Dr. Naren Banik received his Ph.D. from the University of London, UK, in 1970, in research related to Multiple Sclerosis (MS). He joined Stanford University in 1974 and continued his MS research in the Department of Neurology at the VA Hospital, Palo Alto, CA. His training on techniques and experimental models of neurodegenerative diseases, including EAE (an animal of MS), laid the ground work for his research on the mechanisms of myelin breakdown in demyelinating diseases. The mechanisms of myelin sheath degradation in demyelinating diseases remained a mystery in the early 1960s, and Dr. Banik demonstrated a significant role of the co-operative effects of proteases and lipases in myelin breakdown and implicated calpain as a major player.

In addition to research on MS, after joining the Neurology Department at the Medical University of South Carolina in 1976, he embarked on investigating the mechanisms involving calpain in tissue destruction in spinal cord injury (SCI) and examining the agents that can protect and preserve cells for recovery of function following injury. His laboratory has been at the forefront of research on whether calpain is involved in cell death, demyelination, and neurodegeneration in MS and if calpain activity is increased in postmortem MS tissue and EAE as well as in reactive glia and T cells. His work demonstrated increases in calpain activity and expression in both EAE tissue (monophasic, chronic, and relapsing/remitting models) and MS tissue, implicating calpain as a therapeutic target in the treatment of MS. Subsequent studies found changes in calpain expression correlated with pro- and anti-inflammatory cytokines in PBMCs of MS patients during relapse and remission and further demonstrated regulation of Th1/Th17 cytokines by inhibition of calpain in PBMCs of MS patients. Recent studies with a water soluble calpain inhibitor, SNJ1945, administered orally to EAE animals has been found to ameliorate the disease with improvement of clinical score, block T cell activation (shift Th1 to Th2-regulatory cells), protect cells, and preserve axon and myelin. This finding is significant since oral administration is non-invasive, both neuroinflammatory and neurodegenerative arms of the disease are targeted, and progression is halted compared to current therapies which are invasive, are only immunomodulatory, have numerous side effects, and do not slow or halt the progression of disability.

While the mechanisms of tissue degeneration following SCI were not fully understood, his SCI research project was the first to demonstrate the correlation of ultrastructural degeneration of myelinated axons with myelin and axonal protein degradation mediated by calpain and playing a significant role in this degenerative process. And, since tissue destruction in SCI is multi-factorial, treatment with one agent is unlikely to be fully beneficial. One agent affecting numerous pathways is the steroid hormone estrogen (17β-estradiol), which has been found to be beneficial in rat SCI. The initial findings in SCI have significantly influenced the subsequent studies aimed at developing therapeutic strategies, in particular proposing and utilizing estrogen administered IV or via nanoparticle, as a therapy in pre-clinical studies.

Work from his laboratory has also suggested the involvement of calpain in the degenerative process in Parkinson's disease (PD), and that in addition to brain (striata) DA neurons, spinal cord neurons are also affected and damaged in PD. Findings from animal model studies of PD were further confirmed in the postmortem spinal cord tissue of PD patients. Calpain was also implicated in the activation of innate (microglia) and adaptive immune (T cell) cell participation in the progressive degeneration and SNJ1945 administration blocked disease progression in mouse PD.

Dr. Banik is presently a Professor of Neurosurgery and holds a joint appointment in the Department of Microbiology and Immunology and Department of Ophthalmology and Visual Science. His current research interests include SCI, MS, Parkinson's disease, brain tumor, and other related conditions has been supported by NIH, VA, MS Society, and other organizations over the years. He has served as Associate Scientific Director of the South Carolina Spinal Cord Injury Research Fund since 2007.