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Alzheimer's β-amyloid-induced depolarization of skeletal muscle fibers: Implications for motor dysfunctions in dementia

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Abstract

Numerous findings obtained over the last decades suggest that accumulation of β-amyloid peptide (βAP) plays the central role in the pathogenesis of Alzheimer's disease. It is well established that βAP has wide range of toxic effects on neurons in vitro and in vivo, however the influence of βAP in the periphery and on various other types of excitable tissues, eg. skeletal muscle cells, is almost unknown despite the many non-cognitive and other extra-neuronal symptoms associated with Alzheimer's dementia. Here we utilized conventional electrophysiological technique to investigate the effects and mechanisms of βAP action on the resting membrane potential of frog skeletal muscle fibers. βAP in the range of concentrations from 10-6 to 10-8M produced slow, significant, reversible depolarization of muscle fiber membranes. The impact developed and was washed out faster at higher concentrations of βAP (10-6 - 10-7M). The effect of βAP was completely absent when applied in Na+-free Tris + solutions. βAP-mediated depolarization was also prevented by tetrodotoxin (10-5M) pre-treatment and rescued by tetrodotoxin after-treatment. These findings suggest that βAP-induced depolarization of skeletal muscle plasma membranes can significantly disturb the functioning of skeletal muscles and therefore contribute to motor dysfunction observed in Alzheimer's disease and other disorders associated with βAP accumulation. Copyright © 2009 S. Karger AG.

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Keywords

β-amyloid peptide, Alzheimer's disease, Depolarization, Gait disorders, Muscle