Poster Certificate

This is to certify that the poster entitled:

The effect of different amyloid seeds and animal hosts on amyloid propagation in Alzheimer's Disease

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THE EFFECT OF DIFFERENT AMYLOID SEEDS AND ANIMAL HOSTS ON AMYLOID PROPAGATION IN ALZHEIMER'S DISEASE

POSTER SESSION 06 - SECTION: ALZHEIMER'S AND TAUOPATHIES

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Abstract text: Alzheimer's Disease is a neurodegenerative proteinopathy in which recent evidence indicates that Aβ can misfold and aggregate into seeds that structurally corrupt native proteins, mimicking a prion-like process of template protein corruption or seeding. In fact, studies show that Aβ deposition can be induced by the intracerebral infusion of seed-containing brain homogenates, and that the characteristics of both the seeding agent and the host, influence the pathologic signature of the Aβ seeds. However, it is still unknown which Aβ-misfolded species are most efficient in triggering the aggregation process and which is the effect of amyloid seeds on different AD models. Methods: Amyloid seeds from AD patients (stage C for amyloid) from the Alzheimer's Disease Research Center (ADRC) at UCI were administered into 7-8-month-old hAβ-KI mice and 3xTg-AD mice. Next, we intracerebrally injected brain homogenates from the human AD patient and 25mo-3xTg-AD mice into the hippocampus of 7-month-old 3xTg-AD mice, which were analyzed at 18 months of age. Results: Our findings demonstrated that amyloid deposition differentially occurs in 3xTg-AD and hAβ-KI mice, and the Aβ aggregates are developed earlier in the familial model. Moreover, the amyloid seeds from the human patient induce more aggressive amyloid pathology compared to seeds from aged 3xTg-AD mice. Conclusion: These results suggest that multiple factors such as the seed, recipient model and time are critical factors that can modulate the amyloid pathology onset and progression. Thus, more profound understanding of these factors will provide key insight on how amyloid pathology progresses in AD.