

Nikolaos K. Robakis

Nikolaos Robakis is the First A.P. Slaner Professor for Alzheimer's disease research and director of the Center for Molecular Biology and Genetics of Neurodegeneration, at the Mount Sinai School of Medicine of New York University. He obtained his undergraduate training in chemistry at the University of Thessaloniki, Greece, and his PhD in Biochemistry at New York University. He completed his postdoctoral training at the Roche Institute of Molecular Biology, Nutley NJ. He has worked on the gene expression of bacterial systems, on prion disorders, and on the neurodegeneration and genetics of Alzheimer's disease. His laboratory has been awarded numerous competitive research grants totaling millions of dollars from the National Institutes of Health (NIH), pharmaceutical companies, and non-profit organizations. He has been invited to lecture at many academic institutions and as main speaker at numerous national and international meetings. He has been on the editorial board of professional journals and on the Scientific Advisory Board of private organizations that fund AD and Aging research. Dr Robakis has received many awards including the NIH MERIT award, The McKnight Neuroscience Development award, the Zenith Award of the Alzheimer's Association, and the Distinguished Scientist Award of the Hellenic Medical Association of New York. He has served as research advisor to pharmaceutical companies including Merck Sharp and Dohme Research Laboratories (1992-1993) and Bristol-Myers Squibb Co (1991-1995). Many scientists trained in Dr. Robakis laboratory are currently faculty members in universities and other academic institutions or group leaders in pharmaceutical companies in USA and Europe.

Robakis and co-workers have been working on the molecular biology and genetics of AD for more than 20 years. His group was among the first to clone the APP gene that encodes the A β peptides and in 1987 they published the first report on the localization of APP on chromosome 21. His laboratory showed that APP is mainly cleaved by secretases intracellularly in post-Golgi vesicles and that APP also occurs in proteoglycan forms. In 2003 his group published an extensive study where it was shown that most presenilin familial AD (FAD) mutations cause a loss of gamma-secretase cleavage function at the epsilon site of gamma-secretase substrates, a process that produces biologically significant intracellular signal peptides. More recently his and other groups showed that many presenilin FAD mutants are unable to directly increase production of A β peptides suggesting that increased neuronal production of these peptides may be secondary to the primary neurodegenerative effects of the PS FAD mutants. His data suggest that the primary neurodegenerative effects of FAD mutations may be directly on neuronal survival pathways including the PI3K/Akt cell signaling pathway.