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Membrane Pore Formation by Amyloid beta (25-35) Peptide
NABIN KANDEL, SUREN TATULIAN, Univ of Central Florida — Amyloid (A?) peptide contributes to Alzheimer's disease by a yet unidentified mechanism. One of the possible mechanisms of A?toxicity is formation of pores in cellular membranes. We have characterized the formation of pores in phospholipid membranes by the A?$_{-35}$peptide (GSNKGAIIGLM) using fluorescence and circular dichroism (CD) techniques. CD identified formation of ?-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. Calcium is removed from the external medium by a desalting column. Quin-2, a fluorophore that displays increased fluorescence upon Ca^{+}binding, is added to the vesicles externally. Addition of the peptide results in increased Quin-2 fluorescence, which is interpreted by binding of the peptide to the vesicles, pore formation, and Ca^{+}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. The pore forming activity of A?$_{-35}$was dependent on the lipid composition of the vesicles. The effect of membrane cholesterol on A?pore formation may explain the role of cholesterol in AD pathogenesis. Furthermore, combined with FTIR analysis of the structure of membrane pores formed by A?$_{-35}$at various contents of cholesterol, we will provide an experimentally determined structure-function relationship for this highly neurotoxic peptide.

Nabin Kandel
Univ of Central Florida

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