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**International Conference on
Alzheimer's and Parkinson's Diseases
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POSTERS: A01.J. DISEASE MECHANISMS, PATHOPHYSIOLOGY: ASTROGLIA

ASTROGLIAL REACTIVITY IN RESPONSE TO B-AMYLOIDOSIS IS ASSOCIATED WITH MITOCHONDRIAL PATHOLOGY IN THE HIPPOCAMPUS OF ALZHEIMER'S TRANSGENIC MICE

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Aims: Alzheimer's disease (AD) is associated with early energy hypometabolism, synaptic and mitochondrial dysfunction, oxidative stress, inflammation, abnormal proteostasis and progressive neurodegeneration. During the pathogenic process, amyloid-beta and phospho-tau pathologies have a detrimental effect on neurons and glial cells, affecting neuronal stability, and compromising ATP production and energy metabolism. Though mitochondrial dysfunction is thought to be an early event in the pathogenesis of this AD, the vast majority of studies have focused on neurons, and little is known about the functional characteristics and dynamics of mitochondria in astrocytes. Here, we aim to analyze mitochondrial subcellular features of reactive astrocytes in APP/PS1 mice hippocampus in order to a better understanding of this pathology.

Methods: Mitochondrial features were observed by transmission electron microscopy and immunogold labeling. Image analysis was performed to assess morphological changes.

Results: Our results show mitochondrial structural alterations including mitochondrial cristae loss, broken double membrane structure and fragmentation. In addition, an increase in both number and size of mitochondria in this transgenic model compared to age-matched WT mice, was found.

Conclusions: Since mitochondrial morphology is directly related to mitochondrial fusion/fission, the ultrastructural pathology of astrocytic mitochondria in this amyloidogenic model suggests dynamics abnormalities in these organelles that might lead to astroglial functional deficits compromising neuronal survival. *Supported by ISCIII grants PI21-0915 (AG) and PI21-00914 (JV) co-financed by FEDER funds from European Union, by Junta de Andalucía grants P18-RT-2233 (AG) and US-1262734 (JV) co-financed by Programa Operativo FEDER 2014-2020, and by grant PPIT.UMA.B1-2021_32 (LTE).*