PICTURING AN END TO ALZHEIMER'S

After decades of disappointments, a potential breakthrough treatment for Alzheimer's disease is sparking a new symptom in researchers, clinicians, caregivers and families: hope. For the more than 120,000 Wisconsinites living with Alzheimer's and dementia—and an aging baby boomer population projected to double that number in the next 25 years—promising new drugs can't come soon enough.

BY JEFF OLOIZIA

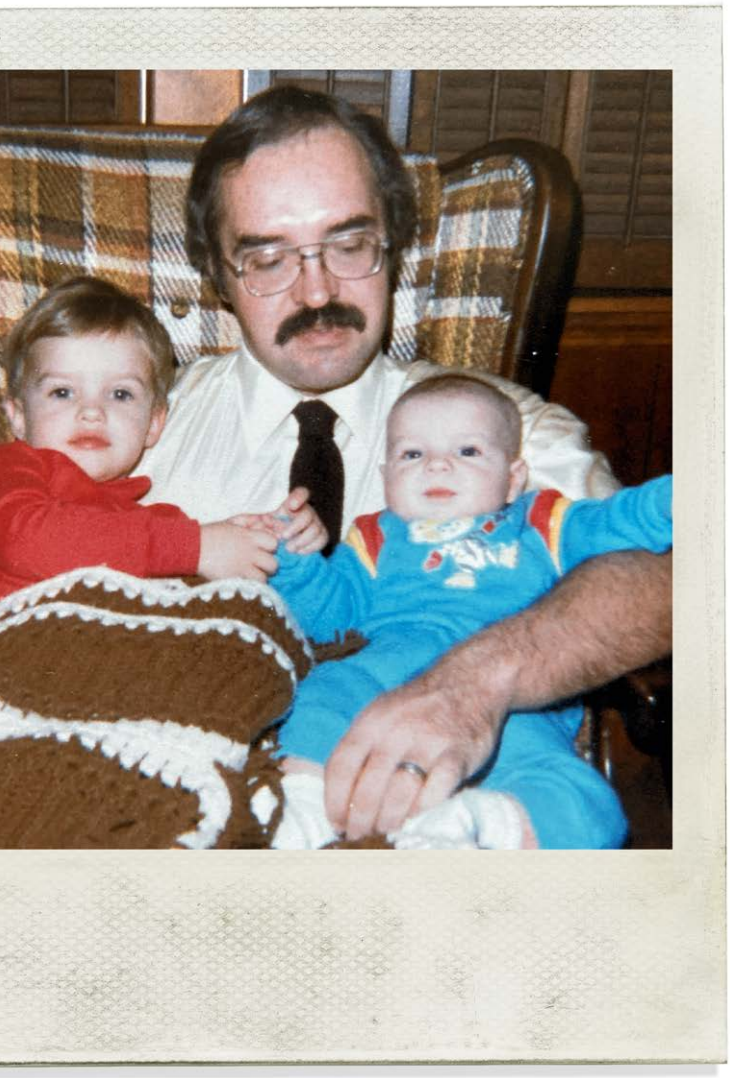
ILLUSTRATION BY NICOLE XU

In March of this year, my father died at age 79 after a series of prolonged hospital stays. As these things go, it was a fairly straightforward affair: He had long suffered from heart and kidney problems, and for the past decade had been in a slow decline due to dementia. By the end, there was little mystery about what had taken his life.

PHOTO COURTESY OF JEFF OLOIZIA

YET WHEN THE FUNERAL HOME DIRECTOR, an exceedingly chatty man named Bob, asked us about the specific nature of my father's dementia, we—my mother, brother and I—fumbled awkwardly. It wasn't that we hadn't thought to pinpoint the cause of his decline; it was simply that, by the time his dementia had progressed, a formal diagnosis seemed to offer little help. Besides, brain ailments can be notoriously tricky to diagnose. Without an autopsy, it's hard to know what's going on in there.

What we did have, however, were the results of an MRI of my father's brain, taken several years before his death and excavated from my mother's files. The first part of the document's title—*MR MEMORY LOSS*—made my father sound like the protagonist in a children's movie about amnesia. *Bilateral hippocampal volumes are decreased and are below the 15th percentile on the normative data*, it began. I tried to recall my ninth grade biology lessons (what was the hippocampus again?) before giving up and tucking the document back into its folder.



DAD AND HIS BOYS: The author as a baby (*on right*) with his brother, Brian (*on left*), and their father, Randy Oloizia, who died in 2024 after a long decline due to dementia.

I can't remember how we responded to Bob; most likely, he let his question go unanswered. Still, in the ensuing days and weeks, it ate at me. So, a few weeks after the funeral, I emailed a respected geriatrician I knew at the University of Wisconsin–Madison, Dr. Nathaniel Chin, and asked if he'd review the MRI.

Chin's reply all but confirmed what we had already suspected: Like millions of other Americans, my father had Alzheimer's disease, the most prevalent form of dementia.

Today, nearly 7 million American adults grapple with Alzheimer's or related dementias. In the next 25 years, driven by an aging baby boomer population, that number is expected to almost double. Alzheimer's currently ranks as the seventh-leading cause of death in the United States. On top of that, it exacts a significant financial toll, devouring around $231 billion annually from Medicare and Medicaid alone—a sum that could exceed $1 trillion by 2050. Some in the field have suggested that, without interventions, it could bankrupt our health care system.

For our family, a diagnosis offered only a latent peace of mind. Then again, even if it had come five years earlier, it might not have been much help. Since its discovery over a century ago, Alzheimer's disease has essentially been a death sentence. Unlike with cancer, heart disease and other major afflictions, there were, until now, no effective means to prevent, slow down or halt its advancement. Diagnosis meant preparing for the worst, with treatments offering little more than palliative care. Except now—too late for my father but perhaps just in time for millions of others—a new class of drugs is calling that bleak prognosis into question.

NEW TREATMENT A GAME CHANGER?

One such possible beneficiary of these new potential therapies is Marilyn Krause. Every month for the past two and a half years, Krause has driven 55 miles from her home in Delafield to UW Health University Hospital in Madison. There, she is fitted with an IV and hooked up to a single-use clear bag containing one of two fluids: a recently approved Alzheimer's drug called lecanemab or an inactive saline solution designed to resemble the drug. If she receives the placebo, she will get up after an hour and leave the hospital none the wiser. But if she receives lecanemab, well, that's where things get interesting.

Krause is among more than 1,000 participants worldwide in the AHEAD study, a first-of-its-kind investigation into whether lecanemab infusions can prevent future memory loss and dementia caused by Alzheimer's disease. Approved by the Food and Drug Administration in 2023, lecanemab works by clearing amyloid, a sticky protein that clumps into plaques in the brains of those with Alzheimer's. While lecanemab doesn't stop, reverse or cure Alzheimer's disease, it does appear to slow cognitive decline, affording those with the condition months—and potentially more—of quality time with their loved ones.

Krause does not have Alzheimer's or any other cognitive impairment—at least not yet. But as an otherwise healthy 72-year-old with an intermediate level of amyloid and a father who died from the disease, she is an ideal candidate for AHEAD's tailored dosing approach to warding off future memory loss.

"My father was the youngest of 10 brothers, and almost all of them had a heart condition," Krause says. "My father was diagnosed with a heart condition on a fluke when he was in his early 40s and was on medication for the rest of his life. He was 88 when he died. He lived so much longer than anybody else, we don't know if anybody else in the family would have developed [Alzheimer's]."

Krause exudes vitality, with a journalist's curiosity and talent for probing questions. She first read about lecanemab and AHEAD in the Milwaukee Journal Sentinel, where she worked—first as a reporter and later as an editor—for 27 years. The tipping point came when her husband was diagnosed with lung cancer in November 2020. When he died just seven months later, she resolved to take charge of her long-term health.

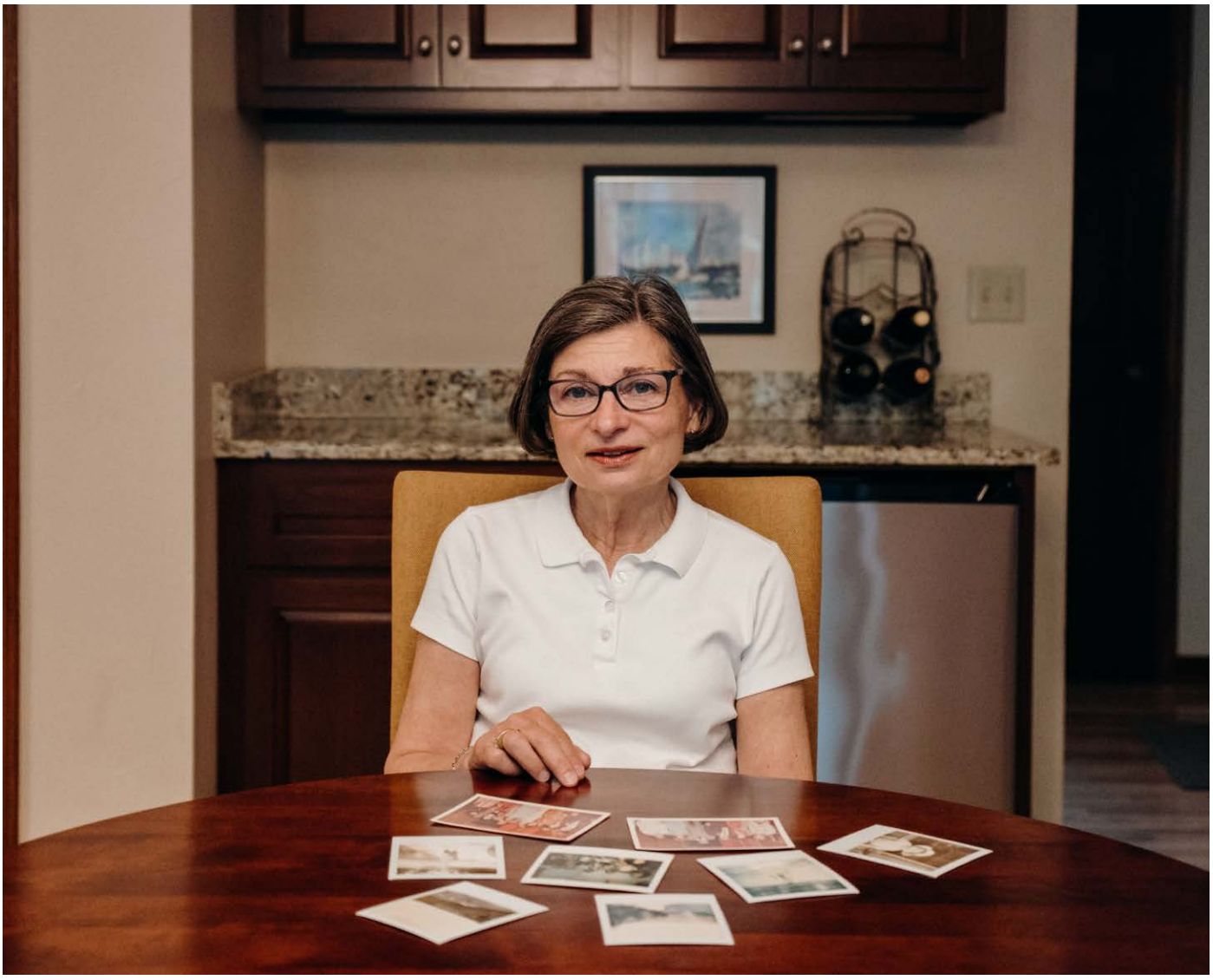


PHOTO BY NICK GARCIA

"If you look at cancer research over the last 50 years, there have been some strides made but there's still a lot that they can't really do much for," she says. "Right now, you have to say that with Alzheimer's, they really can't do much more than they were doing 20 or 30 years ago. This will change that dramatically if it pans out, but it's still too early to know."

Cynthia Carlsson, who oversees the AHEAD study at UW–Madison, calls lecanemab "a game changer." An estimated 1.5 million Americans are believed to be in the early stages of Alzheimer's, diagnosed with either mild cognitive impairment (MCI) or early-stage dementia. Given that amyloid accumulation in the brain precedes symptoms by up to two decades, early intervention is critical. Thus, AHEAD enrolls participants as young as 55, subjecting them to monthly lecanemab infusions and comprehensive cognitive evaluations over a four-year period.

As the oldest of eight children, Krause sees participation in research as "a way to give back"—to potentially protect her family while pushing the boundaries of medical knowledge forward. Yet beyond that altruistic motivation lies a deeply personal incentive: a 50-50 chance of being among the first to benefit from a therapy that could profoundly transform her brain health and memory.

While lecanemab doesn't stop, reverse or cure Alzheimer's disease, it does appear to slow cognitive decline, affording those with the condition months ... of quality time with their loved ones.

"Nobody's doing it for the free lunch," Krause says.

A TROUBLED HISTORY

Just three years ago, the Alzheimer's field was in disarray. Lecanemab's predecessor, an antibody drug called aducanumab, had been released in 2021 to great fanfare. As the first anti-amyloid therapy to receive accelerated FDA approval, it was hailed as a potential breakthrough. But it was also shrouded in controversy.

Both aducanumab and lecanemab are rooted in the "amyloid hypothesis," the prevailing idea that Alzheimer's is caused by the accumulation of amyloid proteins in the brain. This accumulation is believed to trigger the tangling of another protein, tau, which in turn disrupts neuronal function, resulting in the telltale cognitive decline and neurological symptoms experienced by Alzheimer's patients. If you've ever faced the relentless questioning that is so characteristic of Alzheimer's, it's likely because the other person's hippocampus—the brain's seahorse-shaped memory center, which processes short-term memories into long-term ones—has suffered from years of neurodegeneration.

Although this hypothesis shows promise, confirming it has proven challenging. While anti-amyloid therapies command the lion's share of Alzheimer's research funding, some experts have argued that the dogmatic pursuit of such therapies has sidelined other potential approaches, including those targeting inflammation, vascular disease and even viral infections.

By 2020, researchers and clinicians alike had grown frustrated, and who could blame them?

TESTING IT OUT: Inspired by her late father, Marilyn Krause (*pictured left*) drives to Madison every month to participate in a clinical trial for lecanemab.

For almost 20 years, the FDA hadn't approved a single new drug for Alzheimer's. In 2015, Bruno Dubois, an internationally renowned Alzheimer's researcher, was caught on a hot mic disparaging one of the few approved classes of drugs, brain-boosting medications such as donepezil, as "useless." ("Not only do I know that they're useless," he told a French radio journalist, "but I'm obliged to say that they are a little useful because otherwise it loses the trust of the patients who take them.") Closer to home, UW–Madison's Carlsson presented on available treatments only to realize her slide deck hadn't required updating in years.

"When I started, I thought there would be more treatments by now," Carlsson says.

Conferences erupted into heated debates about the relevance of amyloid, reflecting a sense of crisis within the research community. Henrik Zetterberg, a researcher and UW–Madison visiting professor, described the atmosphere at these events as somber, almost funereal at times.

Aducanumab's approval, then, should have been cause for celebration—confirmation of the amyloid hypothesis, once and for all. Instead, it sparked immediate outcry.

Trials for the drug were marred by conflicting data. A congressional inquiry found the FDA's approval process for aducanumab to be "rife with irregularities" and criticized its maker, Biogen, for setting an "unjustifiably high price." Three members of the FDA's own advisory committee resigned in protest after claiming the agency had dismissed their advice. Finally, in January 2024, Biogen made the decision to discontinue the drug entirely.

"It's more of a disappointment, like, why did all these things have to happen on the first shot?" says Chin, who serves as medical director and clinical core co-leader for the Wisconsin Alzheimer's Disease Research Center. "The first impression matters so much. And they ruined it. They ruined the first impression of monoclonal therapies."

Yet from the ashes of that failure rose lecanemab.

NOT A MIRACLE DRUG

From the outset, lecanemab's development process was much improved. Studies were bigger, longer and more diverse. In granting lecanemab full approval, the FDA employed stricter and more cautious language compared to its initial approach with aducanumab, which was inexplicably approved for any person with Alzheimer's, despite having been tested only in patients in the early, "mild" stage of the disease. Perhaps most importantly, lecanemab showed better efficacy compared to its predecessor, if marginally so.

But lecanemab is not without its own controversies. Its effectiveness is limited to patients with mild Alzheimer's. There are still those who feel its impacts aren't clinically meaningful, and its cost—$26,500 annually before Medicare—remains a concern. Additionally, approximately 20% of patients experience side effects, including brain swelling and bleeding. Currently, lecanemab can only be administered in facilities equipped with

EARLY-STAGE PROMISE: Leqembi (*pictured below*) is the brand name for a new drug called lecanemab. UW–Madison's Cynthia Carlsson (*pictured left*) calls it a "game changer."

COURTESY OF EISAI INC., (INSET) CLINT THAYER/ UW DEPT. OF MEDICINE

MRI machines, such as UW Hospital, to monitor these potential complications, making it inaccessible to those in rural areas.

Another issue: convincing patients to try it. As the drug is gradually introduced into clinical settings, those with MCI and early-stage dementia must weigh the benefits against the risks. This decision is especially challenging for communities of color, who face a higher risk of Alzheimer's but may be wary of experimental therapies.

"There's a concern among many communities that the drugs have not been widely tested on people of color," says Maria Mora Pinzon, UW–Madison assistant professor of geriatrics and gerontology. "People have asked, 'Am I a guinea pig? Has this been tested in Latinos? Has this been tested in Black people?'" (In clinical trials of lecanemab, about 25% of U.S. participants were Black or Hispanic.)

Krause, too, admits to worrying about side effects. However, Chin cautions that to dwell on the negatives is to miss the forest for the trees. After decades of disappointments, clinicians finally have a therapy proven to clear amyloid.

"I do know from sitting across from patients more regularly than some of these other [experts], they want something. They want to know that there is some treatment, whether it's today or a year from now, that could potentially help," says Chin. He also rejects the argument that lecanemab's clinical benefits – several extra months of independence, by conservative estimates – are too modest to justify its prescription.

"I don't see oncologists having to justify third-round chemo for a month [of extra life] at best, and we're all OK with that, yet somehow the response to these drugs is, well, is it worth the cost? And to me that feels ageist," Chin says. "It feels like people are saying, 'This group of people, they're not worth it.' And I would say, 'Yes, the 40-year-old with metastatic colon cancer is worth it, but so is my 68-year-old patient with Alzheimer's disease.'"

PROMISE IN BIOMARKERS

Even amid my grief, Chin's assurance that my dad had almost certainly suffered from Alzheimer's brought some relief. However, as spring progressed, another, more self-serving question gnawed at me: What did our dad's Alzheimer's mean for my brother and me? To find out, I arranged to meet Chin at his favorite coffee shop.

Chin is a youthful 40, with a kind face that invites vulnerability. If you or a loved one were going to receive a diagnosis for dementia, you could do much worse than hearing it from him. But there was another reason I'd wanted to discuss this topic with Chin: He'd lost his own father to Alzheimer's.

In 2012, Chin was in the second year of an internal medicine residency in San Diego when he received a call from his mother: His father, Moe, a respected doctor in Watertown, Wisconsin, was starting to decline. All at once, Chin and his family were thrust into the cruel spiral of Alzheimer's disease.

Moe's diagnosis changed Chin. He began prioritizing sleep, aiming for a minimum of eight hours each night. He committed to daily exercise and adopted intermittent fasting – an approach I had also contemplated, which has been shown to lower blood pressure and benefit brain health. Most significantly, Moe's illness inspired Chin to return to UW, where he had received both his undergraduate and medical degrees, and where he would now care for dementia patients.

IT'S PERSONAL: Dr. Nathaniel Chin (*pictured in his home office*) lost his father to Alzheimer's disease. Chin serves as medical director and clinical co-leader for the Wisconsin Alzheimer's Disease Research Center, as well as medical director for the Wisconsin Registry for Alzheimer's Prevention.

As a geriatrician, Chin dedicates most of his time to enhancing the quality of life for his patients aged 65 and older. His research passion, however, lies in the realm of biomarkers – the physical indicators, such as amyloid and tau proteins, that help identify the underlying pathology of Alzheimer's disease.

In addition to his other duties, Chin is the medical director of the Wisconsin Registry for Alzheimer's Prevention, or WRAP, one of the world's largest and longest-running studies of individuals at risk for Alzheimer's disease. Compared to AHEAD, WRAP's more than 1,700 participants are typically younger and more diverse, with varying levels of risk for Alzheimer's. Every two years, these individuals undergo PET scans, spinal taps and cognitive assessments as part of the study protocol. Notably, participation in WRAP is open-ended; many volunteers stay in the study for decades, providing data that helps researchers tease apart normal aging from the earliest signs of Alzheimer's.

PHOTO BY NICK GARCIA

This parallels current thinking on heart disease, diabetes and other age-related chronic ailments: Our genes may load the gun, but lifestyle choices pull the trigger.

One of WRAP's pivotal achievements was the identification of amyloid deposition in participants' brains through PET scans. This breakthrough allowed researchers to establish a timeline of amyloid accumulation, offering insights into disease progression. This seemingly straightforward discovery, Chin told me, was the culmination of decades of rigorous research.

"We finally have it, and now everything is crazy-accelerating to the point where we can say, 'OK, is it predictive? What are the other factors? What happens when you do treat it? How does this relate to the other diseases?'" he says. "So these really exciting questions come up, all because we've been able to identify a protein. To me that still is the landmark change. Lecanemab is merely an offshoot of that."

The disclosure of biomarker results is a touchy subject. For years, research centers routinely declined to return to subjects the results of amyloid PET scans simply because of the lack of available treatments. Certain biomarkers, such as the APOE4 gene – which increases one's risk of developing Alzheimer's by as much as 1,200% – demand even more thoughtful consideration before disclosure. In 2022, this gene was thrust into the spotlight when actor Chris Hemsworth learned he carried it during the filming of his National Geographic docuseries "Limitless with Chris Hemsworth." In response to this revelation, he announced he would step back from acting to focus on his family.

In our conversations, Chin shared his decision to forgo APOE testing, citing the potential emotional burden of a positive result. (I'll likely do the same.)

Still, proponents argue biomarker disclosure is significant because it allows those with a higher risk profile – like Chin, Krause, Hemsworth and yours truly – to make lifestyle changes. A 2022 University of Minnesota study revealed that approximately 41% of Alzheimer's and related dementias were attributable to modifiable risk factors, with high blood pressure, obesity and physical inactivity exerting the greatest impact. This parallels current thinking on heart disease, diabetes and other age-related chronic ailments: Our genes may load the gun, but lifestyle choices pull the trigger.

A ROADMAP FOR PERSONALIZED MEDICINE

Recently, WRAP researchers began testing whether the abnormal proteins in Alzheimer's patients can be detected in a simple blood test. To do this, they are analyzing blood samples collected from WRAP participants dating back to 2011.

Additionally, they are integrating new procedures into study visits to investigate potential comorbidities that could impact cognitive health. These include vascular ultrasounds to evaluate blood vessel integrity, smell tests aimed at identifying proteins linked to Lewy body dementia and Parkinson's disease,

Care for the Caregivers

A former governor helps families impacted by Alzheimer's cope.

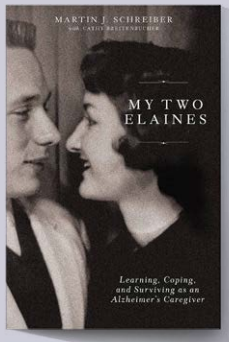
Marty and Elaine Schreiber (*pictured above*) were just beginning to enjoy a well-earned retirement when Elaine was diagnosed with Alzheimer's. Sweethearts at Milwaukee Lutheran High School, the pair were rarely seen apart from each other: first at the University of Wisconsin–Milwaukee, where she helped him pass biology, and later on the campaign trail, where they were regular fixtures. When Marty embarked on a station-wagon tour of Wisconsin during his first bid for lieutenant governor in 1966, Elaine was right there next to him in the passenger seat. And when he became the state's 39th governor in 1977, she served steadfastly as first lady. In later years, she saw him through multiple other campaigns and even a bout with cancer.

Elaine's diagnosis threw Marty into isolation and despair. He developed anxiety and began finding it hard to breathe. His health flagged, to the tune of "hundreds of thousands of dollars" of medical care. To manage the stress, he began to drink in excess, only stopping when his adult children staged an intervention. Slowly, longtime friends began to drift away, unsure of how to handle the complexities of Alzheimer's.

"It's not a chicken casserole disease," Marty says of Alzheimer's. Friends distance themselves "not because they're mean and not because they're not caring, but because they don't know what to do."

Elaine died in 2022. Today, at 85, Marty travels the country, combating what he sees as ignorance around Alzheimer's awareness. Motivated by his own lack of resources while caring for his wife, he wrote the book "My Two Elaines," originally published in 2016, which has helped countless caregivers learn how to cope.

The Schreibers' story isn't unique, but it is instructive. According to the Alzheimer's Association, more than 11 million Americans provide unpaid care for loved ones with dementia, a contribution valued at $350 billion. The total lifetime cost of care for someone with dementia is estimated at almost $400,000, 70% of which is borne by family caregivers in the forms of unpaid care and out-of-pocket expenses. While Medicare offers limited coverage for home services following hospitalization, it largely excludes "custodial care"—a term evoking both janitorial duties and legal guardianship, encompassing not only bathing, dressing and feeding someone with dementia but also keeping them happy and safe.



LOVE STORY: Marty Schreiber tells the story of life with his late wife, Elaine, as they navigated Alzheimer's in the book "My Two Elaines" (*pictured above*), published in 2016.

Moreover, dementia caregivers face heightened risks of anxiety and depression and may see their life spans shortened by as much as four to eight years.

Though the majority of this burden falls on women, Marty emphasizes the importance of all caregivers seeking support from friends and organizations such as the Alzheimer's Association, where he volunteers.

"One of the things I try to point out, particularly to male caregivers, is the fact that it is a matter of courage to ask for help," he says. "That if you ask for help it means you're not giving up."

and a finger prick during blood draws to explore less-invasive methods for studying blood biomarkers. Understanding these comorbidities is pivotal to unraveling the underlying causes of dementia in patients and tailoring effective treatment strategies.

"We're in a remarkable time right now where WRAP is going to be revealing a roadmap for personalized medicine for Alzheimer's disease," says Sterling Johnson, a clinical neuropsychologist who leads the study. Looking ahead, he envisions a future where artificial intelligence can analyze blood samples and other biomarker data to pinpoint candidates for novel therapies and create personalized treatment plans.

Sterling's projection made me think again of my father. When I was 12 and my dad was 52, doctors discovered a significant blockage in one of his arteries, necessitating bypass surgery. At my high school graduation, he pulled me aside and confided that, for many years, his primary goal in life had been to witness my brother and I reach that milestone. Like Krause's father, with medications and lifestyle modifications my dad lived another 20 years. This is the hope for Alzheimer's—that it can evolve from a terminal condition into a manageable, preventable and potentially reversible disease.

"I don't believe that my future will be the same as my father's," Chin told me. "I certainly could still develop Alzheimer's, but as long as that's in 25 to 30 years, I believe that we will have drugs that can stop the progression of the disease right where it is."

THE STOP BUTTON

Imagine Alzheimer's as a cash cube—one of those phone booth-like contraptions, popular at trade fairs and corporate fundraisers, where a participant has 60 seconds to grab as much money as possible as it swirls around them. However, instead of dollar bills, picture Polaroids fluttering about, each encapsulating a cherished memory. At first, your loved one tries their best to catch them. But gradually, robbed of their spatial awareness and problem-solving skills, they struggle and

PHOTOS COURTESY OF MARTY SCHREIBER

ILLUSTRATION BY NICOLE XU

This is the hope for Alzheimer's — that it can evolve from a terminal condition into a manageable, preventable and potentially reversible disease.

grasp at these recollections until they're left clutching only a measly handful. All the while, you, the caregiver, can do little but stand by and watch.

In every Alzheimer's caregiver's journey, there comes a moment when their loved one undergoes a profound shift in identity or personality, marking the onset of a new chapter. In Chin's case, it was watching his father comfortably – though mistakenly – walk off with another family on a trip to Door County. For Krause, that moment arrived when her father, a celebrated chef, could no longer follow a simple recipe. (Eventually, he would fail to recognize his own children.)

But what if such moments could be prevented? What if there was a metaphorical STOP button capable of preserving memories and maintaining the status quo? This is what's at stake as researchers work to uncover new biomarkers and therapies.

Both Chin and Zetterberg liken the current Alzheimer's situation to the HIV crisis of the 1980s. In the early days of HIV treatment, medications were limited in efficacy and often came with serious side effects, much like lecanemab. However, advancements led to the development of highly effective drug cocktails that now enable people with HIV to lead healthy lives while managing the virus. There is hope that Alzheimer's will follow the same trajectory. As clinical studies progress, providing us with a clearer understanding of these drugs' effects and their target populations, lecanemab could be just the first of many breakthroughs.

At the time of publication, researchers at UW were eagerly awaiting FDA Fast Track approval of an injectable form of lecanemab, which would function similarly to insulin for individuals with diabetes. They are also investigating the efficacy of a twice-daily pill designed to prevent nerve endings from being damaged by amyloid, thus staving off cognitive decline. And then there is donanemab, the next in a line of anti-amyloid drugs. It was widely expected to join lecanemab in clinics this year. In March, the FDA announced it was delaying a decision on the drug, surprising many. Perhaps regulators were flummoxed by the trial design, which included patients with more complex medical conditions than those in the lecanemab trials. Or perhaps the agency, feeling burned by the aducanumab debacle, has finally learned its lesson. (On July 2, the FDA approved donanemab at last, giving clinicians another weapon in the fight against Alzheimer's.)

Despite these developments, there is a prevailing sense within the field that we must confront Alzheimer's disease as it is now, lest we leave those currently suffering from its effects behind.

In his book "The Problem of Alzheimer's," Jason Karlawish, co-director of the Penn Memory Center in Philadelphia, writes, "The problem of Alzheimer's disease isn't simply a scientific puzzle to be solved, a menace of 'druggable' pathologies to be conquered. It's the vortex of the scientific, political, cultural, and social problems of aging and disability. It is a humanitarian problem."

Addressing this humanitarian challenge demands that we dismantle stigma and think innovatively. It asks us to reconsider America's pernicious preoccupation with youth, which has allowed diseases such as Alzheimer's to run amok. It will require more – more geriatricians (at least three times the current number), more caregivers, more funding, more empathy. In short, it calls for the creation of a more compassionate world for the millions of individuals already living with this disease.

Wisconsin's first lady, Kathy Evers, has been a leading advocate for this cause, working alongside the Wisconsin Alzheimer's Institute to establish dementia-friendly spaces, including the governor's residence. In Dane County alone, 26 churches have undergone specialized training to become dementia friendly. Internationally, cities such as Bruges, Belgium, have taken this a step further, introducing a sticker depicting a red handkerchief that shopkeepers can display to signal their proficiency in communicating with older adults affected by dementia.

As for Krause, she knows lecanemab alone doesn't ensure a dementia-free future, even if such assurances were possible. Being a study participant hasn't always been easy, either. She briefly considered quitting AHEAD when confronted with the prospect of learning whether she carried the APOE4 gene. (She doesn't.) Worse still are the infusions, which evoke memories of accompanying her husband during his chemotherapy sessions. Nevertheless, she perceives her involvement in research as laying the groundwork for future generations of Alzheimer's survivors.

"In some respect, it is about me," she says. "But it's also an opportunity to contribute to something that could spare others what my family went through long after I'm gone."

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