Se Hee Oh, J Cell Sci Ther 2015, 6:2
http://dx.doi.org/10.4172/2157-7013.S1.033

OMICS International
conferenceseries.com

5th World Congress on
Cell & Stem Cell Research

March 23-25, 2015 DoubleTree by Hilton Chicago - North Shore, USA

MSCs enhance the microtubule network and trafficking in a parkinsonian model

Se Hee Oh
Yonsei University School of Medicine, Korea

Ample evidence has suggested that α-synuclein is a toxic species in synucleinopathies widely distributed in brain. Overexpression of α-synuclein predominantly localize to axons in the neuron of Parkinson's disease (PD) which affects microtubule (MT) network, trafficking and actin polymerization. Furthermore, overexpression of α-synuclein and its mutant resulted in disruption of neurite morphology, microtubule cytoskeleton and the transport of endosomes as well as autophagosomes, but the specific relation of α-synuclein and different compartments of the neuronal cytoskeletal machinery has not yet been elucidated. Therefore, MT stability would play key roles in the pathogenesis and progression of PD and would be clinically relevant in PD treatment. Mesenchymal stem cells (MSCs) secrete various cytotropic factors that have neuroprotective effects through complex mechanisms, such as modulation of neuroinflammation, enhancement of cell survival signals, increased neurogenesis, and modulation of autophagy. In the present study, we identified that eukaryotic translation elongation factor 1A (eEF1A), soluble factors derived from MSCs and MSCs could exert neuroprotective effects through modulation of microtubule binding, bundling and severing on α-synuclein time-dependently. In addition, we showed that MSCs had effect on axonal transport and cell growth, which led to a prosurvival effect on neurons. Furthermore, we presented that MSCs modulate axonal transport of signaling and fuse the outer membrane of the autophagosome with lysosome formation. These studies suggest that MSCs on the microtubule system a potential target of α-synuclein, and might have an impact on neuronal structure, function and survival.

huday11@naver.com

Notes: