Yusuf Issa Olakunle et al., J Neurol Neurosci 2017, 8:6
DOI: 10.21767/2171-6625-C1-005

Fibroblast growth factor 9 suppresses cell death through ERK signaling in Huntington's disease

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Huntington's Disease (HD) is a heritable neurodegenerative disorder characterized by selective and progressive damage of medium spiny neurons in the striatum and there is no cure for HD to date. A type of Fibroblast Growth Factor (FGF), FGF9, has been reported to play pro-survival roles, in other neurodegenerative diseases, such as Parkinson's disease and Alzheimer's disease. With many similarities in the cellular and pathological mechanisms that eventually causes cell death in neurodegenerative diseases, we hypothesize that FGF9 might provide neuro-protective functions in HD. Here, we used STHdh$^{Q7/Q7}$ (WT) and STHdh$^{Q111/Q111}$ (HD) striatal knock-in cell lines as our models and examined the neuro-protective effects of FGF9 on HD. Employing MTT and PI staining assays to determine cell proliferation and survival respectively, we found that FGF9 enhanced cell proliferation and also increased cell survival under a starvation stress condition. In addition, we observed that FGF9 significantly up-regulated FGF signaling through ERK1/2, Akt, JNK and mTOR pathways and increased neuro-trophic factor (GDNF) and anti-apoptotic BcL-xL proteins in HD cells. Especially, ERK pathway plays a critical role in the effects of FGF9 on cell survival and GDNF regulation. These results not only show the neuro-protective effects of FGF9, but also clarify the critical mechanisms in HD cells, further providing therapeutic potential of FGF9 in HD.

Biography

Yusuf Issa Olakunle is currently pursuing his PhD in Taiwan International Graduate Program (TIGP) Academia Sinica in Neuroscience at National Cheng Kung University (NCKU). He has completed his BSc degree in Physiology from Ahmadu Bello University, Nigeria in 2010 and MSc degree in Physiology from the University of Ibadan, Nigeria in 2014.

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