

*He Was Expected to Get Alzheimer's
25 Years Ago. Why Hasn't He?*



Scientists are searching for the secret in Doug Whitney's biology that has protected him from dementia, hoping it could lead to ways to treat or prevent Alzheimer's for many other people.



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By Pam Belluck Photographs and Video by **M. Scott Brauer**

Reported and photographed in St. Louis, Toronto and Port Orchard, Wash.

Oct. 7, 2025

Before dawn on a March morning, Doug Whitney walked into a medical center 2,000 miles from home, about to transform from a mild-mannered, bespectacled retiree into a superhuman research subject.

First, a doctor inserted a needle into his back to extract cerebral spinal fluid — “liquid gold,” a research nurse called it for the valuable biological information it contains. Then, the nurse took a sample of his skin cells. After that came an injection of a radioactive tracer followed by a brain scan requiring him to lie still for 30 minutes with a thermoplastic mask over his face. Then, another tracer injection and another brain scan.

During his three-day visit to the center, at Washington University School of Medicine in St. Louis, he also had cognitive assessments, neurological evaluations and blood draws that extracted multiple tubes for analysis.

For 14 years now, Mr. Whitney has been the one-person focus of exceptionally detailed scientific investigation, for which he travels periodically to St. Louis from his home in Port Orchard, Wash. It is not because he is ill. It is because he was supposed to be ill.

Mr. Whitney, 76, is a scientific unicorn with potential to provide answers about one of the world's most devastating diseases. He has a rare genetic mutation that essentially guaranteed he would develop Alzheimer's disease in his late 40s or early 50s and would likely die within a decade.

His mother and nine of her 13 siblings developed Alzheimer's and died in the prime of their lives. So did his oldest brother, and other relatives going back generations. It is the largest family in the United States known to have an Alzheimer's-causing mutation.

"Nobody in history had ever dodged that bullet," Mr. Whitney said.

But somehow, he has done just that. Something has shielded him from his genetic destiny, allowing him to escape Alzheimer's for at least 25 years longer than anyone expected.





Mr. Whitney waited as research nurses Georgia Stubbs-Cucchi, left, and Wendy Sigurdson, prepared to take blood, skin and cerebrospinal fluid samples from him; Mr. Whitney's lumbar puncture to extract cerebrospinal fluid; with his wife, Ione, at the Dominantly Inherited Alzheimer Disease Conference in Toronto this summer. Bottom: Ian Willms for The New York Times

Scientists are searching for the recipe for his biological secret sauce. Its discovery could potentially lead to medications or gene therapies to prevent, treat or possibly even cure Alzheimer's, goals that continue to frustrate researchers despite decades of efforts.

"This is an amazing case," said Dr. Kenneth Kosik, a neuroscientist at the University of California, Santa Barbara, who is not part of Mr. Whitney's research team. "There are huge implications in the answers and in posing the questions."

Now, after years of studying Mr. Whitney, researchers are unearthing clues about his magic combination of genes, molecules and environmental influences.

Alzheimer's afflicts about 5 million Americans and about 32 million people worldwide. In most cases, the cause is unknown and symptoms begin after age 65.

About 1 percent of cases, however, are known to be caused by one of three genetic mutations. Inheriting one of those almost always causes early-onset Alzheimer's, which often progresses quickly toward death.

Because genetic early-onset Alzheimer's closely resembles typical late-onset Alzheimer's, studying these families can yield important insights.

"Almost everything we know about Alzheimer's today comes from these rare mutations," Dr. Kosik said.

'I should have got sick'

Mr. Whitney's family has the rarest mutation, Presenilin 2. The mutation has been traced to German immigrants who settled in two villages near the Volga River in Russia in the 18th century. Mutation carriers in Mr. Whitney's family, whose roots

are in the farmlands of Oklahoma, usually began exhibiting memory and thinking problems between ages 44 and 53.

When Mr. Whitney turned 50, his wife, Ione, said, she and their two children began watching for signs.

She had been bracing herself since the early 1970s, when Mr. Whitney's mother suddenly forgot how to make her beloved pumpkin pie for Thanksgiving, and the couple learned that doctors had determined that the Alzheimer's afflicting his relatives was hereditary. They were expecting their first child at the time.

"I was just so angry at Doug, at the world, how unfair that was," she recalled.



Doug and Ione Whitney holding hands; sheet music on the Whitneys' piano; a photo of Mr. Whitney, at center, at about age 12, with his brothers Leon, left, and Roger. Roger died of Alzheimer's at age 55.

Mr. Whitney, whose laconic, unruffled temperament reflects his experience serving for two decades in the Navy, responded with characteristic calm. "We've got some choices," his wife recalled him saying. "You can be angry all your life. Do you want to not have this child? Or do we want to enjoy life and have a family?"

Her anger faded, and she embraced his outlook. “Doug’s attitude was, ‘We don’t have to worry about this till there’s something to worry about,’” she said.

When he reached 55, the age his mother and brother died, his family’s antennae became even more attuned.

“‘How’s Dad doing?’” their son and daughter asked whenever they called home.

“‘I don’t see anything,’” Mrs. Whitney replied.

“‘When he turned 60,’” she recalled, “‘it was like, ‘We are good.’”

Then, a cousin, Gary Reiswig, contacted them saying that he was writing a book about the family and that researchers were seeking more members of families with early-onset Alzheimer’s mutations to study.

Mr. Whitney agreed to participate and to undergo genetic testing, assuming that he did not have the mutation. But on his 62nd birthday, he learned that he did.

“‘I was speechless,’” he said. “‘I mean, I was at least 10 or 12 years past when I should have got sick.’”

Dr. Randall Bateman, a neurologist who directs the Dominantly Inherited Alzheimer Network, known as DIAN, at Washington University, was stunned, too.

“‘We tested him three different times,’” he said. “‘We didn’t believe the results that he was positive.’”

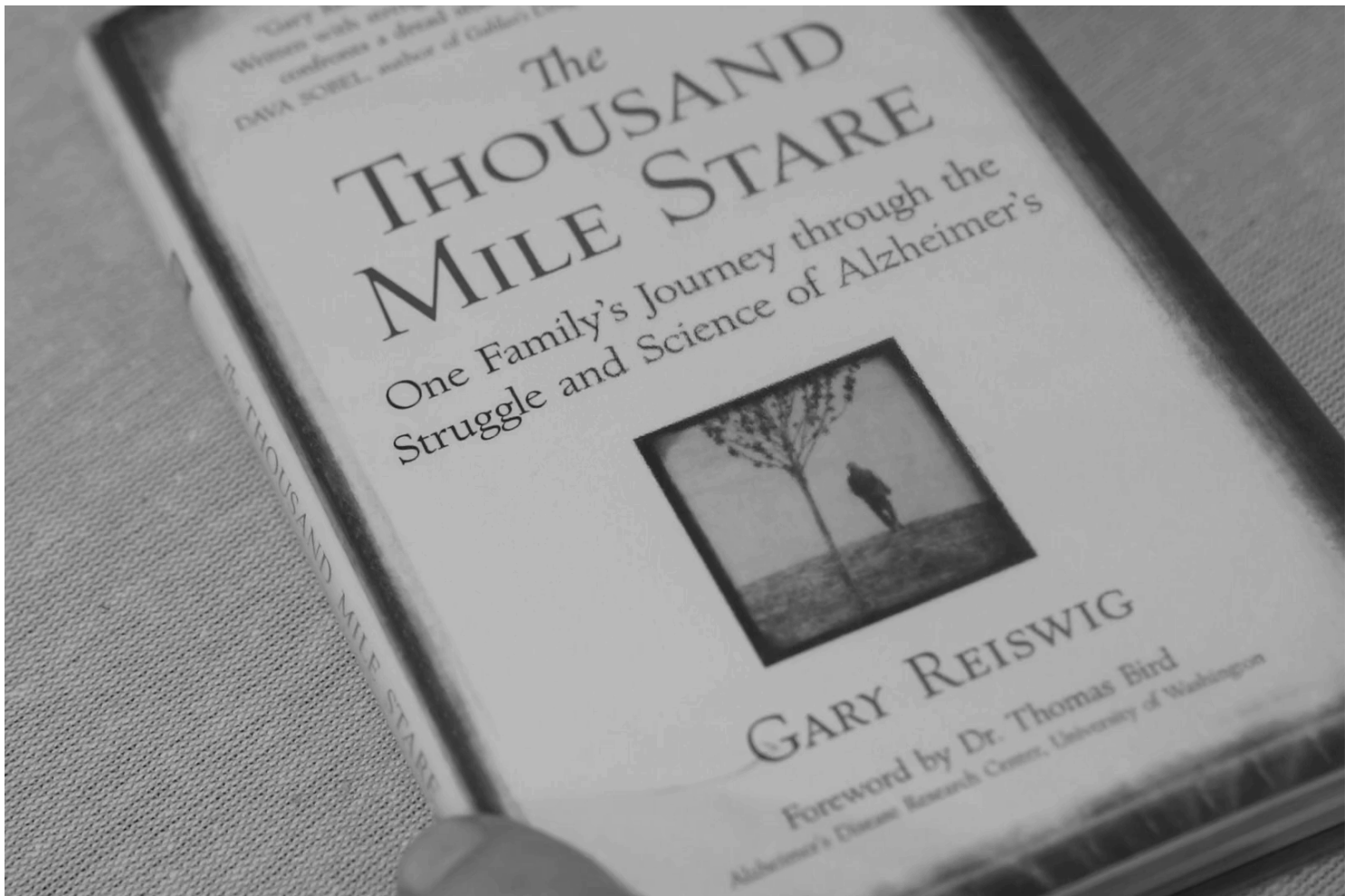
Year after year, researchers became increasingly perplexed that Mr. Whitney remained unimpaired. He continued at his job, organizing submarine maintenance procedures for a military contractor.

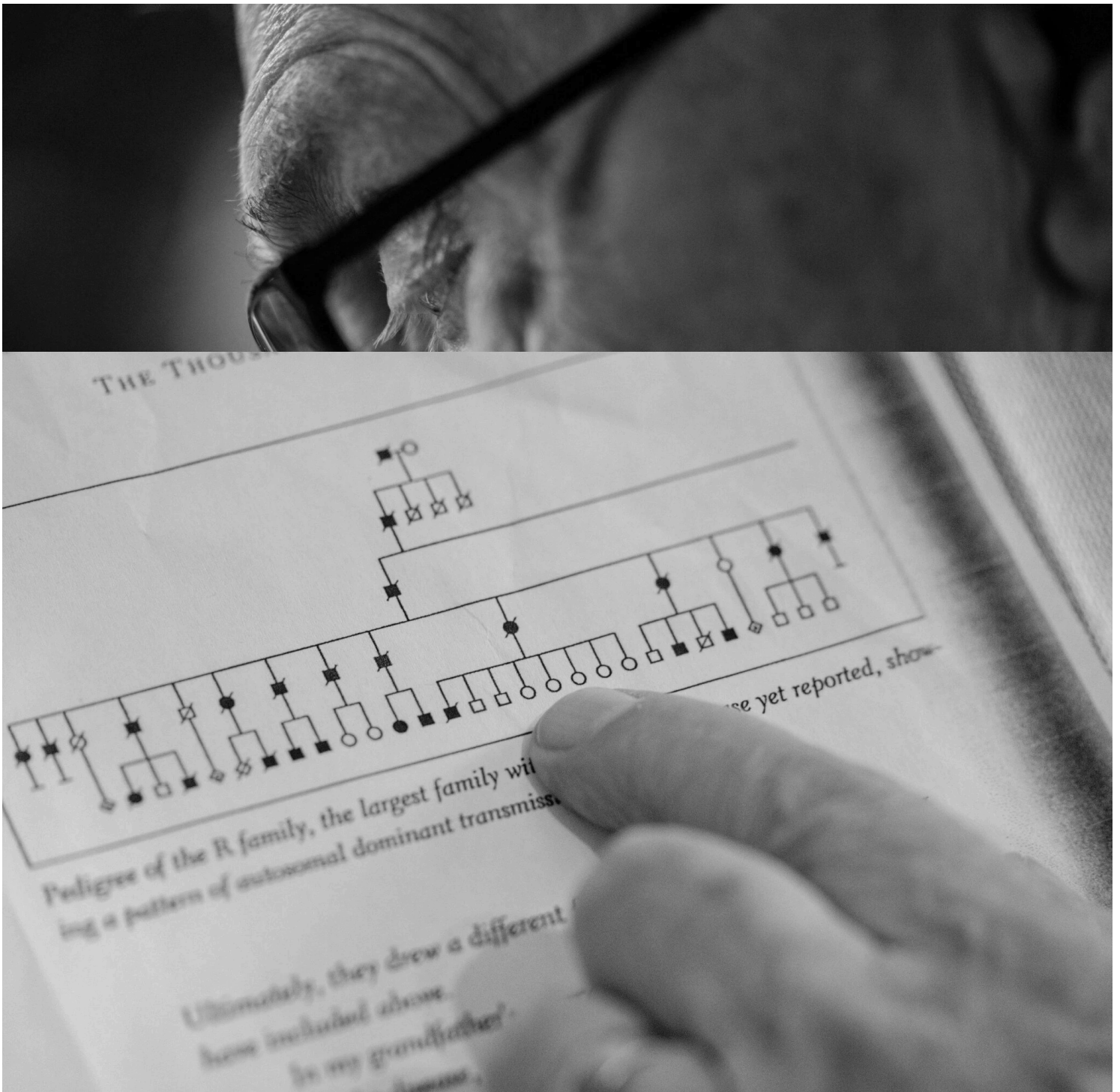
“‘We’re like, ‘What’s going on?’” Dr. Bateman said. “‘He’s still doing OK, still working, still driving around.’”

They set out to determine what was protecting him.

“We came up with a bunch of these crazy ideas,” Dr. Bateman said. “We just started pulling everything off the shelves and anything that we thought we could have him do.” They tested and analyzed. They gave him surveys about his childhood, work history and environmental exposures.

“We just kind of threw the kitchen sink at him,” Dr. Bateman said.





Mr. Whitney flips through "The Thousand Mile Stare: One Family's Journey through the Struggle and Science of Alzheimer's," by his cousin, Gary Reiswig. Mr. Whitney's mother and siblings are in the photo, and the book includes a family tree showing early-onset Alzheimer's inheritance.

Escaping genetic destiny

Researchers call Mr. Whitney an Alzheimer's escapee. Scientists have so far conclusively identified two others in the world who were resilient to the early-onset dementia their mutations should have caused.

Both had another mutation, Presenilin 1, and belonged to a large extended family in Colombia. They remained cognitively unimpaired for at least two decades longer than expected and died in their 70s from other illness.

Alzheimer's is characterized by abnormal accumulations of two proteins in the brain: amyloid, which starts clumping into plaques at least 20 years before symptoms emerge, and tau, which forms tangles after amyloid accumulate. Tau is much more correlated with cognitive decline.

The brains of both Colombian "escapees" were laden with amyloid but had little tau in regions associated with Alzheimer's, said Yakeel Quiroz, a neuropsychologist at Boston University.

She and other scientists believe the Colombian woman was protected by having two copies of an extremely rare genetic variant called the Christchurch mutation. They say the Colombian man's resilience might have come from another variant called RELN-COLBOS.

Not all Alzheimer's researchers are convinced that Christchurch and RELN-COLBOS mutations helped deter Alzheimer's in those cases.





Mr. Whitney undergoing a hand-eye coordination evaluation with Dr. Joy Snider during a neurological exam at Washington University School of Medicine in St. Louis; Dr. Jorge Llibre-Guerra, a neurologist at Washington University, showed the Whitneys results of research into his biology and how it compares to others with the Alzheimer's mutation.

Dr. Michael Greicius, a neurologist at Stanford University School of Medicine who studies the genetics of Alzheimer's, said identifying a mutation that protects one person is difficult without analogous cases for comparison.

“You just can't winnow down the millions of variants that every individual has with one subject and nobody to filter against,” said Dr. Greicius, whose lab is analyzing data on the two Colombians. Nonetheless, he said, “there's incredible potential for these rare, protected individuals to provide critical new insights.”

Mr. Whitney's brain is full of amyloid, probably even more than other mutation carriers in his family because he has lived so long, said Dr. Jorge Llibre-Guerra, a neurologist at Washington University who coauthored a recent study on Mr. Whitney's case. But he has very little tau.

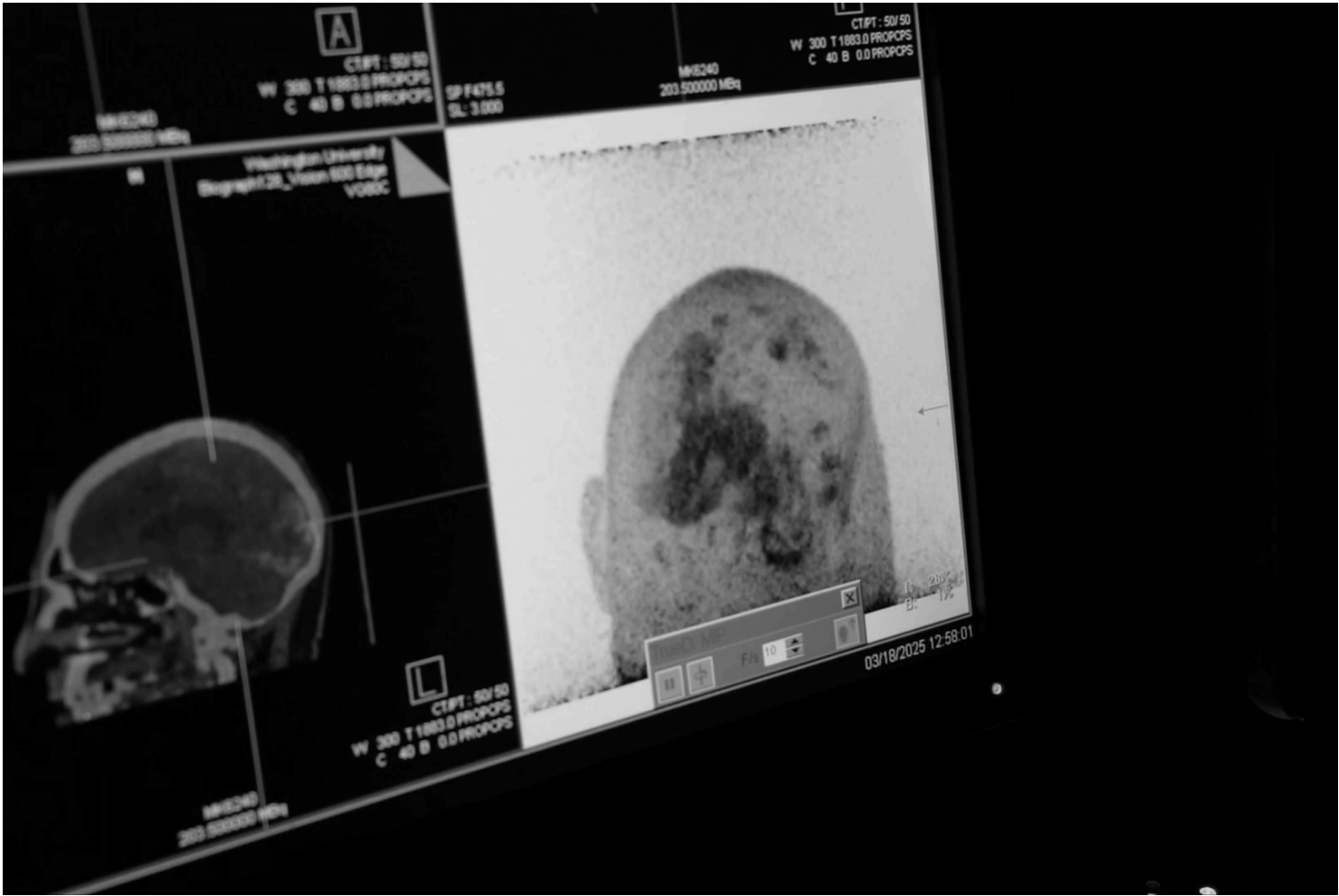
“He’s resistant to tau aggregation and tau spread,” said Dr. Llibre-Guerra, who helps lead DIAN’s clinical trials. “That’s where his resilience is.”

Mr. Whitney has tau accumulation in only one brain region, the left occipital lobe. That area is involved in visual-spatial functions and does not play a major role in Alzheimer’s, Dr. Llibre-Guerra said.

Dr. Quiroz said the Colombian woman’s tau accumulated in the same general area. The cases show that “people can actually have amyloid pathology without having the tau, and that amyloid is not enough to actually create a decline,” she said.

Determining how progression from amyloid buildup to tau accumulation was interrupted could provide a guide for treatment.

“They have now shown the decoupling of amyloid from tau tangles and, when that happens, the sparing of dementia,” said Dr. Kosik, who reviewed the Whitney study for Nature Medicine. “That’s where the science lies.”





A scan of Mr. Whitney's brain — “He’s resistant to tau aggregation and tau spread,” said Dr. Llibre-Guerra, middle; Mr. Whitney's blood plasma.

Clues emerge

Unraveling the riddle of Mr. Whitney’s resilience has revealed an intricate neurological ballet.

There is his D.N.A., which researchers have found includes several gene variants his afflicted relatives don’t have. Most interesting are three mutations possibly involved in neuroinflammation or tau pathology, Dr. Llibre-Guerra said.

There is Mr. Whitney’s immune system. “Your inflammatory response is lower than other mutation carriers,” Dr. Llibre-Guerra told him during the March visit, explaining that his immune system may be shielding him by not overreacting to amyloid.

And there is an especially surprising discovery: Mr. Whitney has an excess of heat shock proteins, which help keep other proteins from folding incorrectly, a defect associated with many neurological disorders.

“The levels you have are significantly higher than what you would expect,” Dr. Llibre-Guerra told him. “It may be that those proteins are preventing the misfolded proteins, especially tau, from spreading throughout the brain.”

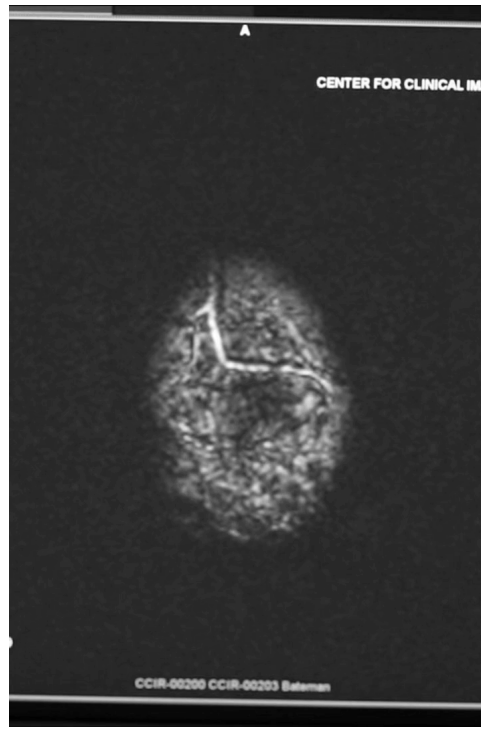
Mr. Whitney’s Navy role — working for about a decade in the engine room of a steam-propelled ship — might have driven his accumulation of heat shock proteins, researchers said.

“The heat down there, you can expect temperatures of 110 degrees for four hours at a time,” Mr. Whitney recalled. “We’d do a lot of sweating.”

It was so hot, he would sometimes need to be hosed down to cool off.

All those factors, possibly with others that remain undiscovered, may be acting in combination to protect him, researchers said.

His case is so complex that Dr. Bateman described his team’s recent published study as “a call to arms” intended “to draw attention from other researchers to say ‘Hey, here’s a really important person, a really important case, and you need to help figure this out.’”



Researchers wonder if Mr. Whitney's accumulation of heat shock proteins, perhaps acquired from years in the Navy working in the engine room of a steam-propelled ship, might be helping to protect him from Alzheimer's.

A generational puzzle

Researchers are also interested in the Whitneys' son, who inherited the mutation from his father.

At 53, Brian Whitney, who works for a flooring store and is a volunteer firefighter in Manson, Wash., remains cognitively healthy. Researchers say he does not have any of the possibly protective gene variants identified in his father. And he didn't have the same yearslong exposure to high heat.

It is possible he has benefited from anti-amyloid drugs he received in a clinical trial led by DIAN, Dr. Bateman said. Researchers recently reported that out of 73 trial participants, the 22 who received anti-amyloid drugs the longest — eight years on average — had half the risk of developing cognitive problems as people who didn't receive the drugs. Researchers cannot disclose if Brian was among those 22 who received a drug and not a placebo in the trial's early phase. But in later phases, he received an anti-amyloid drug, and he currently gets infusions of another one.

“Why am I still asymptomatic?” Brian mused. “Is it because I’m like my dad? Is it because of the drug therapies?”

Brian is acutely aware that a cousin his age developed Alzheimer’s at 50, had to move in with relatives and needs accommodations at his manufacturing job.

Brian plays word games and Sudoku to keep his mind sharp. “Sometimes I’ve had a bad day and forgotten a couple people’s names and sort of got a little concerned,” he said. Then, he’ll test himself on the names, and “usually, I’ll come back around and go, ‘Oh that was so and so.’”

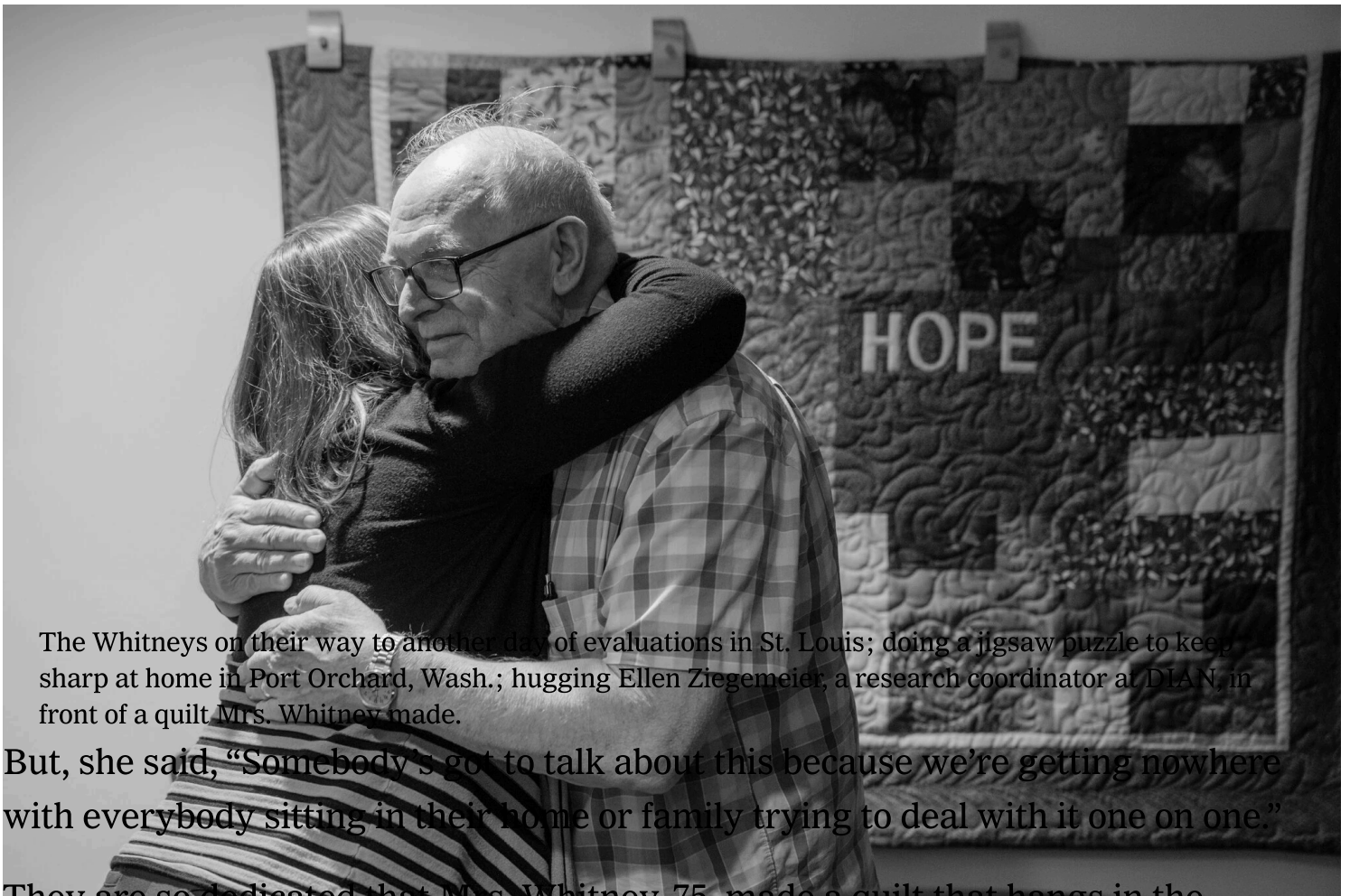
Generally, though, “I don’t really feel like I dwell on it every single day,” he said. “Early on, I really did, but maybe I’ve gotten to the point where this is what it is.”

That approach seems to have been absorbed by Brian’s 15-year-old daughter. Brian and his wife have not emphasized the family’s Alzheimer’s history, but their daughter has watched nurses give him drug infusions at home and has accompanied him to St. Louis. She has told her parents that she isn’t afraid to be tested for the mutation, a decision she can make after turning 18, and that if she has it, she will participate in studies.

“We’re just grateful for being a part of any sort of research regarding Alzheimer’s,” Brian said.

Some relatives are less open about the disease. “There are people in the family who don’t want to talk about it,” Ione Whitney said. “It really is hard to put your medical information out there, to be public about it because everybody’s got a theory — like, if you ate right, this wouldn’t happen to you.”





The Whitneys on their way to another day of evaluations in St. Louis; doing a jigsaw puzzle to keep sharp at home in Port Orchard, Wash.; hugging Ellen Ziegemeier, a research coordinator at DIAN, in front of a quilt Mrs. Whitney made.

But, she said, “Somebody’s got to talk about this because we’re getting nowhere with everybody sitting in their home or family trying to deal with it one on one.”

They are so dedicated that Mrs. Whitney, 75, made a quilt that hangs in the researchers’ offices.

“It’s a chance for you to give back to humanity,” Brian said. “It is scary getting involved in research,” he said. But “it’s been almost freeing in a way because I can face that in a community of other people who are going through the same things.”

During Doug Whitney’s recent testing in St. Louis, he told Dr. Llibre-Guerra he wasn’t experiencing difficulties, except for sometimes forgetting names and recent events.

He accurately answered questions during a cognitive assessment. Asked the difference between a lie and a mistake, he replied, “a mistake is generally unintentional; a lie is generally intentional.”

Later, Dr. Llibre-Guerra said that compared with four years ago, Mr. Whitney’s cognitive scores showed no significant decline, only a slight overall drop, probably attributable to his age. On some tests, he actually scored better, Dr. Llibre-Guerra

said, noting that scores can fluctuate.

The one test on which his scores have steadily worsened involves visual-spatial function, which could reflect the tau accumulation in the brain area linked to such skills, Dr. Llibre-Guerra said.

His performance far exceeds that of relatives with the mutation, most of them much younger. When they reach the family's typical age of impairment, Dr. Llibre-Guerra said, "you start seeing decline and usually it's really consistent."

Scientists have not yet found Mr. Whitney's "missing needle in the haystack and said, 'eureka,'" Dr. Bateman said, but they will keep searching. The puzzle that protects Doug Whitney is too valuable not to be solved.



Pam Belluck is a health and science reporter for The Times, covering a range of subjects, including reproductive health, long Covid, brain science, neurological disorders, mental health and genetics.

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