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Amyloid Beta peptide's effects on NMDA receptors in PC-12 cells

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Amyloid-Beta peptide (Aß), a causative agent in Alzheimer's disease (AD), is known to deposit on cell membranes, disrupt cell functions, and induce cytotoxic effects including over-production of reactive oxygen species (ROS) and disruption of calcium homeostasis. In this project we develop an immortalized neuronal-like cell line (PC-12 cells) for studies of the complex interaction of Aß with neuronal cell membranes, proteins, and signaling pathways relevant to AD. When stimulated with Nerve Growth Factor, PC12 rat pheochromocytoma cells acquire a neuronal phenotype, and unlike primary cells may be passed indefinitely, allowing investigators convenient continuous study of neuronal pathways relevant to AD. In addition to development of PC12 cells for study of AD related pathways, we investigate how local membrane physical property changes induced by oligomeric Amyloid-Beta 1-42($A\beta_{42}$) impact on primary functions of membrane proteins, including the N-methyl D-aspartate receptor (NMDA-R) and NADPH oxidase. A better understanding of this complex pathway may provide insight into the development of new therapies for the treatment of AD.