

Applicant Name (Last, first, middle):

## BIOGRAPHICAL SKETCH

Leonidas Stefanis

Name: Stefanis, Leonidas, MD, PhD  Personal Webpage: <a href="http://www.bioacademy.gr/lab/stefanis?lang=en">http://www.bioacademy.gr/lab/stefanis?lang=en</a> <a href="http://www.bioacademy.gr/faculty-details/GMI/leonidas">http://www.bioacademy.gr/faculty-details/GMI/leonidas</a>	<b>POSITION TITLE:</b> Professor of Neurology and Neurobiology, University of Athens Medical School
--	--

### EDUCATION /TRAINING

INSTITUTION AND LOCATION	DEGREE (if applicable)	YEAR(s)	FIELD OF STUDY
University of Athens Medical School	MD	1987	Medicine
University of Athens Medical School	PhD	1992	Medicine
Columbia University Medical School	Residency	1995	Neurology
Columbia University Medical School	Post-doctoral	1997	Neurobiology

### A. ACADEMIC AND PROFESSIONAL POSITIONS

#### **POSITIONS HELD**

Assistant Professor of Neurology and Pathology Columbia University School of Medicine, 1998-2003  
Researcher Level B, Biomedical Research Foundation, Academy of Athens, 2003-2006  
Associate Professor of Neurology and Neurobiology, University of Athens Medical School, 2006-2012  
Professor of Neurology and Neurobiology, University of Athens Medical School, 2012-present  
Affiliated Investigator, Biomedical Research Foundation, Academy of Athens, 2006-present

#### **ADVISORY-ADMINISTRATIVE DUTIES**

Director of Second Department of Neurology, University of Athens Medical School, 2011-present  
Associate Editor, Journal of Neuroscience, Section of Neurobiology of Disease, 2007-2012  
Member of Editorial Board, Journal of Biological Chemistry, 2009-2014, e-Neuro 2014- , Experimental Neurology, 2013-  
Member of Editorial Board, ERC Panel Member, LS5, on "Neurosciences and Disorders of the Nervous System", 2011, 2013, 2015, 2017  
Member of Programme Committee for the FENS 9th Forum of Neuroscience, 2014  
Member of the Scientific Issues Committee, International Parkinson and Movement Disorders Society, 2015-

Applicant Name (Last, first, middle):

## B. RESEARCH INTERESTS

The interest of the lab lies in the pathogenesis of neurodegenerative conditions, such as Parkinson's, Alzheimer's and Huntington's Diseases and Amyotrophic Lateral Sclerosis. We are studying potential mechanisms through which such conditions may initiate and propagate within the nervous system, with the hope that, if these mechanisms are identified, they can be potential targets for neuroprotective therapies. The pathophysiological mechanisms that are more closely studied include protein misfolding, protein aggregation, inclusion formation and dissolution, impairment of protein degradation systems, synaptic dysfunction and neuronal cell death. These processes constitute common threads in such neurodegenerative conditions. The group is more focused in the pathogenesis of Parkinson's Disease (PD) and, in particular, in deciphering the link between identified genetic defects and the disease. Activities range from the study of biological material from patients afflicted with PD, up to cell culture and animal models. Models are largely based on genetic defects linked to PD, but also include more classical neurotoxin approaches, such as MPTP. Relevant biochemical pathways identified in such models are then examined in patient biological material, while insights from the study of the patients are used to develop new models.

## C. SELECTED PEER-REVIEWED PUBLICATIONS (*max 10*) (*in chronological order*).

1. **Stefanis L**, ...Greene LA (2001) Expression of A53T Mutant But Not Wild-Type  $\alpha$ -Synuclein in PC12 Cells Induces Alterations of the Ubiquitin-Dependent Degradation System, Loss of Dopamine Release, and Autophagic Cell Death. J Neurosci 21: 9549-9560
2. Cuervo AM, **Stefanis L**, .. Sulzer D (2004) Impaired degradation of mutant alpha-synuclein by chaperone-mediated autophagy. Science 305(5688):1292-1295
3. Clough RL, **Stefanis L** (2007) A novel pathway for transcriptional regulation of alpha-synuclein. FASEB J 21(2):596-607
4. Vogiatzi T, ..**Stefanis L** (2008) Wild type  $\alpha$ -synuclein is degraded by chaperone mediated autophagy and macroautophagy in neuronal cells. J Biol Chem 283(35):23542-56
5. Xilouri M, Vogiatzi T, Vekrellis K, Park D, **Stefanis L**. (2009) Abberant alpha-synuclein confers toxicity to neurons in part through inhibition of chaperone-mediated autophagy. PLoS One. 2009;4(5):e5515
6. Vekrellis K, Xilouri M, .. **Stefanis L**. (2011) Pathological roles of  $\alpha$ -synuclein in neurological disorders. Lancet Neurol. 10(11):1015-25
7. Xilouri M, Brekk OR, .. Kirik D, **Stefanis L** (2013) Boosting chaperone-mediated autophagy *in vivo* mitigates  $\alpha$ -synuclein-induced neurodegeneration. Brain 136:2130-46
8. Bozi M, Papadimitriou D, ....Gasser T, **Stefanis L**. (2014) Genetic assessment of familial and early-onset Parkinson's disease in a Greek population. Eur J Neurol 21(7): 963-968
9. Papagiannakis N, Xilouri M, .. **Stefanis L**. (2015) Lysosomal alterations in peripheral blood mononuclear cells of Parkinson's disease patients. Mov Disord.30(13):1830-4
10. Xilouri M, Brekk OR, ... **Stefanis L** (2016) Impairment of chaperone-mediated autophagy induces dopaminergic neurodegeneration in rats. Autophagy 12(11):2230-2247