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AARP Bulletin

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SPECIAL REPORT

THE FUTURE OF ALZHEIMER'S

**WHY DOCTORS HAVE NEW HOPE ABOUT THE DISEASE—
AND WHAT THAT MEANS FOR YOU**

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HEALTH TIPS**

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IS INVOLVED**

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THIS YEAR'S PRIORITIES
CEO JO ANN JENKINS ON THE ISSUES
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ALZHEIMER'S: NEW LEARNINGS, RISING HOPE

SINCE ITS DISCOVERY 117 YEARS AGO, THE BRAIN DISEASE HAS STYMIED RESEARCHERS. BUT THAT HAS BEGUN TO CHANGE, AND FOR THE FIRST TIME, WAYS TO PREVENT, SLOW AND EVEN TREAT THE DISEASE ARE EMERGING

BY SARI HARRAR

Leonard Chayrez called his partner in a panic. “I couldn’t remember how to use the credit card machine at the gas station, so I drove to three,” says Chayrez, a retired floral designer in Phoenix. “I kept forgetting things.”

That was in 2018. One year later, Chayrez, 58, was diagnosed with mild cognitive impairment, and shortly thereafter, Alzheimer’s disease. Since June 2023, he has received twice-monthly infusions of lecanemab (Leqembi), the headline-grabbing medication approved last year by the Food and Drug Administration that removes Alzheimer’s hallmark plaques from the brain and slows down memory loss. “I think I’ll have more days of happiness,” Chayrez says.

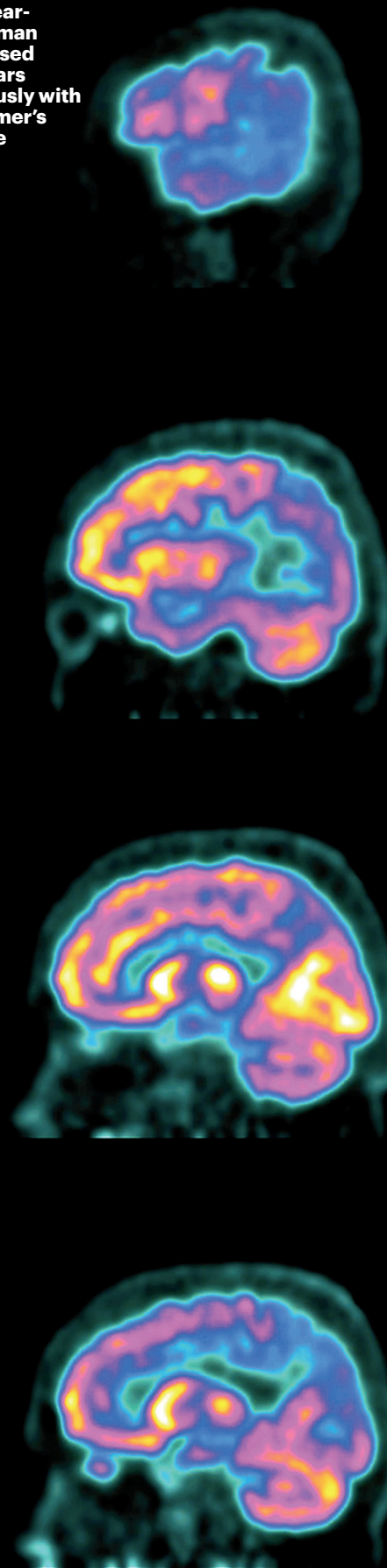
6.7 million
the number of
older Americans
who are living
with Alzheimer’s
disease

It is a measure of how bleak the outlook for patients was that lecanemab garnered so much attention—and hope. It isn’t a miracle drug. It doesn’t stop, reverse or cure this mind-robbing disorder; on average, it can slow mental decline by a mere five months over an 18-month treatment period, the research says. It comes with dangers of its own, including the risk of brain swelling and bleeding. Studies are ongoing. But despite all these drawbacks, it represents a huge breakthrough, a foundation on which the medical community can finally begin to build.

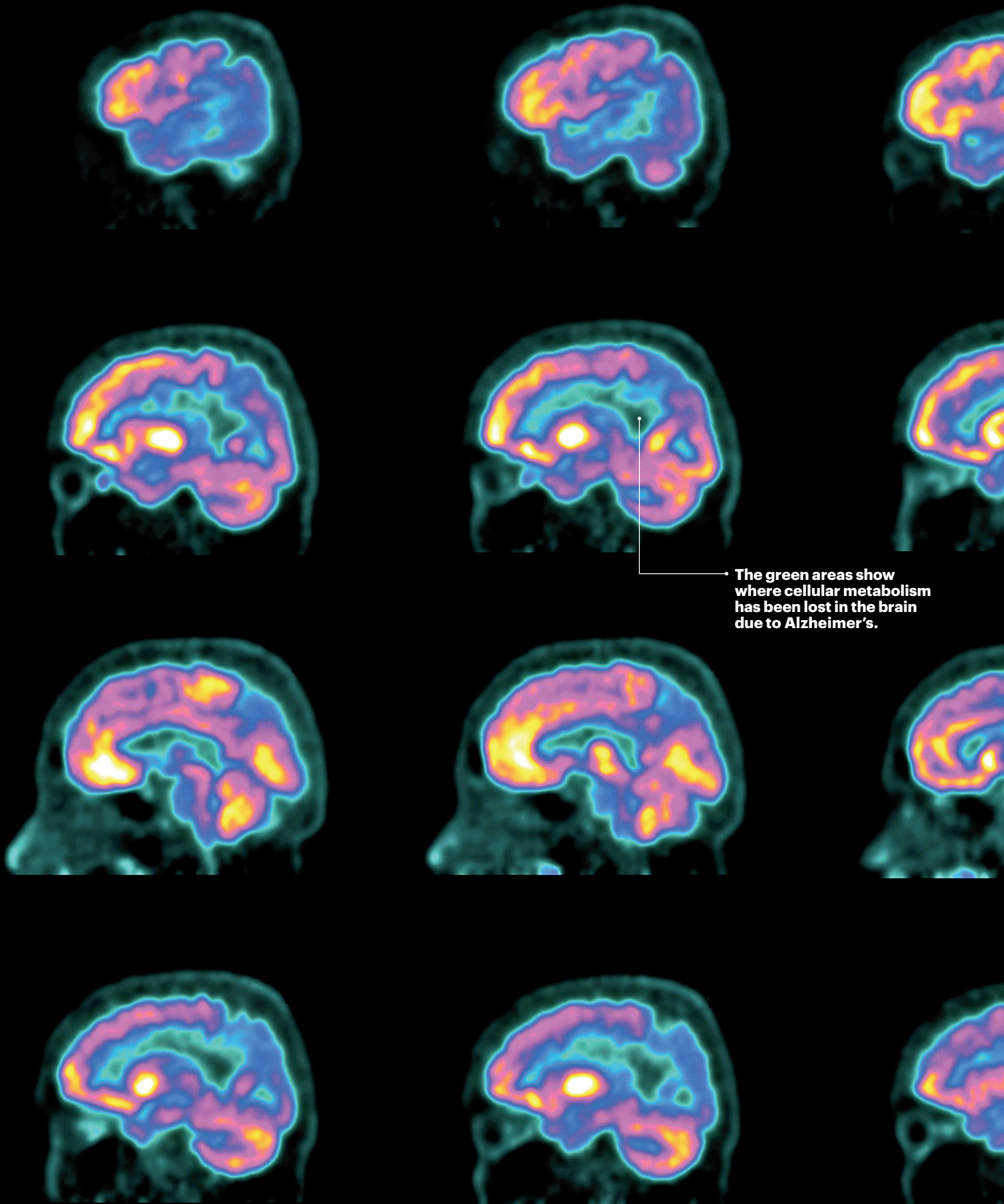
Neurologist and Alzheimer’s researcher Randall Bateman, M.D., puts it this way: “This is the beginning of the ability to treat, and change the course of, Alzheimer’s disease.”

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PET scans of the brain of a 58-year-old woman diagnosed two years previously with Alzheimer’s disease



ZEPHYRUS/SCIENCE SOURCE



The green areas show where cellular metabolism has been lost in the brain due to Alzheimer's.

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THE FIRST LITTLE VICTORY

Only patients with early-stage symptomatic Alzheimer's disease and confirmed high levels of amyloid proteins in the brain are candidates for lecanemab. In a 2023 community-based Mayo Clinic study, only 8 to 17 percent of those were eligible based on the trial criteria. And its costs—an estimated \$6,636 annually, even with Medicare coverage—are prohibitive for many.

Yet after more than 40 years of studies, tens of billions in research funding, at least 146 failed drugs and plenty of public controversy and private despair among scientists, the approval of lecanemab marks a major turning point in the fight against Alzheimer's. It's the first "disease-modifying" drug for Alzheimer's to receive traditional FDA approval and win standard Medicare coverage. Most significantly, lecanemab is one in a handful of recent advances expected to—at last—transform how the medical community diagnoses, treats and eventually prevents one of America's most-feared diseases.

"I've been doing this for 45 years," says geriatrician and neuroscientist Howard Fillit, M.D., cofounder and chief science officer of the Alzheimer's Drug Discovery Foundation. "This really is a watershed time."

Over the next five years, scientists anticipate more breakthroughs: convenient blood tests to diagnose Alzheimer's in your doctor's office; a variety of drugs that can attack the disease on many different fronts; and customized, research-proven lifestyle strategies that will likely become standards of care. Within 10 years, scientists say, Alzheimer's may be handled in your doctor's office the way heart disease and diabetes are now—diagnosed via blood tests and treated with a combination of drugs and lifestyle strategies. The dream of patients, doctors, researchers and society: Instead of a terminal disease, Alzheimer's could become treatable, preventable—and even reversible.

NO TIME TO WASTE

It's a race against time. By 2030, 8.5 million older Americans are projected to have Alzheimer's. A half-million of us will develop it this year.

Once it begins, there's no way to halt it. As brain cells die, connections between them



Leonard Chayrez (left, with his partner, Mark Garrity) has been taking the anti-Alzheimer's drug lecanemab since June 2023. "I think I'll have more days of happiness," he says.

"For the first time, we can bend the curve of cognitive decline. ... But it's not good enough. We need a home run."

—REISA SPERLING, M.D.,
director of the Center for Alzheimer Research
and Treatment at Brigham and Women's
Hospital in Boston

wither and the brain itself shrinks, memory worsens, thinking skills decline and navigating everyday life becomes less and less possible. At least a third of people with early-stage Alzheimer's slip into a more severe stage in about three years, putting lecanemab and possibly other early-stage treatments out of reach. Because heredity plays a role, many of us with a family history of Alzheimer's have long looked to the future with dread.

Rochelle Long, 67, of Shaker Heights, Ohio, cares for her 86-year-old mother with ad-

vanced Alzheimer's while working full-time. Her father, grandmother, four aunts and an uncle all had the disorder. So far, she's OK, but optimism comes hard. "Drug companies have spent how many billions of dollars through the years, and this is where we are?" Long says. "It's disheartening."

Even the most optimistic experts agree that lecanemab itself won't turn the tide—but it could become part of an arsenal that can. "For the first time, we can bend the curve of cognitive decline," says Harvard Medical School neurologist Reisa Sperling, M.D., director of the Center for Alzheimer Research and Treatment at Brigham and Women's Hospital in Boston. Sperling is heading a new study looking at lecanemab to prevent Alzheimer's in people at risk. "As a neurologist who takes care of patients and someone whose father and grandfather died of the disease, I think an extra five to six months of relative stability and independence is worth it," she says. "But it's not good enough. We need a home run."

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And at last, the medical community may be getting its turn at bat.

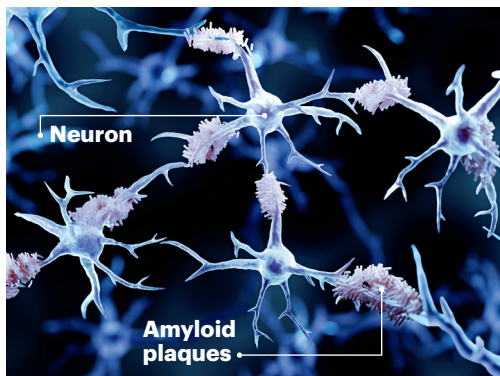
WHY IS THIS SO DIFFICULT?

Across the pharmaceutical industry, up to 90 percent of all experimental medicines that reach clinical trials don't make it to market, either because they don't work or they have bad side effects. Alzheimer's drug research has an even higher failure rate. From 2004 to mid-2021, 98 experimental Alzheimer's drugs failed in later-stage human studies. More were abandoned in labs or early human trials. Not a single new drug for Alzheimer's was approved by the FDA for nearly 18 years. Among the research community, "people were despondent; it was like a clinical depression across the field," Bateman says.

One major obstacle has been the historical difficulty of diagnosing Alzheimer's. Brain scans have shown that 17 to 22 percent of participants in Alzheimer's studies in the recent past did not have amyloid plaque and may not have actually had the disease. And 36 percent of failed phase 3 drug trials from 2004 to 2021 didn't test participants for the presence of amyloid plaques or tau tangles, meaning their cognitive problems could have been due to something else. "You're never going to get a positive result when you've got the wrong people in the trial," says Catherine Mummery, head of clinical trials at University College London's Dementia Research Centre.

Another major issue: Researchers may have been too narrowly focused on one culprit. One-quarter of the failed drugs were aimed at amyloid plaques—globes of protein in the brain that were first spotted 117 years ago by German psychiatrist Alois Alzheimer in the brain of a woman with severe dementia. Since these sticky blobs were the defining feature of the disease, it made sense that they were the primary target of researchers.

"The reason we've been so focused on plaques for so long is that they really stand out as soon as you look at the brains of people with Alzheimer's disease," says Kyle Travaglini, a scientist at the Allen Institute for Brain Science in Seattle, where he is part of a team creating an atlas cataloging how thousands of



“The reason we’ve been so focused on plaques for so long is that they really stand out as soon as you look at the brain. ... You see these giant, ugly balls all over the brain.”

—KYLE TRAVAGLINI
scientist at the Allen Institute for
Brain Science in Seattle

different types of brain cells are changed in Alzheimer's disease. "You see these giant, ugly balls all over the brain. It wasn't unreasonable to look at them initially." Yet drug after drug developed to target the amyloid plaques—most notably aducanumab, an earlier drug similar to lecanemab that was recently discontinued—failed to show any clear impact on the cognitive decline of patients. Lecanemab

is the first to show even modest success. Donanemab, which also clears amyloid plaques and slows disease progression, is expected to receive full FDA approval this year. And researchers are beginning to look at using focused ultrasound to open the blood-brain

barrier and amp up the effectiveness of anti-amyloid drugs. In January, a West Virginia University study of three people that combined focused ultrasound with drug infusions removed more amyloid than the drug alone.

NEW APPROACHES, NEW HOPE

Other theories about what triggers Alzheimer's are getting more attention—and drugs that target culprits other than amyloid are moving into early- and late-stage human trials. In 2016, 56 percent of disease-modifying

Alzheimer's drugs in human studies were aimed at amyloid plaque. By 2023, of 141 treatments in human trials, 84 percent were *not* for amyloid. Fillit says the intensity and variety of research—and lessons learned during years of frustration—should lead to faster progress.

Targeting amyloid is significant, he says, "but it's not the whole story." There's a growing consensus that several factors might work together to cause the disease. Researchers continue to study amyloid plaques, but other human trials are underway on drugs designed to reduce brain inflammation, protect synapses (parts of brain cells that communicate with each other) and improve aspects of brain metabolism (such as processing blood sugar, the brain's preferred fuel).

In particular, researchers are taking a harder look at tau tangles, globs of protein that form inside the brain cells themselves. One medicine attracting a lot of attention is called BIIB080. In a trial led by Mummery at the University College London, it was the first anti-tau drug to successfully remove these damaging protein tangles from brain cells in a human study.

"Tau tangles in the brain seem to mirror much closer to the cognitive symptoms people have," Bateman says. "A lot of us think that's a clue. If we could neutralize tau, people's symptoms could be better managed."

At the University of California San Francisco (UCSF), a study called the Alzheimer's Tau Platform recently won a \$151 million grant from the National Institutes of Health (NIH) to test two anti-tau drugs plus an anti-amyloid drug that will be given together to 900 people with early-stage disease. "At a certain point with amyloid accumulation, tau gets sparked like a forest fire that really starts to do damage," Mummery says.

On a different front, Finnish researchers launched a study that combines brain-healthy lifestyle steps with metformin, a diabetes drug that might improve blood sugar processing in the brain. The study will look at the effects of lifestyle plus the medication in people at risk for Alzheimer's and dementia who do not have thinking and memory problems.

"The brain is only 3 percent of body weight but uses 20 to 25 percent of the body's energy, mostly from glucose," Fillit explains. Trouble absorbing blood sugar, called insulin

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1906
the year German
psychiatrist
Alois Alzheimer
discovered the
brain disease

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Cover Story



Linda Van Eldik in her lab at the University of Kentucky. She says chronic inflammation, by overwhelming the immune system, may be an underlying cause of Alzheimer's.

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resistance, increases with age. "If the brain is deprived of energy, neurons can die," says Fillit, whose foundation helps fund the study. "We want to see if the combination would have more benefit than either one alone."

Having drugs against these various factors, Fillit says, means that within 10 years, customized, multidrug treatments will likely be prescribed based on a simple blood test. (An analogy would be our approach to treating heart disease, which might involve cholesterol medications, blood pressure pills, blood sugar controls, dietary changes or a combination of these treatments.)

"We are starting to give combination drugs in trials," Mummery says. "I believe that in 10 years' time, we will have blood biomarkers that give a fingerprint of what your disease looks like. We'll be able to use that to define an individualized treatment regime. We'll be thinking about brain health in terms of prevention more than just dementia symptoms."

"I'm an optimist," she adds. "We have a foundation. Once you have that foundation, research can accelerate."

IS YOUR BRAIN ON FIRE?

"I've always been intrigued by inflammation," says Linda Van Eldik, director of the Sanders-Brown Center on Aging and the Alzheimer's Disease Research Center at the University of Kentucky in Lexington. She says chronic inflammation may be one of the primary drivers of Alzheimer's.

Van Eldik studies microglia, immune cells that patrol the brain by the tens of billions. Normally they work like trash trucks,

gobbling up little scraps of amyloid protein waste before it can cause trouble. But when overwhelmed by inflammation, microglia pump out a torrent of inflammatory chemicals called cytokines.

"Cytokines damage nerve cells [neurons] and synapses, reducing communication between cells," she explains. It's a vicious cycle. When cells are damaged by cytokines, microglia "start producing more inflammation, you get more damage, then you get more inflammation," Van Eldik says.

This may explain why aging, head injuries, stroke and other brain insults increase risk for Alzheimer's and other dementias, she says.

Van Eldik's NIH-funded research includes

BIIB080
an investigational
anti-tau drug that
successfully removed
the toxic protein
from brain cells

versions of an anti-inflammation drug candidate that blocks microglia from overproducing inflammatory compounds without stopping their normal trash-collection duties, she says. In phase I clinical studies, the drug was found to be safe in healthy adults. It is currently being tested in people who had strokes caused by brain bleeding and with brain cancers treated by radiation. If successful, Van Eldik expects the drug will be tested next in people with Alzheimer's. She also expects it to be used in other cell-damaging brain conditions in which abnormal inflammation plays a role. "This drug has the potential to be a first-in-class disease-modifying therapy for Alzheimer's disease and related dementias," Van Eldik says.

Controlling neuroinflammation is at the forefront of new treatment ideas for Alzheimer's, Fillit agrees. In 2023, there were more drugs in human trials for inflammation than for any other Alzheimer's disease-modifying factor, and Fillit says inflammation drugs could be among the first Alzheimer's compounds to win FDA approval in the next three to five years.

Controlling neuroinflammation is at the forefront of new drug ideas for Alzheimer's. In 2023, there were more drugs in human trials for inflammation than for any other target.

THE BATTLE ON THE HOME FRONT

The difficulty in diagnosing Alzheimer's accurately has long hindered scientists' ability to study the disease. Historically, it could be confirmed only by examining the brain after death. Today, researchers can make a firm diagnosis of Alzheimer's by spotting amyloid plaque via positron emission tomography (PET), or by testing for its presence with a lumbar puncture, says Ronald Petersen, M.D., a Mayo Clinic neurologist and Alzheimer's researcher. "But the first one is expensive. And the second one

is invasive." So they're not routinely used.

New and on-the-horizon blood tests that measure amyloid and tau in the brain aid researchers in tracking brain changes during studies. These blood tests are beginning to be used in doctors' offices. Sperling expects tests to be available in the next year showing not just how much amyloid is in the brain but whether it is triggering the explosion of tau that seems to torpedo thinking and memory. This could help, in the future, determine risk in people who do not have Alzheimer's symptoms. "We're going to do a much better job of predicting individual risk," she says.

That's significant not just for treatment but in terms of prevention as well. For all the deep science that surrounds the war on Alzheimer's, one of the most important weapons against the disease remains the simplest: healthy living. In that way, late-life Alzheimer's resembles heart disease, diabetes and many other chronic conditions linked to aging: There is a genetic component, yet so much of prevention is in our own hands. A 2022 University of Minnesota study found

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Ask The Expert

THE TRUTH ABOUT COCHLEAR IMPLANTS

Straining to hear each day, even when using powerful hearing aids?



DR. MAURA COSETTI,
Cochlear Medical Advisor

Dr. Maura Cosetti, a cochlear implant surgeon and medical advisor to Cochlear, the world leader in cochlear implants, answers questions about cochlear implants and how they are different from hearing aids.

Feeling frustrated and sometimes even exhausted from listening? Whether it happens suddenly or gradually over time, hearing loss can affect you physically and emotionally. Being unable to hear impacts your ability to communicate with your loved ones, hear in noisy environments, talk on the phone, and may force you to become more reliant on your family members.

Cochlear implants work differently than hearing aids. Rather than amplifying sound, they use state-of-the-art electronic components and software to help provide access to the sounds you've been missing. They are designed to help you hear better and understand speech in all situations, including noisy environments.

- Q How are cochlear implants different than hearing aids?** Hearing aids help many people by making the sounds they hear louder. Unfortunately, as hearing loss progresses, sounds not only need to be made louder, they need to be made clearer. Cochlear implants can help give you that clarity, especially in noisy environments.¹ Be sure to discuss your options with a Hearing Implant Specialist in your area.
- Q How do I know a cochlear implant will work for me?** Cochlear hearing implant technology is very reliable.² In fact, it has been around for 40 years and Cochlear has provided more than 700,000 implantable hearing devices.
- Q Is it major surgery?** No, not at all. The procedure is often done on an outpatient basis and typically takes just a couple of hours.
- Q Am I too old to get a cochlear implant?** No, it's never too late to regain access to the sounds you're missing.
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1. The Nucleus Freedom Cochlear Implant System: Adult Post-Market Surveillance Trial Results. 2008 June. 2. Cochlear Nucleus Implant Reliability Report. Volume 16 | December 2017. D1175804. Cochlear Ltd; 2018. Please seek advice from your health professional about treatments for hearing loss. Outcomes may vary, and your health professional will advise you about the factors which could affect your outcome. Always read the instructions for use. Not all products are available in all countries. Please contact your local Cochlear representative for product information. Views expressed by hearing health providers are that of the individual. ©2024 Cochlear Limited. All rights reserved. Trademarks and registered trademarks are the property of Cochlear Limited. CAM-MK-PR-530 ISS1 MAR21

CONTINUED FROM PAGE 17

that an estimated 41 percent of Alzheimer's and related dementias were attributed to a dozen modifiable risk factors, of which high blood pressure, obesity and physical inactivity had the most impact.

"People need to know, 'What are my risk factors for Alzheimer's and other dementias, and are they things I can do something about?'" says Kristine Yaffe, M.D., director of the Center for Population Brain Health at UCSF. Yaffe was the lead researcher on a November 2023 study of 172 adults ages 70 to 89 who had two or more risk factors for dementia. Those who worked on one or more personally chosen risk factors for two years boosted their scores on tests of thinking and memory and lowered their dementia risk more than those who simply received educational materials.

"Instead of a one-size-fits-all program, we wanted to see what would happen if we personalized it," Yaffe says. Strategies included

taking care of high blood pressure, diabetes and/or depression; exercising; getting sound sleep; socializing; and quitting smoking.

Dean Ornish, M.D., founder of the non-profit Preventive Medicine Research Institute and clinical professor of medicine at UCSF, is directing the first randomized controlled trial to determine if the same lifestyle changes that he pioneered to reverse coronary

heart disease might slow, stop or reverse the progression of mild cognitive impairment due to Alzheimer's disease. "What's good for your heart is also good for your brain, because they share many of the same underlying biological mechanisms," Ornish says. "We're at a state of evidence with Alzheimer's similar to where we were 45 years ago with coronary artery dis-

ease. At that time, it was thought that heart disease could only get worse. We found that bigger lifestyle changes could often reverse the progression of heart disease. We are studying if the same may be true for mild cognitive impairment due to Alzheimer's dis-

ease as well. One man shared, 'I have a sense of hope.'" *UnDo It!*, a book Ornish wrote with his wife, Anne, describes his lifestyle medicine program for both heart disease and Alzheimer's.

Prevention studies using drugs are gearing up. Researchers study medications like lecanemab in people with early evidence of Alzheimer's brain changes who do not yet have life-changing thinking and memory problems. In the future, Sperling says, drugs could be started before memory troubles appear—the way people with high cholesterol take statins to lower risk for a heart attack and those with diabetes take blood-sugar-lowering drugs to protect against complications such as kidney disease.

"This is the way forward," she says. "It's exactly what we do in heart disease and diabetes. We lower cholesterol before someone has a heart attack. This is what we have to do in Alzheimer's disease.

"For the first time, it's very feasible." ■

Sari Harrar is a contributing editor for AARP The Magazine and frequently writes on health for the Bulletin, including recent cover stories on cancer and heart disease.

41%
of Alzheimer's and related dementias were attributed to a dozen modifiable risk factors, including high blood pressure, obesity and physical inactivity.

AARP and Brain Health

STAYING MENTALLY SHARP AND MAINTAINING A HEALTHY BRAIN ARE AMONG THE TOP GOALS OF AGING AMERICANS, WHICH IS WHY SUPPORTING BRAIN HEALTH IS ALSO AMONG THE TOP PRIORITIES OF AARP. HERE ARE SEVERAL WAYS WE ARE ADDRESSING THE TOPIC IN 2024.

▶ Staying Sharp

AARP's well-established online program to help you fit brain-healthy habits into all aspects of your life is available free to both AARP members and nonmembers who register on our website. Signing up gives you free access to the Cognitive Assessment, a test to get a snapshot of your reasoning, memory and attention performance that day. AARP members get additional resources, including more challenges, daily activities, recipes, meditations and learning videos.

stayingsharp.aarp.org

▶ Brain Health Resource Center

Launched in January, the resource center has answers to all your questions about vital brain health topics, among them brain maintenance, Alzheimer's and other dementias, stroke, depression and anxiety, traumatic brain injury and Parkinson's disease. The resource center also is a gateway to AARP's Hearing Center, which has a free hearing test for members; brain health research reports; information and resources for caregivers; and much more.

aarp.org/brainhealth

▶ AARP's Global Council On Brain Health

Convened by AARP, the GCBH is a collaborative of scientists, health professionals, scholars and policy experts from around the world working in areas of brain health related to human cognition. The alliance's goal: Offer the best possible advice about what older adults can do to maintain and improve their brain health. The GCBH regularly issues new research reports, videos and other resources for both professionals and consumers.

aarp.org/GCBH

▶ Dementia Discovery Fund

AARP in 2018 established the AARP Brain Health Fund, which then invested \$60 million in the Dementia Discovery Fund, an international investment pool specifically targeting research toward treatments to prevent, defer and/or slow the decline from dementia. Five years into the initiative, the fund has invested in 17 biotech and health science companies with 40 active programs focusing on dementia; 13 of those programs are in clinical trials.

www.ddf.vc

▶ Brain Health Action

Finally, AARP is launching a collaborative called Brain Health Action to spread the word that cognitive decline is not an inevitable part of aging. Organizations that join will be part of the national conversation to help their members, employees and communities make brain-healthy choices.

aarp.org/brain-health-action

