What if autism and Alzheimer's come from the same place? (Emma Kumer/The Washington Post; iStock)

Surprising links between autism, Alzheimer's could change how we treat both

The idea that two conditions at opposite ends of life might be biologically linked is beginning to upend long-standing assumptions in brain science, blurring a divide that has shaped the field.

Yesterday at 6:51 a.m. EDT

12 min Summary

By Ariana Eunjung Cha

Joseph Buxbaum was initially unconvinced. When early hints of a connection between autism and Alzheimer's began to appear in the medical literature a few years ago, they struck him as implausible — one a condition of early brain development, the other driving decline in old age.

But the signals kept accumulating, and over time, his skepticism gave way to a new line of inquiry that could transform scientists' understanding of the two diseases.

"I came to this kicking and screaming. I didn't want to believe it," said Buxbaum, a professor of psychiatry, neuroscience and genetics/genomic

sciences at the Icahn School of Medicine at Mount Sinai.

Autism has long been treated almost exclusively as a childhood condition, with little attention paid to how it evolves with age. First formally recognized as a distinct diagnosis in 1980, it went largely unidentified in older generations. Only recently — as awareness grew and the first large diagnosed cohort reaches middle age — have researchers begun to study autistic adults in later life.

The data remains sparse: An analysis published last year found that just a tiny fraction of the more than 40,000 autism papers published between 1980 and 2021 included people over 50. But the number of studies about aging with autism is growing rapidly. Advances in brain imaging, DNA sequencing and molecular biology are revealing remarkable overlaps between autism and Alzheimer's, scientists say — in genes, in neural circuitry, even in patterns of disease.

The idea that two conditions at opposite ends of life might be biologically linked is beginning to upend long-standing assumptions in brain science, blurring a divide that has shaped the field for decades. Now, some researchers have begun to see the two as intertwined: that understanding Alzheimer's may require looking back to how the brain develops, and that insights into autism might, in turn, reshape how we understand Alzheimer's itself.

B. Blair Braden, who directs the Autism and Brain Aging Laboratory at Arizona State University, discusses the organ with students Melissa Walsh and Broc Pagni in 2018. (Deanna Dent/ASU Now)

Much of the research is still early, and in some cases conflicting and speculative, and it does not yet show that autism and Alzheimer's are

part of a single biological continuum. But the implications are profound: Both conditions remain mysterious and difficult to treat, and studying them together may open new paths for intervention.

“There are strong indications that something is going on — that the traditional differences demarcate neurodevelopment versus neurodegeneration may be fairly artificial when it comes to biology,” said Andy Shih, chief science officer of Autism Speaks, an advocacy group that funds research and is increasingly focused on this emerging area.

Unexpected risk

Separated by decades, both autism and Alzheimer’s take root in the same living circuitry — the human brain — a network of billions of neurons and trillions of synapses, constantly wiring and rewiring itself over a lifetime. In one, those connections form differently; in the other, they slowly come undone.

The connection first drew attention in the late 1990s to early 2000s, with unsettling findings: case reports of autistic adults developing dementia at a young age, followed more recently by larger, population-level studies suggesting elevated risk for this group.

Hard numbers are elusive. Many people now over 65 were never identified, making it difficult to estimate how many are on the spectrum. But if prevalence mirrors that seen in children — roughly 1 in 31 — researchers say the number could be as high as 1.97 million. And with 1 out of 9 Americans that age with Alzheimer’s, the overlap could be roughly 220,000 people.

Brian Lee, an epidemiologist at Drexel University, pointed to a 2021 analysis of Medicaid records published in Autism Research which found that people with autism were about 2.6 times more likely to be diagnosed with early-onset Alzheimer’s and related dementias compared with the general population. (The work was replicated in 2025 in a JAMA research letter that reached similar findings using Medicaid and Medicare data.)

Autism’s links to other brain disorders may extend beyond Alzheimer’s, with some studies pointing to a higher risk of Parkinson’s disease — a neurodegenerative disorder that affects movement, causing tremors, stiffness and slowed motion.

That work has led to a cascade of questions. Some are more practical, focused on individuals' health over a lifetime. Do communication barriers make it harder to receive adequate medical attention? Are routines around exercise different? What are the long-term effects of medication? And could coordination challenges lead to more head injuries? Alongside all of this is another factor: higher lifetime stress.

> "The idea is that autism as a condition leads to lifestyle changes that might predispose neurodegeneration."
>
> — Brian Lee

"The idea is that autism as a condition leads to lifestyle changes that might predispose neurodegeneration," Lee said.

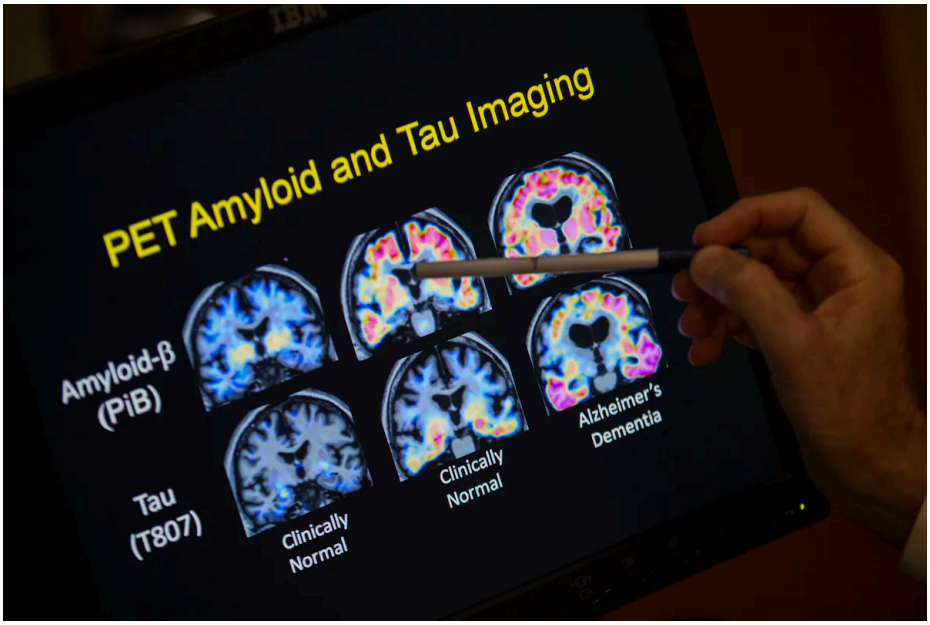
But health habits and environment alone do not seem to explain the pattern. Increasingly, researchers are finding that the overlap runs deeper — into the biology itself.

Faltering synapses

Nowhere is the overlap between autism and Alzheimer's more apparent — nor more concrete — than in the growing list of shared genes. A 2025 review published in the International Journal of Molecular Sciences identified at least 148 genes in common, many of them tied to the same fundamental processes that shape and sustain the brain over time.

The list of shared genes is long — and still growing. MECP2, ADNP, GRIN2B, SCN2A, NLGN, CNTNAP2: Many of them are deeply involved in how brain cells connect, signal and adapt over time.

Not all of their functions are fully understood, but taken together, they point to a common thread: Changes in the number, quality and placement of synapses — the junctions where neurons communicate — may shape how minds take form and, later, unravel.



A professor at Georgetown University Hospital points to PET scan results that are part of a study on Alzheimer's disease. (Evan Vucci/AP)

Among the common genes is SHANK3, one of the most well-known in autism.

In autism, mutations in SHANK3 — which encodes a protein of the same name that acts as a kind of structural framework at synapses, helping neurons communicate — can disrupt those connections early in development, altering how neural circuits are built. In Alzheimer's, levels of the same protein have been found to decline as the disease progresses, a shift that is associated with the gradual loss of connections.

Buxbaum, who has spent decades studying Alzheimer's, is probing this overlap directly.

In his lab, mice engineered with SHANK3 mutations and autism-like characteristics are trained to navigate mazes — first to learn a location, then to relearn it after the rules change. As they age, the mice struggle to adapt, taking longer to relearn the task. The deficits echo a hallmark of Alzheimer's: impaired cognitive flexibility.

Yet the mice present a paradox. Despite these impairments, they are unusually resistant to developing full-blown dementia-like pathology. "So you have to double or triple down in introducing bad things into the mouse brain to even get something that looks like Alzheimer's in a mouse," Buxbaum said.

While mouse brains are commonly studied in neuroscience, they still are critically different from human brains in a way that limits how

experiments translate to people. But that resistance in mice may offer important clues. If researchers can understand what protects mouse brains, they may uncover mechanisms that could one day be harnessed for humans.

Cellular 'housekeeping'

Even at rest, the brain is busy — and messy. Packed with roughly 170 billion cells, it is constantly generating waste that must be cleared away to keep its circuits running. Many of the shared genes between autism and Alzheimer's point to a single system: cellular "housekeeping."

Roughly half are linked to the mTOR pathway, which controls autophagy — the process by which cells clear debris, recycle components and remove toxic proteins.

When that system falters, the profound consequences unfold slowly. Waste accumulates. Proteins misfold. Communication between neurons begins to break down. Over time, researchers theorize, these disruptions could both help alter brain development and lead to the kind of degeneration seen in Alzheimer's.

In a largely interpretive paper published in January in Frontiers in Neuroscience, researchers described possible commonalities in MRI findings between autism and Alzheimer's, particularly involving the glymphatic system — a brain-wide network that helps clear metabolic waste, especially during sleep.

Patterns such as enlarged spaces around blood vessels and increased fluid around the brain were reported in both conditions, though these findings are preliminary. The work remains largely hypothesis-generating; while it may point to shared biological pathways, it does not establish a direct link between the two conditions.

William Phillips, a nuclear medicine physician at UT Health San Antonio who is an author of the study, said the findings caught his attention because the brain's cleanup system is closely linked to smell. In Alzheimer's, people often lose their sense of smell before memory problems begin — and although smell issues have been reported in autism, they have largely been dismissed as a sensory quirk rather than as a possible clue to brain health.

By concentrating on these mechanisms, the authors wrote, scientists might be able to develop "integrated treatment strategies that address

both disorders simultaneously, ultimately improving the quality of life for affected individuals."

Brain architectures

With the rise of brain imaging, researchers can now watch these conditions unfold in living brains — and the patterns are beginning to look unexpectedly similar.

For years, research in both autism and Alzheimer's focused on individual regions: which parts were larger or smaller, more or less active. Scientists were intrigued, for example, that Alzheimer's is associated with the shrinking of a brain region known as the amygdala, a structure involved in emotion, fear and social processing; in autism, the amygdala is often enlarged, although the findings have varied by age and study design.

Increasingly, though, attention has shifted to the connections between those regions — the networks that allow the brain to function as an integrated whole.

In two fields that have long operated separately, researchers have, in effect, converged on the same idea.

In autism, findings presented last year at the American Neuropsychiatric Association's annual conference suggest that the density and strength of synaptic connections may correlate with functioning; in some cases, more robust connectivity is associated with better functioning in daily life. In Alzheimer's, by contrast, the loss of those same connections correlates very strongly with cognitive decline, and some believe it may be a better anatomical correlate than the buildup of amyloid plaques or tangles of a protein known as tau, long considered the disease's defining indicators.

How those brain connections evolve over time — and what that might reveal about aging in autism and its potential links to Alzheimer's — is now becoming a central question, one that researchers like B. Blair Braden have begun to explore.

Braden directs the Autism and Brain Aging Laboratory at Arizona State University and has spent more than a decade recruiting dozens of adults with autism from the greater Phoenix area, asking them to return again and again for brain scans.

Her first major paper on the subject, published in 2022, pointed to changes in the hippocampus, a region critical for memory that shrinks with age in both autistic and non-autistic adults, but to a more severe extent and earlier for those with autism.

Braden has been struck by how the brain scans seemed to tell a similar story to the genetic and molecular work: “It’s kind of amazing to see how the results are coming together.”

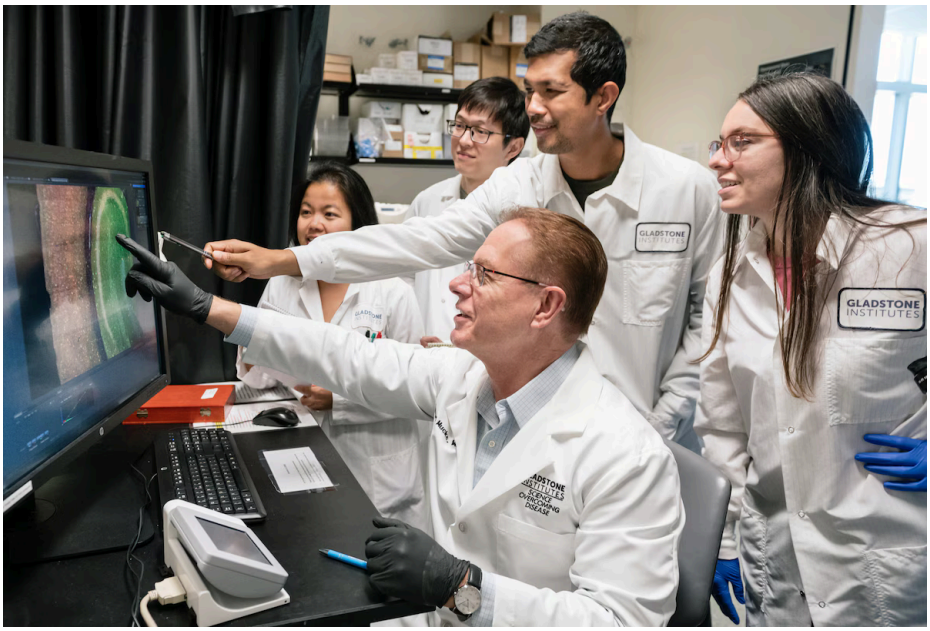
New hope

What’s emerging from this and other research labs is not just a shift in thinking, but the early outlines of new treatments.

Braden’s work, along with findings from other labs, points to a reorientation for Alzheimer’s treatments: away from amyloid and tau alone and toward synapses and connectivity as potential targets.

Meanwhile, a separate line of research points back the other way: tau, a hallmark of Alzheimer’s, may also play a role in autism.

In San Francisco, scientists at the Gladstone Institutes reported in the journal Neuron in 2020 they were able to prevent core autism symptoms in mice that model some of the more severe presentations of the condition by decreasing levels of tau by 50 percent. Follow-up studies that will be published shortly showed that this effect was not temporary but lasted for a lifetime, said Lennart Mucke, the lead author of the study and a neuroscience professor at the University of California at San Francisco.



Lennart Mucke, a neuroscience professor at the University of California at San Francisco, is surrounded by other scientists at the Gladstone Institutes. (University of California at San Francisco)

In the brain, tau acts as a kind of regulator of what Mucke called brain cell "excitability." Reducing it, he said, may help the brain "cool off" or suppress a pathway that is overactive and creates abnormal brain connections.

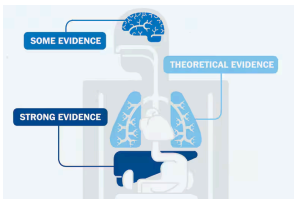
"Imagine an orchestra. … You want everyone to play in harmony," said Mucke, director of the Gladstone Institute of Neurological Disease. "If the conductor fails, there's dysregulation."

The work ahead in both autism and Alzheimer's, he said, is learning how to bring that harmony back.

Most read Health

We may have less control over how long we live than previously thought

He couldn't think, eat or recover — and no one knew why



13 surprising ways GLP-1s may benefit the body, according to science

View 2 more stories

By Ariana Eunjung Cha
Ariana Eunjung Cha is a national reporter. She has previously served as The Post's bureau chief in Shanghai and San Francisco, and as a correspondent in Baghdad. @arianaeunjung