Three New Studies Advancing the Alzheimer’s Conversation

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Researchers have new clues about how the disease kills brain cells, how it spreads, and why some brains resist it. Here’s what the science says and what it could mean.

In the span of a few weeks this summer, three separate research teams published findings that together offer something the Alzheimer’s field has been working toward for decades: a clearer picture of how the disease actually works. Not just what it looks like in the brain, but how it kills cells, how it moves from one neuron to the next, and, perhaps most importantly, why some people seem to be protected from its worst effects even when their brains show all the hallmarks of disease.

None of these studies offers a cure, and experts say there are important caveats to consider before accepting any of them as established fact. But they *do* offer important new layers to what we know in areas previously underexplored.

“For a long time, the focus in Alzheimer’s research has been on the accumulation of amyloid plaques and tau tangles,” says Dr. **Manisha Parulekar**, MD, director of the Division of Geriatrics at Hackensack University Medical Center and co-director of the Center for Memory Loss and Brain Health at Hackensack University Medical Center. “While those are still crucial pieces of the puzzle, these new findings highlight other critical processes,” she tells Super Age.

Taken together, the studies represent a meaningful shift in understanding: one that points toward new therapeutic targets and, for anyone focused on longevity and brain health, raises questions worth thinking about now.

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How Alzheimer’s Kills Brain Cells: A Missing Piece, Found

For decades, scientists have known that Alzheimer’s involves the buildup of toxic proteins inside neurons. They’ve also known that those neurons eventually die, leading to memory loss and cognitive decline. What they haven’t known with any real precision is the mechanism connecting those two facts. How, exactly, do toxic proteins kill brain cells?

Now, a **new study** from King’s College London, published in *Nature Communications*, may have found a critical missing link. The researchers identified a process called karyoptosis, a series of chemical reactions triggered when toxic proteins accumulate inside a cell. As the process unfolds, the cell’s nucleus, which contains its genetic material, gradually shrinks and eventually breaks apart.

To understand why this matters, it helps to know what *wasn’t* explaining the damage before. Scientists had long catalogued various forms of cell death, including a well-studied process called **apoptosis**. But those mechanisms never fully accounted for the scale of neuron loss seen in Alzheimer’s and related dementias. (Scientists often cite **necroptosis**, **excitotoxicity**, **autophagy failure**, and **oxidative stress**, as corroborating factors.)

Karyoptosis, the King’s College team suggests, may be part of what was missing. This can be induced by “proteotoxic stress,” occurring when damaged or misfolded proteins accumulate inside cells.

The researchers examined 3,000 brain cells from 28 people with either Alzheimer’s or frontotemporal dementia (FTD). They found signs of karyoptosis in 35% of cells from

the frontal cortex of people with Alzheimer's, compared with just 15% in healthy older adults. In laboratory experiments using the cortical neurons of rats, they also identified a promising molecular switch — an interaction between a kinase called p38 MAP kinase and a protein called LaminB1 — that appears to control the process. Blocking that switch reduced markers of karyoptosis.

"By specifically targeting the interaction between p38 MAP kinase and LaminB1, we may slow down the process of cell death, buying time for more pinpointed therapies," said Dr. Manolis Fanto, PhD, of King's College London, in a **news release** for the study.

A Possible Answer for How Alzheimer's Spreads: A Protein With a Double Life

Separately, a ***Cell* study** on mice published by a team at the University of Utah Health tackled a different but equally important question: how does Alzheimer's spread through the brain?

The disease is marked by the buildup of a toxic protein called Tau. In a healthy neuron, Tau performs normal functions. In Alzheimer's, it begins clumping into what the researchers describe as "glue monsters," tangles that block the cell's internal transport system and eventually kill it. But these tangles can break down into smaller fragments, called Tau seeds, which can travel to neighboring neurons and corrupt the healthy Tau they find there.

Using mice as their subjects, the Utah researchers discovered that a brain protein called Arc appears to enable that travel. Under normal circumstances, Arc helps neurons communicate, packaging itself inside tiny membrane-bound sacs called **extracellular vesicles**, which carry cellular signals from one neuron to another. The researchers found that toxic Tau exploits this system, hitching a ride inside the

protein and traveling from diseased neurons into healthy ones, seeding new damage.

When Arc was removed from mice with Alzheimer's pathology, Tau transfer dropped dramatically. In a **news release** for the study, the researchers describe it as "severely, severely reduced" and, even more encouragingly, "almost gone."

There's one catch: Arc also plays a protective role in the early stages of disease, so blocking it entirely isn't straightforwardly beneficial.

"When Arc is absent, Tau becomes trapped inside neurons and accumulates to toxic levels. When Arc is present, Tau can be released in extracellular vesicles. While this helps reduce Tau buildup within the original neuron, the released Tau can be taken up by neighboring healthy neurons, promoting the spread of pathology," Dr. Mitali Tyagi, PhD, postdoctoral research associate at Washington University in St. Louis and first author of the study, said in that news release.

If the results hold in humans — a supremely important *if* — the more promising strategy would be to intercept the Tau-containing vesicles after they leave diseased cells but before they enter healthy ones, the researchers suggest. This could help stop the spread of Tau without disrupting Arc's protective function in the process, safely curbing disease progression.

Why Some Brains May Resist: The Role of Immature Neurons

The **third study**, from the Netherlands Institute for Neuroscience and published in *Cell Stem Cell*, addresses what may be the most important question in Alzheimer's research: why does the same disease affect people so differently?

Around 30% of older adults whose brains show the biological hallmarks of Alzheimer's — amyloid plaques, Tau tangles, structural changes — never develop dementia symptoms and remain cognitively sharp. The question of what protects them is one researchers have been trying to answer for years.

The Dutch team focused on a rare and underexplored cell type: immature neurons in the hippocampus, the brain's primary memory center. These are cells that have not fully matured, remaining in a kind of developmental in-between state.

What they found surprised them. Resilient individuals, those whose brains showed Alzheimer's pathology but who never developed dementia, didn't have dramatically more of these cells than people who did develop the disease. The difference wasn't in the quantity of immature neurons, but in their behavior.

"In resilient individuals, these cells seem to activate programs that help them survive and cope with damage," said senior author Dr. Evgenia Salta, PhD, in another **news release**. "We also see lower signals related to inflammation and cell death."

The immature neurons in resilient brains appeared to be doing something active. Not just surviving the damage, but potentially supporting the surrounding tissue and helping the brain maintain function. Salta describes it as "fertilizer in a garden that has started falling apart."

The study is careful about the limits of its conclusions. Because the research was conducted on donated brain tissue rather than living brains, the team wasn't able to observe how the cells function in real time. Their hypotheses about what the cells are doing are inferences from the data, not confirmed mechanisms. And Dr. Salta is explicit that cognitive resilience is unlikely to have a single explanation.

"This is one piece of a very large puzzle," Dr. Salta says. "There will never be just one factor that explains resilience."

Which Finding Is Most Likely to Translate Into a Therapy?

Alzheimer's looms large in our collective imagination because it risks us losing everything that makes us *us*. And since it hits that existential nerve, it's tempting when reading the research to latch onto ideas still in their infancy as promises for future

therapies.

But each study here has its own limitations worth noting, Dr. Parulekar notes: “It’s important to remember that these are early-stage findings. The karyoptosis and resilience studies were done on postmortem brain tissue, so we need to see if these processes can be observed and influenced in living brains. The Arc protein research was done in mice, and we know that mouse models don’t always translate perfectly to humans,” she tells Super Age. “So, while these are very exciting developments, we are still a long way from seeing new treatments in the clinic based on this research,” the doctor adds.

Asked which of these studies held the most near-term promise, she was enthusiastic about the King’s College team’s findings.

“The discovery of karyoptosis, a new form of cell death, gives us a more precise target for how to keep brain cells alive. It seems to be the most promising for a near-term therapy. The researchers have already identified a specific protein interaction that could be targeted by a drug. This is a very concrete starting point for drug development.”

She adds: “The other two studies are also incredibly valuable, but their paths to a treatment are a bit more complex. The Arc protein, for example, has both harmful and protective roles, so a simple ‘block Arc’ strategy might not work. The resilience finding is fascinating, but it’s not yet clear how to translate the unique properties of those ‘immature neurons’ into a therapy for people who don’t have them.”

What It Might Mean for the Future

Taken together, these studies suggest that the future of Alzheimer’s treatment may not be a single intervention but a combination of strategies that targets cell death, interrupts spread, and amplifies the brain’s own resilience mechanisms. That’s a more complex picture than a single drug could address, but it’s also a more complete one.

“This trio of studies really underscores a shift that’s been happening in the field,” says

Dr. Parulekar who treats patients with Alzheimer's disease. "We're moving beyond a singular focus on plaques and tangles and toward a more holistic understanding of the disease. We're now looking at how to protect brain cells from dying, how to stop the disease from spreading, and how to harness the brain's natural resilience. This multi-pronged approach is, in my opinion, the most promising path toward effective treatments and, eventually, a cure," she emphasizes.

The research also suggests that the lifestyle habits underpinning our broader brain health are as important as ever. The growing evidence that exercise, healthy diet, cognitive engagement, sleep quality, and stress management all influence the brain's capacity to withstand damage isn't disconnected from this research. It's probably pointing at the same underlying biology.

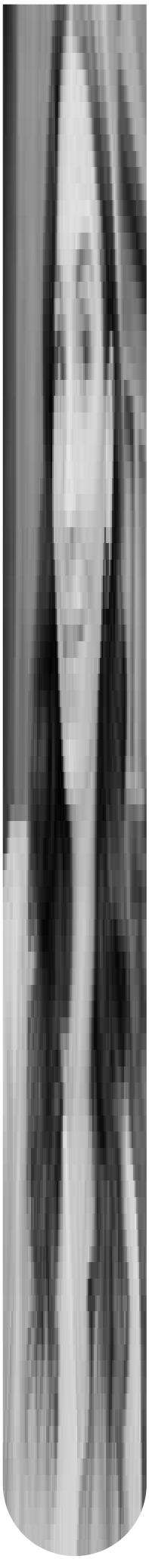
"These studies are a great example of how complex Alzheimer's is, and why there won't be a single 'magic bullet' that cures it," says Parulekar. "Instead, we're likely to see a future where we have a combination of therapies that target different aspects of the disease, much like how we treat heart disease today. It's a time of great hope and progress in our field."

The field is moving fast. These studies, published within days of each other, are a reminder that what we don't know about Alzheimer's is still vast, but shrinking — with three new steps in what could be the right direction.

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