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Small molecule drug therapy to increase neurogenesis in Alzheimer's disease

Despite decades of investigations in both basic and clinic, the cause of Alzheimer's disease (AD) still remains unknown. Current problem of developing AD research is that many treatments are focusing AD hallmarks, amyloid plaque and neurofibrillary tangles, and they have been very effective in AD animal models but never be successful showing any significant effect in clinical trials. Thus, establishment of an effective treatment in a model, which represents pathophysiology of AD is needed. Previously, we were able to show improved cognitive function of aged, memory-impaired animals through the implantation of human neural stem cells (NSCs), which produced much excitement throughout the research world and the overall medical community; given the implication that this could lead to a cure for all neurodegenerative diseases, including AD. However, when we transplant NSCs to a transgenic animal model produces Amyloid-β (Aβ) plaque formation in the brain by expressing familial AD mutant amyloid precursor protein (APP), mimicking the pathological condition of AD, we did not find any new neuronal development formed from the donor cells. This indicates that transplantation of NSCs by itself may not be a cure for AD. Here, we show that the combination drug therapy of Phenserine (reduce APP level) and NBI-18 (increase endogenous NSCs) increased neurogenesis and significantly improved memory in the transgenic AD mouse model. This combination therapy could bring us an effective treatment for AD. I will further discuss the use of iPS cell to confirm the efficacy of this therapy *in vitro* 3D human AD brain model.

Speaker Biography

Kiminobu Sugaya is a Professor of Medicine, the Head of Neuroscience and the Chair of Multidisciplinary Neuroscience Alliance, Burnett School of Biomedical Sciences-College of Medicine University of Central Florida.

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