

Doctor Buxbaum was Professor of Medicine at New York University School of Medicine from 1981 to 1999 where he carried out seminal studies on Immunoglobulin L-chain (AL) amyloidosis and light chain deposition disease (LCDD) and was among the first to propose and use the myeloma therapeutic regimen of melphalan and prednisone to successfully treat patients with AL amyloidosis. His laboratory also defined 4 of the first 10 transthyretin mutations responsible for Familial Amyloidotic Polyneuropathy (FAP) and Familial Amyloidotic Cardiomyopathy (FAC) and subsequently established a transgenic mouse model of Senile Systemic Amyloidosis.

In 1999 he moved to The Scripps Research Institute (TSRI) in LaJolla, California in order to collaborate more closely with Dr. Jeffery Kelly's protein chemistry laboratory. In collaborative studies they established the kinetic basis of the amyloidogenicity of the late onset cardiomyopathic transthyretin mutation (TTR V122I), defined the nature of murine transthyretin as a highly stable, in vivo trans-suppressor in transgenic mice and (with other collaborators) proposed a new adaptive model for protein transport and secretion from cells. Most recently, his laboratory, with collaborators from TSRI, UCSD and the Mayo Clinic (Fla), have definitively established the role of transthyretin as a suppressor of the Alzheimer's neuropathologic and behavioral phenotypes in transgenic mouse models of Alzheimer's disease. Doctor Buxbaum has served as the chair of the American Cancer Society Research Council, as a member of the Scientific Review Panel of the Israel Cancer Research Fund, the Advisory Council National Human Genome Research Institute (NIH) and the Scientific Advisory Board of Arthritis National Research Foundation. He is presently Director, Division of Rheumatology Research, Head of the Keck Autoimmune Disease Center and Professor in the Departments of Molecular and Experimental Medicine and Molecular Integrative Neuroscience at TSRI.