

BRODERICK PROBE® microelectrodes/biosensors: Clinical and preclinical use for Neurodegenerative diseases.

Patricia A. Broderick

CUNY Grad. Ctr., New York University Langone, USA

Progressive cell death in brain neuronal circuits, neurodegeneration, occurs far too often through our lives from brain injury caused by brain diseases, e.g. Epilepsy, Parkinson's, Alzheimer's and Huntington's. Studying the similarities among neurodegenerative diseases is the subject of this paper and the purpose is to find pharmacologic therapies for such. We focused on studies of clinical epilepsy and preclinical Parkinson's disease using an inventive biotechnology, Neuromolecular Imaging (NMI) with the BRODERICK PROBE®. NMI with the BRODERICK PROBE® allows the selective detection of specific neurotransmitters in discrete parts of the brain in vivo in the animal and human (resolution- temporal msec/spatial nanometers; time period, acute/chronic). Intraoperative Clinical Epilepsy studies were performed in mesial and neocortical temporal lobe epilepsy patients (IRB, NYU). Studies began with presurgical evaluation with electroencephalographic (EEG) monitoring; the site of cortical resection was delineated and subdural EEG electrodes were placed on the surface of the brain to identify epileptogenic cortex. The BRODERICK PROBE® [γ -irradiated (11.6-12.7 kGy)] was placed by direct visualization in the exposed cortical region with and without epileptic spike activity in regions destined for resection. In separate studies, each layer of neocortex and hippocampus was imaged in resected tissue, in situ (tissue was frozen at -80 degrees C). Results showed that in Intraoperative studies, wherein the neocortex was decidedly more degenerated than in resected tissue, in situ, neuropeptides were imaged dramatically more than were the monoamines. In the Preclinical Parkinson's study, there were two groups of male, Sprague-Dawley *rattus norvegicus* studied; one group was stereotactically lesioned with 6-hydroxydopamine (6-OHDA) in Substantia Nigra pars compacta (Charles River Lab, North Carolina) and the other group (same age and weight) not lesioned, served as controls. BRODERICK PROBE® was inserted in dorsal striata. The results showed that the lesioned group, exhibited neuropeptides and not monoamines whereas in the non-lesioned group, monoamines were abundantly imaged and neuropeptides were not observed. The data show that neuropeptides are triggered during the process of cell death, thereby playing an important role in neurodegeneration. This body of evidence shows a clear similarity in biochemical function during the process of cell death in Epilepsy and Parkinson's diseases. The data suggests that neuropeptides would provide novel treatments for Epilepsy, Parkinson's and perhaps other diseases of cell death.

Biography

Patricia A. Broderick completed her Ph.D. degree in Pharmacology at St. John's University, College of Arts and Sciences in 1979, completed her postdoctoral fellowship at the Albert Einstein College of Medicine, Montefiore Hospital, 1985 and a Research Associate Position at Cornell University, Neurology NY, 1986. She began her Professorship in CUNY in 1986 and her Professorship in NYU Langone, NY, 2000. Patricia is the inventor of 4 patents, held by CUNY and NYU Langone and she is a member of Editorial Boards in Austria, Switzerland and China. Patricia has published extensively, over 200 publications and presentations and has founded the Broderick Brain Foundation. She has mentored over 100 undergraduate, graduate students, medical doctors and industrial scientists in Micro- and Nanotechnology, Pharmacology and Neuroscience.

broderick@med.cuny.edu