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Evaluation of the spontaneous activity of hippocampal neurons exposed to different concentrations of beta-amyloid (Aβ)

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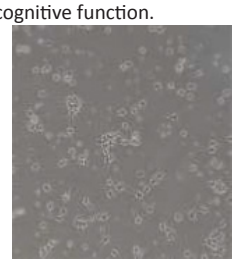
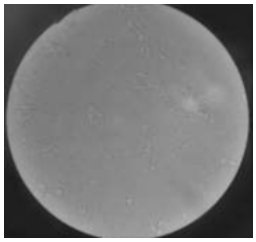
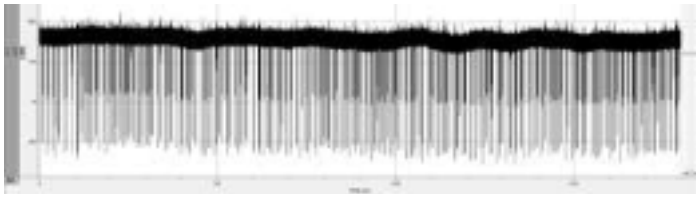
Abstract

Alzheimer's disease (AD) is a neurodegenerative disorder related to age and characterized by producing a cognitive deficit, specifically memory loss, and deterioration of learning processes. One of the main features of AD is the extracellular accumulation of beta-amyloid (Aβ), which can form beta-amyloid oligomers (AβO). These AβO applied ex vivo and in vivo act on the mechanisms underlying the learning processes, such as long-term depression (LTD) and long-term potentiation (LTP); facilitating the first (1,2) and deteriorating the second (3,4). In spite of the recent findings that relate the alterations of the neuronal synaptic activity with the deterioration of the cognitive processes, characteristic of AD; there is little or nothing that is known about the synaptic cellular mechanisms that would explain this detriment. Given the determinant role of spontaneous activity in the maturation and maintenance of synapses, homeostasis, and plasticity (5,6,7), it is critical and interesting to study how it changes This type of basal activity in primary culture of hippocampal neurons exposed to different concentrations of AβO. Specifically, how the probability of release of neurotransmitters changes, the sensitivity of postsynaptic receptors, the expression of new postsynaptic receptors and the flow of current through these receptors. In the present work, we applied different concentrations of oligomeric beta-amyloid (AβO) on primary cultures of hippocampal neurons of rat embryos, followed by evaluation of spontaneous electrical activity, specifically excitatory postsynaptic potentials sEPSP through the patch clamp technique whole-cell mode, finding changes in amplitude and frequency of the sEPSP. Understand the effects of AβO on sEPSP, crucial for neuronal functioning and communication; it will allow us to elucidate the synaptic cellular bases behind the cognitive deterioration characteristic of AD, which will facilitate the typing of sEPSP patterns that contribute to improving the diagnosis of AD; as well as, the identification of new therapeutic targets that mitigate the deleterious effects of AβO on cognitive function.







Biography

Marcela Cuestas, from her beginnings as a scientist, has had a great interest in the cellular mechanisms behind the processes of synaptic plasticity related to the formation of memory and learning. Since one of the characteristic features of Alzheimer's disease (AD) is the deterioration of cognitive processes and that, the processes of synaptic plasticity underlie these. She and her group of collaborators decided to evaluate how the synaptic activity of hippocampal neurons changes in Alzheimer's models. Therefore, he has extensive experience in the electrophysiological evaluation of neuronal activity in vivo and in vitro using patch clamping techniques and field registration in Alzheimer's models. Its field of action seeks to contribute, both to the diagnosis of AD, through the typing of patterns of spontaneous synaptic activity; as to the treatment of AD, by identifying new therapeutic targets to mitigate the harmful effects of beta-amyloid accumulation on cognitive function. She hopes to continue making important contributions to the basic knowledge related to the disease and clinical application. As well as continuing to promote the development of Alzheimer's neurophysiology in Colombia.

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