

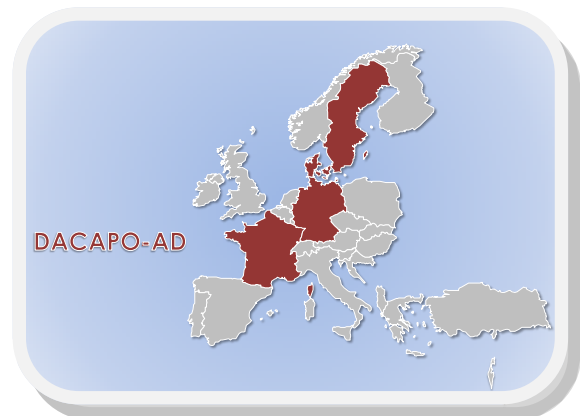
DACAPO-AD

Deciphering Interactions of Acquired Risk Factors and ApoE-mediated Pathways in Alzheimer's Disease

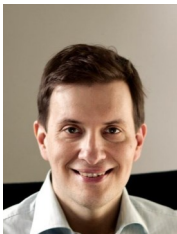
Alzheimer's disease (AD) imposes an enormous personal burden on patients and caregivers, as well as a tremendous socio-economic impact on society. However, there is a paucity of pharmaceutical or interventional strategies that have a proven impact on the incidence or progression of AD. One reason is that most models are based on familial (early-onset) AD pathogenesis, but typically do not reflect the multifactorial pathophysiology of sporadic (late-onset) AD, which is associated with genetic risk factors such as the apolipoprotein E (ApoE) ϵ 4 polymorphism, as well as environmental risk factors, such as a high-fat diet, cardiovascular disease, traumatic brain injury, systemic inflammation and perturbed sleep regulation.

We aim to create novel AD models that combine the most common genetic risk factor (ApoE- ϵ 4) and many of the most prevalent acquired risk factors, enabling us to better understand the multifactorial and highly prevalent, yet currently understudied, interplay between inherited and acquired risk factors in the pathophysiology of late-onset AD. These models may therefore be more predictive of possible translatability into clinical studies, and may potentially lead to the development of new avenues of primary prevention or treatment.






Start Date: January 2016
Duration: 3 years
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