

## **Elliott J. Mufson**

Dr. Mufson is a Professor in the Department of Neurological Sciences and the Alla V. and Solomon Jesmer Chair in Aging at Rush University, Chicago, IL, USA. He received a PhD from the Downstate Medical Center and later spent 3 years as a postdoctoral fellow at the Harvard Medical School and rose to the rank of Assistant Professor in the department of Neurology. Dr. Mufson is a molecular neuroanatomist in the area of dementia in the aged and diseased brain. He was recognized as one of the hundred most cited neuroscience researchers by the ISI. The focus of Dr. Mufson's laboratory revolves around understanding of the molecular mechanisms underlying the selective vulnerability of neuronal populations, which degenerate in people with mild cognitive impairment (MCI), Alzheimer's disease (AD) and Parkinson's disease (PD). In AD, he has been in the forefront of human tissue studies aimed at defining the mechanisms that play a role in the degeneration of cholinergic basal forebrain (CBF) neurons during the progression of AD. In particular, his laboratory has performed studies aimed at determining the involvement of the neurotrophin NGF, its high affinity trkA receptor, the low affinity p75 NTF receptor and pro NGF, in the selective vulnerability of CBF neurons. Recently, Dr. Mufson was involved in a clinical trial testing the ability of NGF gene therapy for the treatment of dementia in people with mild AD. Dr. Mufson's laboratory has performed single cell gene array analysis of the molecular signature of CBF neurons in tissue harvested from people who died with a clinical diagnosis of MCI compared to AD and those with no cognitive impairment. In Parkinson's disease (PD), Dr. Mufson has investigated the ability of the neurotrophin brain derived neurotrophic factor (BDNF) as a potential treatment for this disease. Other studies performed in his laboratory using tissue samples of the human substantia nigra revealed a striking decline in the expression of the dopamine transporter (DAT) with age, which is exacerbated in PD. The findings generated from these human clinical pathological studies, have formed the foundation for Dr. Mufson's investigation of transgenic mouse models of AD, to further define the mechanisms underlying neuronal selective vulnerability in AD. Currently, the laboratory is studying the triple transgenic mouse model of AD in relation to the onset of tau pathology and its regulation by estrogen. Dr. Mufson is currently the leader of an NIA funded Program Project entitled "Neurobiology of Mild Cognitive Impairment in the Elderly" as well as other NIH funded grants.