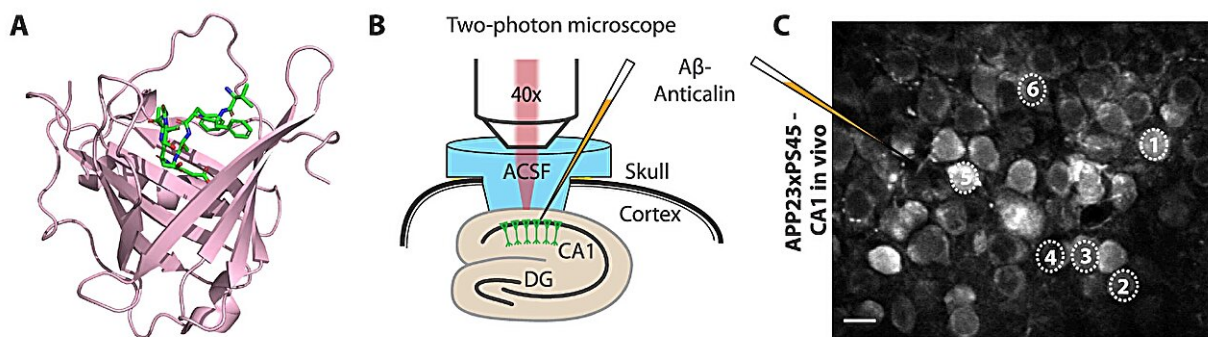


# Scientists target amyloid beta molecule in search for preventive treatment strategy for Alzheimer's

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Aβ-anticalin treatment suppresses neuronal hyperactivity in vivo. A) Structure of the Aβ-anticalin. B) Two-photon imaging setup. C) Representative two-photon image of the pyramidal layer of the hippocampal CA1 region. Credit: *Nature Communications* (2024). DOI: 10.1038/s41467-024-50153-y. *Nature Communications* (2024). DOI: 10.1038/s41467-024-50153-y

In the fight against Alzheimer's, researchers at the Technical University of Munich (TUM) have developed a promising, preventative therapeutic approach. They specifically targeted the amyloid beta biomolecule, which triggers the hyperactivity of nerve cells typical of the brain disease in its early stages.

The team led by Dr. Benedikt Zott and Prof. Arthur Konnerth from the

TUM School of Medicine and Health and Prof. Arne Skerra from the TUM School of Life Sciences succeeded in developing and using a [protein drug](#) that can suppress the effects of the harmful molecule.

The study is [published](#) in *Nature Communications*. The results obtained on mice in the laboratory indicate that neuronal dysfunctions could even be repaired. The researchers hope that the protein they investigated, which experts call amyloid-beta-binding anticalin (H1GA), can halt the progression of the severe neurodegenerative disease at an early stage.

According to experts, there are an estimated 55 million people worldwide living with dementia, and most of them have Alzheimer's. Each year, about 10 million new cases are diagnosed. There is currently no medication to combat the basic mechanisms of the disease. Only symptoms such as declining mental performance can be treated.

Dr. Zott emphasizes, "We are still a long way from a therapy that can be used in humans, but the results in [animal experiments](#) are very encouraging. The effect of completely suppressing neuronal hyperactivity in the early stages of the [disease](#) is particularly remarkable."

The researchers obtained the anticalin H1GA by protein design and produced it in genetically modified bacteria of the species *Escherichia coli*. The active ingredient was injected directly into the hippocampus region of the brain. Regarding measurable behavior, the previously hyperactive brain cells could no longer be distinguished from healthy nerve cells.

It is still unclear whether the effect can be achieved in [human patients](#) outside the laboratory. In any case, a more effective form of administration of the active ingredient is currently being developed.

In 2016, the active substance solanezumab, which was supposed to have a similar effect, proved to be a failure in large-scale [clinical trials](#); this can be explained by its different molecular structure.

Zott and his colleagues also directly compared their new [active ingredient](#) with solanezumab in the trials. H1GA showed more evident positive effects.

**More information:** Benedikt Zott et al,  $\beta$ -amyloid monomer scavenging by an anticalin protein prevents neuronal hyperactivity in mouse models of Alzheimer's Disease, *Nature Communications* (2024). [DOI: 10.1038/s41467-024-50153-y](https://doi.org/10.1038/s41467-024-50153-y)

Provided by Technical University Munich

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