Brain-Blood Barrier might influence progression of Alzheimer's

Contributed by Brain22
Thursday, 01 October 2015

More and more data from preclinical and clinical studies strengthen the hypothesis that immune system-mediated actions contribute to and drive pathogenesis in Alzheimer's disease. The team of Roosmarijn Vandenbroucke in the Claude Libert Group (VIB/UGent) combined their knowledge and expertise related to inflammation with the expertise in Alzheimer's disease present in the Bart De Strooper Group (VIB/KU Leuven). This collaboration lead to the insights that A² indeed induces a strong inflammatory response, thereby destroying an important but often neglected brain barrier, called the blood-cerebrospinal fluid (CSF) barrier. Disruption of this blood-CSF barrier disturbs brain homeostasis and might negatively affect disease progression. Strikingly, these effects could be blocked in the presence of a matrix metalloproteinase (MMP) inhibitor.

Roosmarijn Vandenbroucke: "Although further research is needed, these data suggest that blocking MMP activity or upstream inflammatory signalling, might have therapeutic potential to treat Alzheimer's disease. It is important we could demonstrate the role of the blood-cerebrospinal fluid barrier, because this would be an easier target to reach in comparison with the targets of current therapies."

The publication of Vandenbroucke et al. was picked up by Alzforum.org who combined it together with another publication about the Blood-Brain Barrier: Barriers Between Blood and CSF, Brain Yield to A² - Not a Bad Thing? 18 Sep 2015.

The barrier between the blood and central nervous system crumbles in Alzheimer's disease, but researchers have known little about how this happens, or what it does to brain pathology. Two new papers shed some light on how A² damages the cells that protect the brain parenchyma and cerebrospinal fluid. The studies examine different systems and describe distinct mechanisms, but both add to the picture of what may happen in disease.