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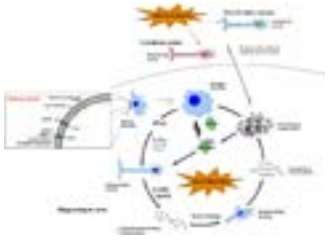
The neuroimmunomodulation theory in Alzheimer's disease

Alzheimer's disease (AD) is a progressive neurodegenerative disease, characterized by behavioral disorders, loss of memory and cognitive impairment affecting more than 48 millions worldwide. Cumulative evidence shows that innate immunity participates in the pathogenesis of AD. According with our neuroimmunomodulation hypothesis, microglial activation modifies the cross-talks between microglia and neurons. Thus, glial activation by the so called "damaged signals" triggers a cascade of pathological events leading to hyperphosphorylation and oligomerization of the tau protein, associated with cognitive impairment. This activation depends on the type and intensity of the stimulus. In AD, a persistently active microglial condition could generate neuronal damage and neurodegeneration favored by ApoE4, causing the release of pathological tau toward the extraneuronal environment. Released tau would subsequently cause reactivation of microglial cells, thus promoting a positive feedback and generating continuous cell damage. However, from the pathophysiological point of view, AD is significantly more complex that just inducing a loss of memory. As initial events in the pathogenesis of this neurodegenerative disease, alterations in the dopaminergic pathway together with serotonin depletion in the elderly lead to late onset depression according with recent evidences. These events seem to occur together with immunomodulatory alterations that lead to tau oligomerization in the course of neurofibrillary tangles formation. Interestingly, mood disorders are followed by neuroinflammatory processes and structural/functional alterations that lead to cognitive impairment in the context of AD (supported by Innova Corfo and the ICC).



Biography

Ricardo B. Maccioni is the Professor of Neurology and Neurosciences at University of Chile and the Director of the Laboratory of Neurosciences and the International Center for Biomedicine (ICC). He served as Professor at CU University, USA. He received Doctoral degree in 1975, was a postdoctoral fellow at the NIH and the CU Medical School. Maccioni is a world-class scientist that has made some of the leading contributions to the study of Alzheimer's disease (AD). His discoveries on the role of tau in AD, his neuroimmunomodulation theory of neurodegenerative disorders, the design of a new in vivo neuroimaging technology, novel biomarkers for its early diagnosis and innovative therapeutic approaches are among major contributions. He has served as Senior Editor and Regional Editor of the Journal of Alzheimer´s Disease and several other journals. He is co-author with George Perry of the book "Current Hypotheses and Research Milestones in Alzheimer´s Disease", among other 12 books. He is considered by the scientific community as a natural leader for his solid publications, but also for directing global projects for science development and human welfare. He is the author of around 200 publications in high-impact journals and 18 patents. His scientific findings are characterized by innovative approaches to elucidate complex problems of medical research. He is Scientific Director of Neuroinnovation, advisor for several international programs an elected member of the Danna Alliance for Brain Initiatives.

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