Leonidas Stefanis, MD, PhD

Professor

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Research	Molecular and Cellular Biology of Parkinson's Disease and related synucleinopathies Genetics and biomarkers of Parkinson's Disease and related synucleinopathies Protein degradation systems in neurodegenerative disorders
Keywords	Autophagy, Chaperone-Mediated Autophagy, Synuclein, Lysosome, Protein aggregation
Links	PubMed: https://pubmed.ncbi.nlm.nih.gov/?term=stefanis+l&sort=date Scholar: https://scholar.google.com/citations?user=kAxaHG0AAAAJ Webpage: https://eginitio-en.uoa.gr/1st_department_of_neurology/cv/1st_dept_neurology_active_university_faculty/stefanis_leonidas/

Biography

Dr. Stefanis obtained his MD from the NKUA Medical School in 1987 and his PhD from the same University in 1992. In 1991, he moved to the US, where he trained as Resident in Neurology at Columbia University in New York. In 1995, he embarked on a post-doctoral fellowship in the laboratory of Dr. Lloyd Greene, in the Dept. of Pathology, while in parallel he completed a two-year fellowship on Neurobehaviour . His work centered on mechanisms of neuronal cell death. In 1998 he was appointed Assistant Professor of Neurology in the Center for Neurodegenerative Diseases in the Dept. of Neurology at Columbia University, position which he held up till 2003. He focused on the pathogenesis of neurodegenerative disorders, with an emphasis on Parkinson's Disease (PD). In 2003 he moved back to Greece as Researcher Level B at the BRFAA, and set up a laboratory focusing on mechanisms of neurodegeneration, in particular in relation to protein degradation systems, alpha-synuclein and PD. In 2006 he was appointed Associate Professor in the NKUA Medical School, while he continued his work at BRFAA as affiliated investigator. Since 2017, he is Professor and Director of the First Department of Neurology at Eginitio Hospital. Currently, he is investigating various areas of PD pathogenesis, ranging from the bench to the bedside. He is examining the genetic underpinnings of the disease in the Greek population. A special focus is rare cases with genetic synucleinopathies , carriers of the p.A30G or p.A53T SNCA mutations, as well as patients with GBA1 or PRKN mutations. He is examining the utility of using alpha-synuclein, elements of the Autophagy Lysosome Pathway (ALP), or measurements of PRKN expression as disease biomarkers. He is examining pathways of neurotoxicity and aggregation induced by aberrant alpha-synuclein, with an emphasis on the involvement of protein degradation pathways, such as Chaperone-Mediated Autophagy. Conversely, he applies experimental therapeutics aiming to boost such intrinsic pathways, to confer neuroprotection in synucleinopathies . He is using a wide array of approaches, ranging from cellular and animal models to studies of patients' biological material. -