Poster Certificate

This is to certify that the poster entitled:

WAT alterations in diabetic mice: its connection and implication in AD pathogenesis

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At the:

FENS Forum 2022

9-13 July 2022 | Paris, France

Jean-Antoine Girault

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FENS Forum 2022
9-13 July 2022 | Paris, France

BOARD NUMBER: S05-194

WAT ALTERATIONS IN DIABETIC MICE: ITS CONNECTION AND IMPLICATION IN AD PATHOGENESIS

POSTER SESSION 05 - SECTION: ALZHEIMER'S DISEASE: FROM NEUROINFLAMMATION TO NEUROPROTECTION

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Alzheimer's disease (AD) is a complex disorder and multiple cellular and molecular mechanisms are involved in AD onset and progression. Recent evidences have suggested that metabolic alterations are an important pathological feature in disease progression in AD. Likewise, diabetes and obesity, two mayor metabolic illnesses associated with white adipose tissue expansion, are risk factors for AD. Here, we hypothesize that the white adipose tissue may serve as a key communicator organ between the brain and peripheral metabolic illnesses. We used histological stains, immunohistochemistry and biochemical means to determine changes in the white adipose tissue from WT and db/db mice. Moreover, similar techniques were used in the brain of 3xTg-AD mice that received white fat pads from WT and db/db donors to determine any changes in amyloid and tau pathology. Our study shows that recipient 3xTg-AD mice from db/db fat pads mice develop profound changes in tau pathology due to increased CDK5/p25 expression compared to 3xTg-AD mice that received fad pads from WT mice. This increment in tau level was associated with elevated levels in IL-1β and microglial activation. However, we found that Aβ levels were reduced in recipient 3xTg-AD mice from db/db fat pads compared to 3xTg-AD mice that received fad pads from WT mice. These reduction in Aβ levels were correlated with an increment in microglia phagocytic capacity. Overall, our study demonstrates a novel important crosstalk between AD and diabetes type II through white adipose cells and a differential effect on tau and Aβ pathology.