

POSTERS

DIVERSITY OF PLAQUE-ASSOCIATED MYELOID CELLS SUBTYPES IN HUMAN ALZHEIMER'S DISEASE BRAIN

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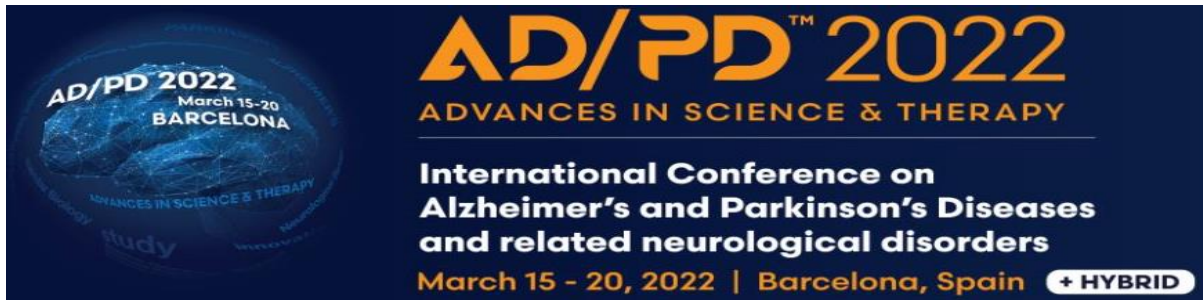
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Aims: Parenchymal microglia, as well other myeloid cells, have been postulated as a critical factor in Alzheimer's disease (AD) pathogenesis since the identification of genetic risk factors related to their functions. However, the different phenotypes and the implication of the diverse immune cells in the human pathology have not been determined yet. In this work, we have further analyzed the phenotypic profile of the damage-associated myeloid cells in two AD vulnerable brain regions, the frontal cortex and hippocampus.

Methods: Immunohistochemistry and image analysis approaches have been carried out in postmortem brain samples from patients with AD (Braak V-VI) and aged controls without neurological symptoms (Braak II).

Results: Damage-associated microglial cells were clustered around amyloid plaques and expressed Iba1, TMEM119, CD68, Trem2 and CD45^{high}. Moreover, AD brains exhibited parenchymal infiltration of CD163-positive monocyte-derived cells that invaded plaque near blood vessels. While the frontal cortex showed strong microglial activation similarly to that reported in amyloidogenic mice, the hippocampus of the same patients showed an attenuated microglial activation with a degenerative phenotype.

Conclusions: These findings suggest the existence of different myeloid populations associated with A β plaques that correlates with disease severity. These results open the opportunity to design targeted therapies, not only to microglia, but also to the population of macrophages to modulate amyloid pathology and provide a better understanding of the immunological mechanisms underlying AD progression. Supported by ISCiii of Spain grants PI18/01557 (AG), PI18/01556 (JV) co-financed by FEDER funds from EU, and by Junta de Andalucía grants UMA18-FEDERJA-211(AG), P18-RT-2233(AG) and US-1262734(JV) co-financed by Programa Operativo FEDER 2014-2020.



POSTER CERTIFICATE

This is to certify that

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presented the abstract entitled

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A handwritten signature in black ink, which appears to read 'Abraham Fisher'.

Abraham Fisher

President