Taiwan, also published last year, found that people diagnosed with herpes infections were 2.6 times as likely to develop dementia as herpes-free individuals, but that antiviral drugs cut the risk 90 percent. In 2017 the first [clinical trial](#)¹⁸ investigating antiviral drugs in people with mild to moderate Alzheimer's got underway at Columbia University.

“I just wonder if we'd be farther along if those of us studying the role of pathogens in Alzheimer's had gotten the imprimatur of journals and meetings that the amyloid people did,” Itzhaki said.

In fairness, the true believers had evidence implicating amyloid in Alzheimer's disease.



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Auguste Deter, the first person to be diagnosed with Alzheimer's disease, died in 1906. *Public domain*

In 1906, when German neuropathologist Dr. Alois Alzheimer examined the brain of a 51-year-old woman who had died from what he called presenile dementia, he identified sticky plaques between neurons and tangles of filamentous proteins within them. Plaques in particular, and tangles secondarily, have been the disease's defining characteristics ever since.

Flash forward to 1984: Scientists [determined](#)²⁰ the weight, length, and precise molecular sequence of the protein fragment, or peptide, that makes up the brain plaques. It has come to be known as beta-amyloid.

Hard on the heels of the 1987 discovery by Neve and her colleagues of one amyloid-related gene came several more, all linking mutations in genes called PS1, PS2, and APP to early-onset Alzheimer's, which accounts for about 5% of cases. The 1991 [discovery](#)²¹ of one APP mutation (there are many) was the most-cited paper in all of biomedicine that year. The mutations all raise amyloid levels, and people with them develop Alzheimer's before age 65.

In an era enamored of genes "for" this or that disease, it was an article of faith that genetics would produce cures. "When the genetics discoveries came out, it all pointed to amyloid having a critical role," said UT's Perry, a longtime critic of the amyloid model.

By the mid-1990s, a now-defunct San Francisco biotechnology company, Athena Neurosciences, [created](#)²² the first genetically engineered mice with a mutated, amyloid-producing human gene. The animals' brains filled with amyloid plaques, and their memories were destroyed. The mice were hailed as a "model for testing therapeutic [Alzheimer's] drugs" and rodents that "shook the world": Eliminating the amyloid in their brains at least partly reversed their memory loss and other cognitive deficits.

"If you stopped the amyloid, the mice got better," said Alkon, the former NIH neuroscientist. "Everyone said, this must be the way to treat Alzheimer's."

[Related:](#)²³

[One of the world's best drug hunters went after Alzheimer's. Here's how he lost](#)²³

And so they tried. The biopharma giants Pfizer and Eli Lilly and Merck and Biogen, the now-defunct Elan (which acquired Athena in 1996), and hundreds of academic researchers each crafted a vaccine or antibody or small molecule to prevent the formation of amyloid plaques, to remove soluble (pre-plaque) amyloid, or to destroy the plaques.

"You had a whole industry going after amyloid, hundreds of clinical trials targeting it in different ways," Alkon said. Despite success in millions of mice, "none of it worked in patients."

Scientists who raised doubts about the amyloid model suspected why. Amyloid deposits, they thought, are a response to the true cause of Alzheimer's and therefore a marker of the disease — again, the gravestones of neurons and synapses, not the killers.

The evidence? For one thing, although the brains of elderly Alzheimer's patients had amyloid plaques, so did the brains of people the same age who died with no signs of dementia, a pathologist [discovered](#)²⁴ in 1991. Why didn't amyloid rob them of their memories? For another, mice engineered with human genes for early Alzheimer's developed both amyloid plaques and dementia, but there was no proof that the much more common, late-onset form of Alzheimer's worked the same way. And yes, amyloid plaques destroy synapses (the basis of memory and every other brain function) in mouse brains, but there is no correlation between the degree of cognitive impairment in humans and the amyloid burden in the memory-forming hippocampus or the higher-thought frontal cortex.

"There were so many clues," said neuroscientist Nikolaos Robakis of the Icahn School of Medicine at Mount Sinai, who also [discovered](#)²⁵ a mutation for early-onset Alzheimer's. "Somehow the field believed all the studies supporting it, but not those raising doubts, which were very strong. The many weaknesses in the theory were ignored."

When the 'real thing' becomes a mirage: How patients in Alzheimer's trials are coping with the treatment's failure ²⁶

Reluctance "to admit that maybe we have it wrong," as the McDonnell Foundation's Fitzpatrick put it, made it difficult for researchers who, mindful of those red flags, wanted to explore ways to understand, prevent, and treat Alzheimer's that didn't revolve around amyloid. Alternative ideas ranged from infectious microbes and inflammation as causes, to blood flow and synapse restoration as treatments. Today, therapies based on all of those ideas, and more, are being tested in people, evidence of their promise.

But many of the scientists with alternative ideas "became roadkill on the highway to nowhere," Perry said, their careers stalled or worse. Perry fared better, rising to become dean of the College of Sciences at UT San Antonio, but saw many of his NIH grant proposals rejected by NIH study sections. A friend on one "told me my NIH grant was sacked due to my anti-amyloid stance," Perry said. "The sacker came up to me at a meeting a few weeks later and asked why I wrote articles questioning amyloid."

Not every young scientist has the stomach for the Alzheimer's wars. Neurophysiologist Malú Tansey of Emory University has focused on neuroinflammation for nearly two decades, convinced that this hyped-up immune response kills synapses and neurons and explains many other discoveries about Alzheimer's brains.

If that's right, "it suggests a role for non-amyloid drug targets," she said.

But this was no subject for a young scientist trying to gain a foothold in the field. In her first university talk as a junior faculty member, in 2002, Tansey described her research. "A senior faculty member stood up and said neuroinflammation has nothing to do with Alzheimer's," she recalled. "It was intimidating."

She has since focused on Parkinson's disease, but still has two grants to study neuroinflammation in Alzheimer's.

Alzheimer's and amyloid: 'It's time to do something else' ²⁷

Similarly, at Mount Sinai, one of Robakis's postdoctoral fellows witnessed Robakis's NIH grant proposals to study things like neuronal survival (without a role for amyloid) getting such a low score that the study section didn't even discuss it.

"He left Alzheimer's to study something safer, the blood-brain barrier," Robakis said. "That happened all the time."

For young academics, biotech executive Dr. Raymond Tesi said, "it's difficult to break into a field with so many strong voices supporting a single target. Alzheimer's has egos and superstars and big personas unlike anything I've seen elsewhere."

Tesi was persuaded enough by the neuroinflammation explanation of Alzheimer's that he vowed to found a company to develop a treatment based on it. Starting nearly a decade ago, he said, "I talked to everyone: Lilly and Novartis, venture capitalists, arguing that neuroinflammation was the core pathology of Alzheimer's. We couldn't get past the front door. If you weren't doing amyloid, you could barely get a meeting."

He called it an example of "the groupthink that occurs in biopharma. Every company goes to key opinion leaders in academia and asks, 'What should we do?'" Since amyloid had a lock on such leaders, from Harvard to the

University of Southern California and in between, the answer was always the same: eliminate amyloid.

In 2015, Tesi and others eventually scraped together enough support, including a \$1 million grant from the Alzheimer's Association, to found INmune Bio and develop a compound to quell neuroinflammation. That year, he estimated, 90% of NIH, industry, and private foundation spending on Alzheimer's research and drug development was premised on the idea that eliminating or reducing amyloid was the route to success.

INmune launched a Phase 1 clinical trial this month of its anti-inflammatory agent XPro1595. It may well fail, but INmune is targeting people with mild to moderate Alzheimer's, whom virtually every big biopharma has given up on. If an anti-inflammatory helps such patients, that could have been discovered millions of ravaged minds ago. "We probably lost five years," said Tesi, the company's chief executive.

[Related:](#) ²⁸

That pathetic Alzheimer's pipeline? It's even worse than you think ²⁸

Alkon said he lost at least that many. During three decades at NIH, he did groundbreaking work on the cellular and molecular basis of memory, rose to the position of lab director, published hundreds of scientific papers ... and never cured anyone of anything. But in 1999, he felt, he just might.

He therefore resigned from NIH to head a cure-focused, neurological institute founded by the Rockefeller family, studying an odd compound called bryostatin-1 (made by sea mosses, of all things) and its remarkable ability to increase synapse-boosting molecules. Hoping to turn bryostatin into an Alzheimer's drug, Alkon co-founded Neurotrope BioScience in 2012, and soon tried to interest a leading pharmaceutical company in collaborating to develop bryostatin faster than a little startup could alone.

In a meeting at the drug maker's headquarters, Alkon ran through the data. In human neurons growing in lab dishes, bryostatin provided protection against amyloid and preserved synapses. In mice, it improved learning and memory even when amyloid levels remained high. It not only preserved synapses, it also sopped up amyloid molecules, the protein fragments whose clumping into sticky plaques between brain neurons is considered the hallmark of the disease. And in earlier studies, when tested against cancer, bryostatin was extremely safe.

Cool, great, the executives said. There was just one thing.

"The only way they would consider bryostatin was if we could show it was superb at stopping amyloid," Alkon recalled. "They were just interested in that, not its effect on synapses," whose loss causes the memory and other cognitive decline of Alzheimer's patients and which Alkon believes is the key to treating the disease.

The company (which he declined to name so as not to jeopardize future collaborations) passed. It had better ways to eliminate amyloid, it decided, and didn't much care about restoring synapses. The next companies Alkon approached said much the same thing: It was all amyloid, all the time. "There weren't many I didn't talk to," he said.

No one claims that if deep-pocketed pharma had gotten behind bryostatin seven years ago there would be a treatment for Alzheimer's today. The experimental drug produced so-so [results](#)²⁹ in a small clinical trial, according to data published in January in the Journal of Alzheimer's Disease, though it improved cognitive function enough in some patients that Neurotrope is running a larger study, convinced the improvement is real.

The failure of every amyloid-based experimental compound has, finally, triggered soul searching about how it all went so wrong that, in 2019, there is nothing for people who develop Alzheimer's and likely nothing for many

more years. What happened?

“People who said, wait, it might not be so simple as eliminating amyloid, they were not able to go against the wave,” said Mount Sinai’s Robakis. “Critical thinking gave way to dogma. What you believe can be influenced by what is in and what is out. How else do you explain the widespread acceptance of a theory despite its weaknesses?”

Robakis has been continually funded by NIH (including for studies unrelated to Alzheimer’s), so his criticism isn’t sour grapes. Yet he began to feel that the amyloid camp saw him as a “traitor,” the more he pointed out flaws in the theory. “I definitely lost grants,” he said. “If amyloid wasn’t in the grant proposal, it was an uphill battle. There were very big egos involved and they couldn’t stand to be wrong. It wasn’t science anymore.”

He paused. “We should have known better,” he continued. “You can’t say what would have happened if things had been different, but maybe if there had been more support for alternative ideas, we would be better off [in terms of Alzheimer’s treatments] than we are.”

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It isn’t hard to understand why hundreds of academics lined up behind the amyloid model over the years, Fitzpatrick said. “Once a field commits to a particular hypothesis, the research resources — funding, experimental models, and training — all get in line,” she [wrote](#) ³² in a 2018 analysis. That brings backers of the dominant idea accolades, awards, lucrative consulting deals, and well-paid academic appointments. Admitting doubt, let alone error, would be not only be a blow to the ego but also a threat to livelihood.

Academics who took part in clinical trials of amyloid-premised drugs greeted each failure with “some lame excuse,” said Jack de la Torre of the University of Texas, Austin, who studies the idea that reduced blood flow within the brain is a key contributor to Alzheimer’s. “This way, the money from big pharma would mercifully not dry up.”

Harder to understand is why drug companies embraced the dogma even after the repeated failures of experimental drugs based on it, which has cost them billions of dollars. A longtime pharma scientist who recently joined a biotech startup offered one explanation: If company executives greenlight the development of an amyloid drug and it fails, they don’t lose their jobs because “the smartest guys in the room, meaning academia, said this was the way to go,” he said. “But if you greenlighted a different kind of Alzheimer’s therapy, and it failed, good luck with your career.”

While there is growing recognition that there could have been more progress if non-amyloid ideas had received greater support early on, those alternatives are now being explored in both basic research and clinical trials. The NIH, for instance, is funding the 130-patient study of whether an antiviral can help Alzheimer’s patients; Columbia’s Dr. Davangere Devanand, who is leading it, expects results in three years.

The increasing diversity of approaches might finally bring help to the millions of people already suffering from Alzheimer’s and the tens of millions more who will develop it. “The tragedy is,” said UT’s Perry, “we could have gotten to this point many, many years sooner.”

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