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## Behavioral and molecular pathobiology of Neurological diseases

The increasing prevalence of overweight and obesity is a major public health concern in these days. Recently, growing evidence indicate that the obese population are more susceptible to some neurological conditions such as cognitive disorders, including Alzheimer's disease (AD) or mood disorders, including major depression disorder (MDD). These evidences indicate that changes in the organism that accompany overweight and obesity can ultimately lead to CNS dysfunction. However, the pathophysiological mechanisms and molecular players underlying this connection are poorly known. In this study, I focus on the mechanistic correlation between obesity and neurological disorders (such as Alzheimer's disease or Depression). To address this correlation, I approach using multidisciplinary way from gene to behavior level. Using this systematic exploration of this relationship would help to elucidate causal mechanism and opportunities for prevention and treatment.



**Curriculum Vitae**

2013~Present : Principal Investigator, KBRI  
 2008~2013 : Postdoctoral Fellow, Department of Pathology,  
 Division of Neuropathology, The Johns Hopkins  
 University School of Medicine, USA  
 2007~2008 : Postdoctoral Fellow, Department of  
 Pharmacology, College of Medicine,  
 Seoul National University, Korea

**Academic Credential**

2007 : Ph.D., Interdisciplinary Program in Neuroscience,  
 Seoul National University

2004 : M.S., Interdisciplinary Program in Cognitive Science, Seoul National University  
 2002 : B.S., Department of Genetic Engineering, SungKyunKwan University

**Grant**

General Researcher Program  
 (Young Researchers), National Research Funding of Korea, Principal Investigator  
 – Title : "Elucidation of the role of TDP-43, an ALS/FTD linked protein,  
 in eating disorder (anorexia nervosa, bulimia nervosa) and obesity"  
 Total costs : 149,430,000 Inclusive dates : 7/1/2014–6/30/2017  
 – Title : "Study on molecular connectome difference of stress susceptible and  
 stress resilient in various models of depressive mood disorders"  
 Total costs : 500,000,000 Inclusive dates : 3/1/2019–2/29/2024

**Research keywords**

Neurodegenerative disorder, Stress, Obesity, Depression, Mental illness, Feeding behavior, Impulsive control disorder, Neurometabolic disorder, Eating disorder (anorexia nervosa, bulimia nervosa).

**Key techniques**

Behavioral modeling, Genetic modification (cre/lox), Transcriptomics, Epigenomics,  
 Viral gene transfer, Optogenetics, Chemogenetics, FACs, Cell-type specific seq., Single cell seq.

**Research Interests/Topics**

- Mechanistic cross-talk among Neurodegenerative disease, Mental illness and Obesity.
- Study of correlation between neurological disorders and environmental factors. (ex. stress, environmental endocrine disruptors, etc.)
- Molecular mechanisms of eating behaviors in impulse disorder.
- Study of neurological disorders using multiple approach of behavior analyses.
- Study of transcriptome and epigenome in neurodegenerative/neurometabolic disorders.

**Research Publications (selected)**

- Donde A\*, Sun M\*, **Jeong YH\***, Ling J, Lin S, Braunstein K, Wang S, Chen L, Wong PC. Upregulation of Atg7 attenuates motor neuron dysfunction associated with depletion of TDP-43. *Autophagy*, 7:1-11, 2019. (Co-First)
- Labonte' B\*, **Jeong YH\***, Parise E, Issler O, Fatma M, Engmann O, Cho K, Neve Rachael, Nestler EJ, Koo JW. Gadd45b mediates depressive-like role through DNA demethylation. *Sci Rep.*, 9(1):461, 2019. (Co-First)
- **Jeong YH\***, Ling J\*, Lin S\*, Donde A, Braunstein KE, Majounie E, Traynor BJ, LaClair K, Lloyd TE, Wong PC. Tdp-43 cryptic exons are highly variable between cell types. *Molecular Neurodegeneration*, 12:13, 2017. (Co-First)
- Tsao W, **Jeong YH**, Lin S, Ling J, Price DL, Chiang PM, Wong PC. Rodent Models of TDP-43: Recent advances. *Brain Research*, 26(1462):26-39, 2012.
- **Jeong YH**, Kim JM, Yoo J, Lee SH, Kim HS, Suh YH. Environmental enrichment compensates for the effects of stress on disease progression in Tg2576 mice, an Alzheimer's disease model. *J Neurochem*, 119(6):1282-1293, 2011.