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Deutsches Zentrum für Neurodegenerative Erkrankungen e.V. (DZNE)
Dr. Marcus Neitzert

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Lab Findings Support the Concept that Reducing Neuroinflammation Could Help Fight Alzheimer's

Scientists from DZNE, University Hospital Bonn (UKB) and the University of Bonn provide new evidence that preventing brain inflammation is a promising approach for the treatment of Alzheimer's disease. Their findings, based on studies in cell culture, mice and tissue samples from patients, may contribute to the development of more effective therapies. They are published in the scientific journal "Immunity".

Alzheimer's disease, the most common form of dementia, is associated with the deposition of proteins in the brain. The aggregation of these proteins, which are known as "amyloid-beta", give rise to a chain of events that ultimately harm neurons and lead to their loss. "Alzheimer's disease involves a complex interaction of different mechanisms. One of these is neuroinflammation. That's what we looked at in our studies. Specifically, we pharmacologically manipulated a molecular complex called the NLRP3 inflammasome. It is found in microglia, which are the immune cells of the brain," says Dr. Róisín McManus, a DZNE research group leader, investigator at UKB's Institute of Innate Immunity and also a member of the "ImmunoSensation2" Cluster of Excellence at the University of Bonn.

Previously unknown pathways

The "NLRP3 inflammasome" is like a control switch: In Alzheimer's disease, its activation triggers an inflammatory response that harms neurons. For this reason, researchers have been exploring ways to inactivate this molecular complex using drugs. The current results support this approach. "It is known that inhibiting NLRP3 not only reduces neuroinflammation, but also helps microglia clear the harmful amyloid-beta deposits, a process called phagocytosis. The novelty of our findings is that they provide a better understanding of the important role that NLRP3 plays in microglia and we also unravel the mechanism behind why its inhibition is so beneficial", says McManus. "In our studies we have identified previously unknown signaling pathways influenced by NLRP3. In particular, we found that NLRP3 regulates how microglia use nutrients and how these act on genes that have a major impact on the function of microglia. This is very relevant for their ability to carry out phagocytosis. These findings could help in the development of therapies for dementia. In any case, our research shows that NLRP3 is a promising target for the treatment of Alzheimer's disease."

International endeavor

In this project, the Bonn-based researchers collaborated with the Luxembourg Centre for Systems Biomedicine, University of California San Diego, Technische Universität Braunschweig, Novartis Switzerland and other institutions in Europe and beyond.

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About Deutsches Zentrum für Neurodegenerative Erkrankungen, DZNE (German Center for Neurodegenerative Diseases): DZNE is one of the world's leading research centers for neurodegenerative diseases such as Alzheimer's, Parkinson's and ALS, which are associated with dementia, movement disorders and other serious health impairments. These diseases place an enormous burden on patients and their families, but also on society and the economy of healthcare. DZNE contributes significantly to the development and translation into practice of novel strategies for prevention, diagnosis, care and treatment. DZNE comprises ten sites across Germany and collaborates with universities, university hospitals, research centers and other institutions in Germany and throughout the world. DZNE is state-funded and a member of the Helmholtz Association and of the German Centers for Health Research.
https://www.dzne.de/en

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