



HE-JIN LEE

PROFESSOR

DEPT. OF ANATOMY,
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Educations

1994 B.S., POSTECH, Pohang, Korea

2001 Ph.D., Boston University School of Medicine, Boston, USA

Professional Background

2016-Present Professor: Dept. of Anatomy, School of Medicine, Konkuk University

2011-2016 Associate professor: Dept. of Anatomy, School of Medicine, Konkuk University

2007-2011 Assistant Professor: Dept. of Anatomy, School of Medicine, Konkuk University

2006-2007 Research Assistant Professor: Institute of Biomedical Science and Technology, Konkuk University, Seoul, Korea

2003-2006 Staff Scientist: The Parkinson's Institute, Sunnyvale, CA, USA

2000-2003 Post-doctoral fellow: The Parkinson's Institute, Sunnyvale, CA, USA

Top 5 Publications

■ **Lee H-J**, Bae EJ and Lee S-J (2014) Extracellular alpha-synuclein-a novel and crucial factor in Lewy body diseases. *Nat Rev Neurol* 10: 92-98

■ **Lee H-J**, Suk JE, Patrick C, Bae EJ, Cho JH, Rho S, Hwang D, Masliah E and Lee S-J (2010) Direct transfer of alpha-synuclein from neuron to astroglia causes inflammatory responses in synucleinopathies. *J Biol Chem* 285: 9262-9272

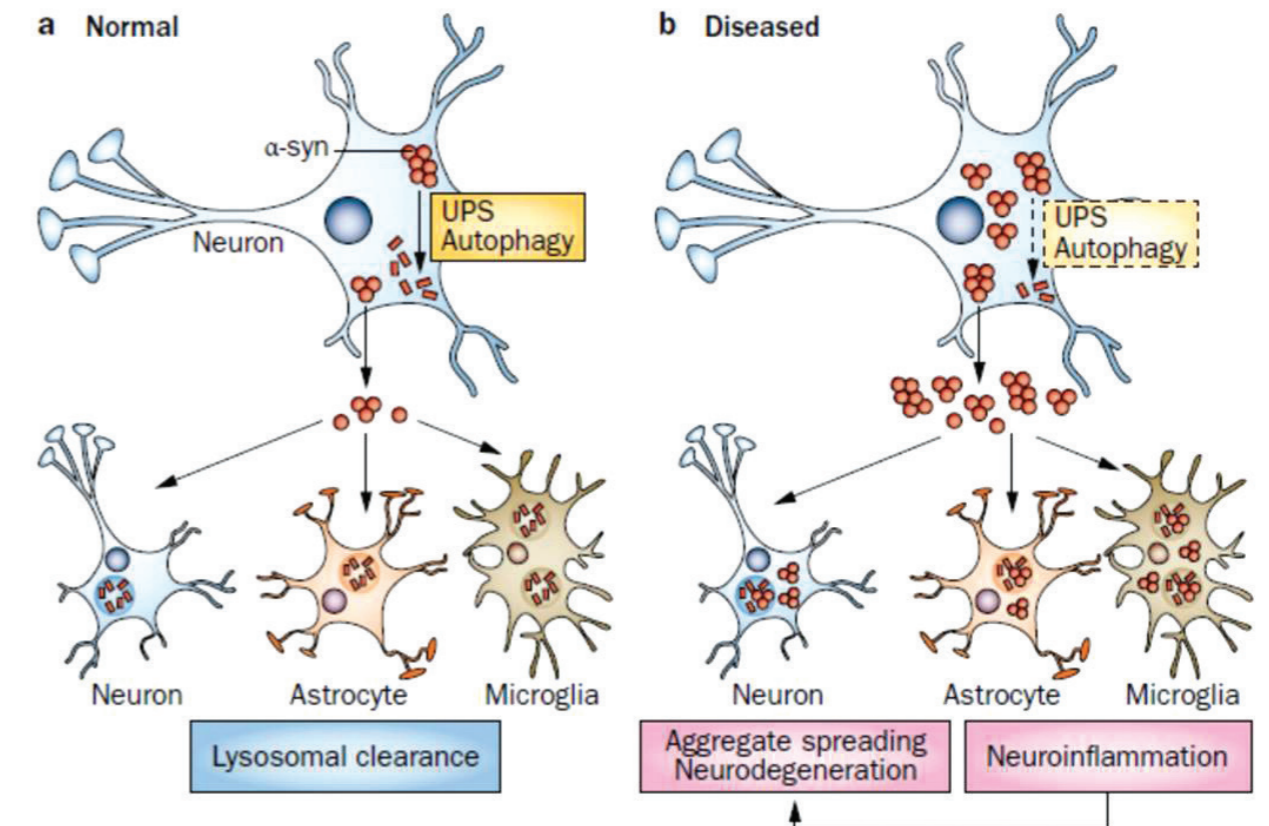
■ Desplats P*, **Lee H-J***, Bae EJ, Patrick C, Rockenstein E, Crews L, Spencer B, Masliah E and Lee S-J (2009) Inclusion formation and neuronal cell death through neuron-to-neuron transmission of alpha-synuclein. *Proc Natl Acad Sci U S A* 106: 13010-13015. (* co-first authors)

■ **Lee H-J**, Patel S and Lee S-J (2005) Intravesicular localization and exocytosis of alpha-synuclein and its aggregates. *J Neurosci* 25: 6016-6024

■ **Lee H-J**, Khoshaghideh F, Patel S and Lee S-J (2004) Clearance of alpha-synuclein oligomeric intermediates via the lysosomal degradation pathway. *J. Neurosci.* 24: 1888-1896

RESEARCH INTERESTS

Extracellular α -synuclein in Age-related Lewy Body Diseases



Lee et al. (2014) *Nature Rev Neurol*

Abnormal intracellular aggregation of α -synuclein is considered important in the pathogenesis of Lewy body diseases (LBDs), such as Parkinson's disease. However, the mechanisms underlying its activity are not fully understood.

Recent studies, including those from our group, have shown that small amounts of α -synuclein are released from neuronal cells by unconventional exocytosis, and that this extracellular α -synuclein

in contributes to the major pathological features of LBD, such as neurodegeneration, progressive spreading of α -synuclein pathology and neuroinflammation.

Our research group focuses on unraveling the mechanisms by which extracellular α -synuclein contributes to LBD pathology and finding possible therapeutic targets to halt and/or prevent disease progression.