

# Brain changes following traumatic brain injury share similarities with Alzheimer's disease

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The image displays a TBI-affected brain's white matter connectivity as inferred using diffusion tensor imaging and streamline tractography. The brain surface is rendered as a translucent layer to provide anatomical context for the streamline display. White matter connections and the brain surface are displayed using different colors across for the same subject. Credit: Kenneth Rostowsky

Brain changes in people with Alzheimer's disease and in those with mild traumatic brain injuries (TBIs) have significant similarities, a new USC study shows, suggesting new ways to identify patients at high risk for Alzheimer's. The findings appear this week in *GeroScience*.

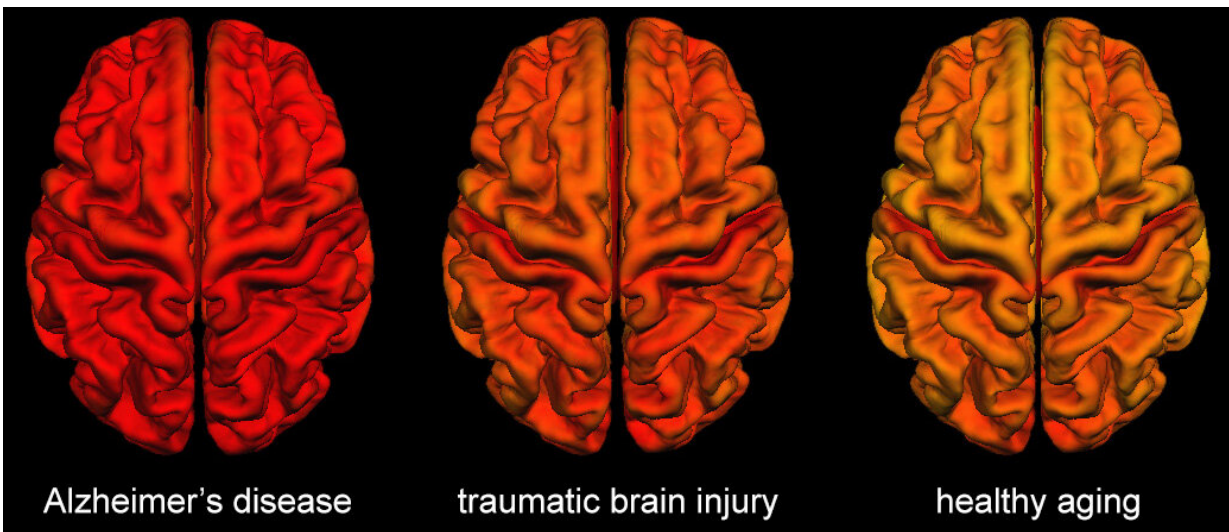
TBIs, which affect over 1.7 million Americans every year, are often followed by changes in brain structure and function and by cognitive problems such as memory deficits, impaired social function and difficulty with decision-making. Although mild TBI—also known as concussion—is a known risk factor for Alzheimer's disease, prior studies haven't quantified the extent to which these conditions share patterns of neural degeneration in the brain.

USC researchers hypothesized that comparing these patterns could reveal not only how the degenerative trajectories of the two conditions are similar but also which features of brain atrophy could predict Alzheimer's risk after TBI.

The study included 33 study participants with TBIs due to a fall, another 66 participants who had been diagnosed with Alzheimer's disease and 81 healthy control participants without either TBI or Alzheimer's. The researchers analyzed MRIs of the patients' brains and created additional computer-generated models to compare dozens of different brain structures, ultimately mapping similarities and differences between the three different groups.

In multiple [brain areas](#) of both TBI and Alzheimer's participants, the researchers found reduced cortical thickness when compared to the healthy controls. Cortical thickness is roughly correlated with brain age and its thinning is often associated with reductions in attention, memory and verbal fluency, as well as with decreased ability to make decisions, integrate new information and adapt one's behavior to new situations, among other deficits.

"These findings are the first to suggest that cognitive impairment following a [traumatic brain injury](#) is useful for predicting the magnitude of Alzheimer's-like brain degradation," said study author Andrei Irimia, an assistant professor of gerontology, neuroscience and biomedical engineering at the USC Leonard Davis School of Gerontology and the USC Viterbi School of Engineering. "The results may help health professionals to identify TBI victims who are at greater risk for Alzheimer's disease."



Displayed is the cortical thickness for each group studied (Alzheimer's disease, traumatic brain injury and healthy aging) after mapping each participant's cortical surface to an atlas average. The color scale ranges from bright red (thinner cortex) to yellow (thicker cortex). Healthy participants have the thickest cortex, whereas patients with Alzheimer's disease exhibit pronounced cortical thinning, which is a hallmark of the disease. As expected, TBI patients fall in between the two groups based on their typical cortical thickness. This reflects their faster rate of cortical thinning, which is accelerated by injury and by subsequent pathological processes. Such accelerated cortical thinning is illustrative of TBI patients' higher risk of neurodegeneration along Alzheimer's disease-like trajectories. Credit: Kenneth Rostowsky, USC

Using MRIs, the study identified significant similarities between TBI and Alzheimer's disease in how the brain's gray and [white matter](#) degrade after injury. In [gray matter](#)—the part of the brain that contains neuron cell bodies and their short-range connections—the most extensive similarities were in areas involved in memory (temporal lobes) and decision-making (orbitofrontal cortices).

In white matter—which connects different brain regions and allows their neurons to communicate across longer distances—the researchers found comparable degeneration patterns in structures such as the fornix, corpus callosum and corona radiata. Whereas the fornix is involved in memory function, the corpus callosum facilitates information exchange between brain hemispheres. The corona radiata is involved in limb movement, and its injury can lead to poorer coordination and balance.

The scientists also used machine learning techniques to accurately predict the severity of Alzheimer's-like [brain changes](#) observed during the chronic stage of mild TBI based on cognitive assessments conducted shortly after such injuries.

At least 15% of Americans have a history of TBI. Chronic TBI effects on cognitive function may be particularly severe in older people, who are approximately three times more likely to sustain a TBI than other age groups.

Studies of TBI effects on [brain structure](#) have identified both amyloid plaques and neurofibrillary tangles—twisted fibers found inside the brain's cells—which resemble those observed in Alzheimer's disease. Despite this evidence, the study authors said, few studies have investigated whether TBI can alter brain trajectories toward Alzheimer's, particularly at older ages.

The new findings do not establish a cause-and-effect relationship

between TBI and Alzheimer's disease but do add to the evidence that the two conditions share common trajectories, researchers said. The study, which was co-authored by USC alumnus Kenneth Rostowsky, is a follow-up to the team's earlier study outlining TBI-related changes in [brain](#) function.

**More information:** Rostowsky, K.A., Irimia, A. & for the Alzheimer's Disease Neuroimaging Initiative. Acute cognitive impairment after traumatic brain injury predicts the occurrence of brain atrophy patterns similar to those observed in Alzheimer's disease. *GeroScience* (2021). [doi.org/10.1007/s11357-021-00355-9](https://doi.org/10.1007/s11357-021-00355-9)

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