Modulating Blood Vessel Growth in Alzheimer's Disease

https://neurodegenerationresearch.eu/survey/modulating-blood-vessel-growth-in-alzheimers-disease/ **Principal Investigators**

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Modulating Blood Vessel Growth in Alzheimer's Disease

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5

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Research Abstract

Alzheimer's disease (AD) is a condition where patients experience loss of neurons in the brain, resulting in memory loss and cognitive decline. With AD being the most common form of dementia and as the population ages, this disease is a serious burden for patients and caretakers alike. It has been shown that AD patients have deposits or plaques comprised of a protein called amyloid in their brains. It is thought that amyloid could be responsible for the neuronal death seen in AD patients, as well as for altering the blood vessels in the blood-brain barrier, a structure that filters and protects the brain from regular blood circulation. Recently, we have reported that amyloid causes an increase in the growth of blood vessels in the brain of AD mouse models, and this phenomenon is also seen in AD patients. The uncontrolled sprouting of

blood vessels can lead to increased leakiness in the blood vessels, allowing unwanted blood products to enter the brain and worsen the disease. Moreover, other conditions that arise due to impaired blood vessels, such as stroke, heart disease and high cholesterol, have been implicated as risk factors for AD. Our key objective is to examine how blood vessels change throughout the course of the AD, specifically looking at the loss or reorganization of proteins that form the structural basis of the blood-brain barrier. We will then attempt to curb the damage seen in the brains of AD model mice and improve cognition by treating them with drugs that prevent the formation of new blood vessels. A fundamental aspect of this proposal is that these drugs are already approved for use in humans, expediting the translational potential of this research into clinical trials in human patients.

Further information available at:

Investments < €500k
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