β-Amyloid protein enhances the mitogen-induced calcium response in circulating human lymphocytes

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The role of beta-amyloid in Alzheimer's disease and its cellular mechanism of action on neurons are still unclear. There is growing evidence that beta-amyloid or its fragment, 25-35, influence neuronal calcium regulation. To investigate the effects of beta-amyloid on calcium homeostasis in man we used peripheral human lymphocytes as a model system for central neurons. beta-Amyloid fragment 25-35 exposed to lymphocytes for 60 s elevates the phytohemagglutinin (PHA)-induced Ca2+ rise in a dose-dependent manner. Small effects were already seen at concentrations as low as 50 nmol/l. Similar effects were also observed with fragment 1-40, whereas fragments 1-28 or 12-28 did not affect the Ca2+ response after PHA stimulation. Our findings support the hypothesis of an enhanced calcium response as a general feature of beta-amyloid's neurotoxicity. The lymphocyte seems to be a valuable model to study this effect in man.

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