

P3-460: Cognitive impairment acceleration after late-life depression in a model of Alzheimer's disease

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Background: Clinical studies suggest that depressive symptoms could be considered an important risk factor for the future development of cognitive impairment and even Alzheimer's disease (AD). In fact, there is a strong association between depression in later life and AD. The age of onset of AD has been shown to be accelerated in patients with mild cognitive impairment (MCI) with a history of depression, and women appear to be particularly more vulnerable to this condition. In addition, individuals with MCI who present with depressive symptoms have an elevated burden of amyloid-beta ($A\beta$), the main toxic protein associated with Alzheimer's pathology, and a higher risk of developing AD compared to non-depressed MCI patients. However, it is unknown whether depression can be considered a risk factor for the development of AD. Although it has been described that some transgenic models of AD can develop signs similar to depression in advanced stages, the induction of Alzheimer's pathology due to a depressive process has not been studied under experimental conditions to emulate late-life depression as a risk factor for AD.

Method: In this study, we induced chronic unpredictable mild stress (CUMS) in P301S tau transgenic mice to determine whether depression is a cause, rather than a consequence, of the development of AD.

Result: The results of our study indicate that the induction of CUMS in transgenic animals of the disease give rise to changes in depressive state of the animals.

Conclusion: The findings generated in this project could provide evidence of depression as a risk factor for AD, its mechanisms of action, use as early biomarkers, as well as the discovery of new therapies for AD.

Words: Depression, stress, Alzheimer's disease