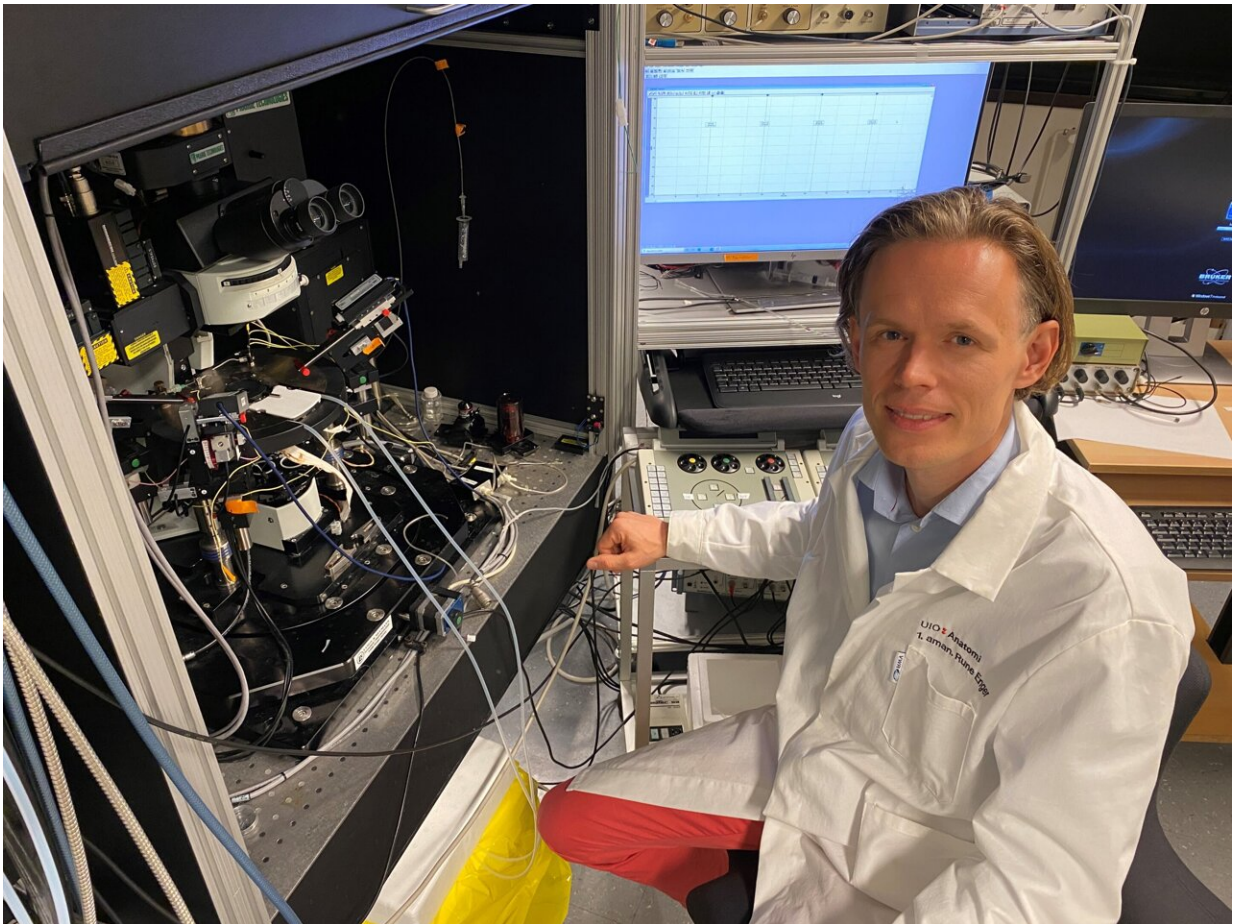


Alzheimer's disease: Reduced signals in the brain's forgotten cells

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Associate Professor Rune Enger at the Institute of Basic Medical Sciences at the University of Oslo. Credit: Cecilie Bakken Høstmark, UiO

Alzheimer's disease is associated with amyloid plaques. Nerve cells are destroyed and brain functions fail. There is still a lot we do not know about the disease. Why does it become more difficult to remember? Why are you no longer able to maintain your levels of attention and concentration?

A group of scientists at the Institute of Basic Medical Sciences at the University of Oslo have found a new piece of the puzzle in understanding how the brains of Alzheimer's patients work. The hope is that the findings can lay the foundation for developing new ways of treating Alzheimer's disease in the future.

"We use advanced laser microscopes ... that now allow us to look into the brains of awake Alzheimer's mice. In previous studies, scientists have studied mice that have been anesthetized," says Rune Enger. He is an associate professor at the Letten Center which is part of the Institute of Basic Medical Sciences at University of Oslo.

The scientists studied pupil responses, behavior and activity in the brain

In a new study, Enger and colleagues have studied types of brain cells called astrocytes. They are important handymen in the brain that are located around the nerve cells and act as support cells. The cells make it possible for the nerve cells to function normally.

"Astrocytes use calcium signals inside the cell to communicate. It is thought that these signals can help coordinate nerve cell activity over a large area," explains Enger.

The scientists studied several things: what happened to the pupils of the mice with Alzheimer's when they moved, what the mice did at the same

time and what happened inside the brain. Using genetically encoded nanosensors that light up where there is activity in the brain, they were able to study the calcium signals produced by the astrocytes. They could then compare what happened in Alzheimer's mice with healthy mice.

"Among other things, the mice received a sudden puff of air on their faces so that we could see what happened when they were startled," Enger says.

Calcium signals in astrocytes were reduced in Alzheimer's mice

Because the scientists were able to study mice that were not anesthetized but were free to move around, a slightly different picture emerged of what happens in the brain than in previous studies.

"Scientists who studied anesthetized Alzheimer's mice saw that activity in these cells increased. At the same time, we know that anesthesia influences astrocytes. In the new study, we found that it was not a given that activity increased. Instead, we saw that calcium signals in the astrocytes were weaker when the mice ran about and when they were startled compared to healthy mice. The astrocytes in the sick mice were also significantly different from the healthy mice in that they were larger and had altered the shape and expression of specific proteins linked to inflammation," says Enger.

Likely that the connections between astrocytes and concentration centers become damaged

"One can imagine that the role of astrocytes is a bit like the volume knob on a radio that can affect many [nerve cells](#) at the same time. In the Alzheimer's mice, this mechanism seems to be disrupted. The reduced

activity of astrocytes may be due to the connection being damaged between these cells and one of the brain's systems related to stress and concentration," says the associate professor.

He envisions a possible future drug that could help people with Alzheimer's disease.

"Perhaps in the future, we can use drugs that affect calcium activity in [astrocytes](#) in order to affect [brain](#) function in those with this diagnosis," Enger says.

The research was recently published in *eLife*.

More information: Knut Sindre Åbjørsbråten et al, Impaired astrocytic Ca²⁺ signaling in awake-behaving Alzheimer's disease transgenic mice, *eLife* (2022). [DOI: 10.7554/eLife.75055](https://doi.org/10.7554/eLife.75055)

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