# In vivo permeability of the human bloodcerbrospinal fluid barrier in dementia

https://neurodegenerationresearch.eu/survey/in-vivo-permeability-of-the-human-blood-cerbrospinal-fluid-barrier-in-dementia/

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Contact information of lead PI Country

USA

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In vivo permeability of the human blood-cerbrospinal fluid barrier in dementia

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## **Research Abstract**

DESCRIPTION (provided by applicant): The choroid plexus (CP) forms an interface between the blood and cerebrospinal fluid (CSF) and works in concert with brain capillaries to assure fluid homeostasis throughout the brain. Bounded on one side by ventricular CSF and on the

other by a dense, highly permeable vascular network, the CP contains the blood- CSF barrier, a single layer of epithelial cells that secrete the majority of CSF in the brain and, by virtue of tiht intercellular junctions, regulates the exchange of macromolecules between the blood and CSF. Once across the barrier, solutes are transported to the surface of the brain where they are absorbed into the venous blood or mix with interstitial fluid and are carried deep into the parenchyma. Alzheimer's disease (AD) is the most commonly diagnosed form of dementia in the elderly and the accumulation of amyloid-? peptides the histopathological hallmark of the disease. Amyloid-? levels in the CP increase in AD and could alter the permeability of the CP. We hypothesize that permeability disturbances could have large effects on CSF hydrodynamics and increase concentrations of amyloid-? throughout the brain. The proposed project will use ultra-high field dynamic contrast enhanced magnetic resonance imaging and a compartmental tissue model that explicitly accounts for intercompartmental water exchange to quantify CP permeability in vivo and determine the extent to which it is associated with cognitive function in early AD. We expect that the accurate, non-invasive measurement of CP permeability, together with cognitive assessments, will provide critical insight into the role of the blood-CSF barrier in the cognitive decline that characterizes AD. Such information will be important in understanding the pathophysiology of AD and the rational treatment of incipient disease.

## Further information available at:

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