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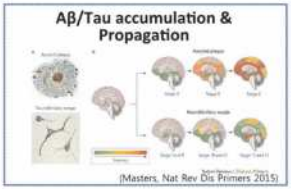
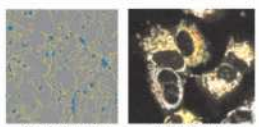
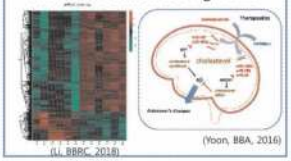
Neurobiology of Alzheimer's disease

Alzheimer's disease (AD) is the most common form of dementia in the elderly. Although details of AD pathogenesis still remain elusive, abnormal accumulation of amyloid-β (Aβ) and Tau in the brain is hypothesized to trigger pathogenic cascades that lead to AD. Abnormal accumulation of Aβ and Tau starts from specific brain regions and progressively propagates throughout the brain. Therefore, elucidating the molecular mechanisms of their accumulation and propagation is critical to understand AD pathogenesis. We are currently studying the molecular mechanisms for their accumulation and propagation utilizing cellular and mouse models.

Mounting evidence suggests that clearance of damaged mitochondria, termed mitophagy, is dysregulated, thereby leading to accumulation of damaged mitochondria and synaptic deficits in neurons. However, the underlying mechanisms for mitochondrial dysfunction and mitophagy deficits are largely unknown. We are currently studying the role of mitochondria and mitophagy in the pathogenesis of Alzheimer's disease as well as aging.

Accumulating evidence suggests that dysregulation of microRNAs is closely linked to the pathogeneses of various human diseases. However, the functional and therapeutic implication of microRNAs in AD remains largely unknown. Understanding the role of miRNAs in AD may provide new opportunities to develop novel therapeutic interventions for AD. We are currently seeking to probe the role of microRNAs in AD pathogenesis.

Aim	Elucidating the molecular pathogenesis of Alzheimer's disease and identifying targets for therapeutic intervention
Tool	Proteinopathy modeling + miRNA biology + mitochondria biochemistry + Mouse genetics
TARGET	Aβ/Tau accumulation & Propagation  (Masters, Nat Rev Dis Primers 2015) Synaptic deficits & Mitochondrial dysfunction  Neurite/synapse Mitophagy Assay microRNA & non-coding RNAs  (Yoon, BBA, 2016) (Li, BBRC, 2018)

Curriculum Vitae

2017~ : Principal Investigator, KBRI, Korea
2017 : Assistant Professor, Mayo Clinic, USA
2013~2017 : Senior Research Fellow/ Research Associate, Mayo Clinic, USA
2010~2013 : Postdoc/ Staff Scientist, Washington Univ., USA
2009~2010 : Postdoc, Univ. of Minnesota, USA
2008~2009 : Postdoc, Johns Hopkins Univ., USA
2007~2008 : Postdoc, Seoul National Univ., Korea

Academic Credential

2001~2007 : Ph.D., Biological Sciences, Seoul National, Univ., Korea
1996~2001 : B.S., Microbiology, Seoul National Univ., Korea

Awards/Honors/Memberships

2019~ : Member of the board of directors, Korean Society of Neurodegenerative disorder
2008~ : Member, Society for Neuroscience

Research keywords

Alzheimer's disease, Propagation, Synaptic deficits, Mitochondrial dysfunction, MicroRNA.

Key techniques

Modeling proteinopathies, Mouse genetics, Histology, Protein biochemistry, Primary neural cell-based assays, MicroRNA biology, Mitophagy assay, Somatic transgenesis.

Research Interests/Topics

- Dissect the molecular pathogenesis of Alzheimer's disease.
- Study the mechanisms of Aβ/Tau accumulation and propagation.
- Probe the role of miRNAs in the brain and Alzheimer's disease.
- Elucidate the molecular link between aging and Alzheimer's disease.
- Determine the role of mitophagy in the pathogenesis of Alzheimer's disease and aging.

Selected Publications

- **Kim J***, Fiesel FC, Belmonte KC, Hudec R, Wang WX, Kim C, Nelson PT, Springer W, Kim J*. miR-27a and miR-27b regulate autophagic clearance of damaged mitochondria by targeting PTEN-induced putative kinase 1 (PINK1). *Mol Neurodegener, 11:55, 2016.* (*co-corresponding authors)
- **Kim J**, Yoon H, Chung DE, Brown JL, Belmonte KC, Kim J. miR-186 is decreased in aged brain and suppresses BACE1 expression. *J Neurochem., 137:436, 2016.* (Editorial highlight)
- Choi J*, Gao J*, **Kim J***, Hong C, Tontonoz P. The E3 ubiquitin ligase Idol controls brain LDL receptor expression, ApoE clearance, and Abeta amyloidosis. *Sci transl Med., 7:314ra184, 2015.* (*equally contributed)
- **Kim J**, Yoon H, Horie T, Burchett JM, Restivo JL, Rotllan N, Ramirez CM, Verghese PB, Ihara M, Hoe HS, Esau C, Fernández-Hernando C, Holtzman DM, Cirrito JR, Ono K, Kim J. microRNA-33 Regulates ApoE Lipidation and Amyloid-beta Metabolism in the Brain. *J Neurosci., 35:14717, 2015.* (Featured Article)