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Alterations of intracellular calcium regulation during aging and Alzheimer's disease in nonneuronal cells

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Because of its function as an intracellular messenger in many cells, calcium plays an important role in signal transduction. Changes in intracellular free calcium concentration occur in central neurons during aging and Alzheimer's disease (AD). It is possible that similar changes in peripheral cells could mirror or, at least parallel, similar abnormalities in the brain. Assuming that manifestations of the aging process and AD are also present outside the central nervous system, nonneuronal tissues like lymphocytes could be used to search directly for abnormalities in cellular calcium regulation in man. Consistent with observations of reduced depolarization-induced Ca2+ rises in dissociated neurons of aged mice, corresponding age-related changes of reduced mitogen-induced Ca2+ responses were observed both in mouse lymphocytes and, more importantly, in circulating human lymphocytes. With respect to AD, Ca2+ responses after stimulation were unaltered (compared to normal controls). In addition, freshly prepared human lymphocytes showed elevated mitogen-induced Ca2+ responses after exposure to beta-amyloid, the main component of senile plaques in AD. These findings again parallel our observations that this peptide amplifies the K(+)-induced Ca2+ rise in acutely dissociated mouse brain cells. Thus, the lymphocyte seems to be a valuable model to study the effects of beta-amyloid in man. In a preliminary study with AD-patients, sensitivity of the lymphocytes to beta-amyloid's effects on Ca2+ rise was reduced, an observation which was entirely unexpected. Nevertheless, such studies indicate lymphocytes may represent a promising candidate for a peripheral marker of AD and can contribute to the understanding of the disease process.

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