IJAE
Vol. 121, n. 1 (Supplement): 40, 2016
ITALIAN JOURNAL OF ANATOMY AND EMBRYOLOGY

Membrane protein remodeling in microglia exposed to amyloid peptides

Cinzia Fabrizi [1] - Elena Pompili [1] - Bruno Maras [2] - Giuseppina Mignogna [2] - Laura Di Francesco [2] - Virginia Correani [2] - Maria Eugenia Schininà [2] - Lorenzo Fumagalli [1]

[1] Dipartimento di Scienze Anatomiche, Istologiche, Medico Legali e dell'Apparato Locomotore, Sapienza - Università di Roma, Roma, Italia - [2] Dipartimento di Scienze Biochimiche, Sapienza - Università di Roma, Roma, Italia

Infection, neurodegeneration, and other conditions associated with loss of brain homeostasis, induce changes in microglial morphology, gene expression and function, generally referred to as "activation". Alzheimer's disease (AD) is the most common dementia and is characterized by neuroinflammatory changes, including alterations in the morphology and distribution of microglia and astrocytes, and deposition of complement and other inflammatory mediators. Our previous observations show that microglial cells challenged in vitro with amyloid peptides clustered and rounded up, dramatically changing their morphology. Besides, in these cells we observed the early acetylation and then the phosphorylation of STAT3 which is required for the expression of the epsilon isoform of 14-3-3, a marker of Abeta-activated microglia (1, 2). We applied affinity partitioning approach combined with high throughput mass spectrometric analysis in order to identify variation of proteins on plasma membrane of BV2 immortalized microglia upon treatment with amyloid peptides. By this method several proteins up- or down-regulated by amyloid treatment were identified in microglial plasma membrane. Among them annexins (5 and 7), IFITM3 and MARK3. These data have been confirmed in primary microglial cultures.

In microglia, plasma membrane plays a relevant role in the cross-talking with the external neuronal environment and in the resulting trophic or inflammatory response of these sentinel cells. As such, knowledge of the microglia responsiveness to beta amyloids in term of changes in its plasma membrane proteome is imperative for unveiling the molecular landscape in which AD occurs.

References

[1] Di Francesco et al. Proteomics 2012; 12(1):124-34.
[2] Eufemi et al. Neurochem Int 2015; 81: 48-56.

Keywords
Microglia; Alzheimer's disease; neurodegeneration.