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Visceral adipose tissue triggers tau pathogenesis in transgenic mice

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Abstract Text:

Background: 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, are risk factors for AD. These two overwhelming diseases are associated with a significant expansion of visceral adipose tissue. Here, we hypothesize that the visceral adipose tissue may serve as a key communicator organ between the brain and peripheral metabolic illnesses and affecting both types of disorders.

Method: We used histological stains, immunohistochemistry and biochemical means to determine changes in the visceral adipose tissue from WT and db/db mice. Moreover, similar techniques were used in 3xTg-AD mice that received white fat pads from WT and db/db donors to determine any changes in amyloid and tau pathology.

Result: Our study shows that recipient 3xTg-AD mice from db/db fat pads mice develop profound changes in tau pathology due to increased CDK5 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-1 β and profound microglia activation. Moreover, we found the opposite effect on amyloid pathology, in which insoluble A β levels and Thioflavin positive plaques 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 A β levels were correlated with an increment in microglia phagocytic capacity.

Conclusion: Overall, our study demonstrate a novel important crosstalk between Alzheimer's disease and obesity/diabetes type II through visceral adipose cells and a differential effect on tau and A β pathology mediated by an activated immune response.

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Preferred Presentation Method:

In-Person

Was this research funded by an Alzheimer's Association grant?

Yes

Abstract Submission Affirmations:

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Do you plan to upload figures or tables to supplement your abstract text?

No

Theme:

Basic Science and Pathogenesis

Topic:

Molecular and Cell Biology

Sub Topic:

Tau

Learning Objectives:

Determine the involvement of adipose tissue in the development and progression of Alzheimer's disease

Keywords:

inflammation, microglia and tau

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Yes. The presenting author has read the Fellowship guidelines above, understands them, and agrees to accept the stated conditions.

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Any relevant financial relationships? No

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