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**Membrane Pore Formation by Amyloid beta (25-35) Peptide**
NABIN KANDEL, SUREN TATULIAN, University of Central Florida — Amyloid (A$\beta$) peptide contributes to Alzheimer's disease by a yet unidentified mechanism. One of the possible mechanisms of A$\beta$ toxicity is formation of pores in cellular membranes. We have characterized the formation of pores in phospholipid membranes by the A$\beta_{25-35}$ peptide (GSNKGAIIGLM) using fluorescence, Fourier transform infrared spectroscopy (FTIR) and circular dichroism (CD) techniques. CD and FTIR identified formation of $\beta$-sheet structure upon incubation of the peptide in aqueous buffer for 2 hours. Unilamellar vesicles composed of a zwitterionic lipid, 1-palmitoyl-2-oleoyl-phosphatidylcholine (POPC), and 70% POPC plus 30% of an acidic lipid, 1-palmitoyl-2-oleoyl-phosphatidylglycerol (POPG), are made in 30 mM $CaCl_2$. Quin-2, a fluorophore that displays increased fluorescence upon $Ca^{2+}$ binding, is added to the vesicles externally. Peptide addition results in increased Quin-2 fluorescence, which is interpreted by binding of the peptide to the vesicles, pore formation, and $Ca^{2+}$ leakage. The positive and negative control measurements involve addition of a detergent, Triton X-100, which causes vesicle rupture and release of total calcium, and blank buffer, respectively.

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