

The Increase of AMP-activated Protein Kinase during Exercise and its Effect on Reducing Parkinson's Disease Symptoms

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Letter to the editor

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Parkinson's disease (PD) is a neurodegenerative disorder of the central nervous system which is related to disruption of dopaminergic neurons and is considered an abnormality of the brain [1,2]. It is also important to know that, there is an estimation which shows that seven to ten million people worldwide are living with PD. As a result, the most focus should be placed on its treatment or prevention in order to decrease its rate of occurrence.

The most significant issue that should be considered is that, Adenosine 5'-Monophosphate (AMP)–Activated Protein Kinase (AMPK) main role is to maintain the whole body energy balance [3]. Mainly, during exercise which is a muscular activity, ATP decreases and AMPK activity increases in the brain as well as skeletal muscles [4]. Moreover, some recent studies have shown that activation of AMPK suppresses neuronal polarization [5].

An equally important issue that should not be ignored is that, some researchers have revealed that AMPK integrates growth factor signaling with cell cycle control, which leads to enhance brain development [6].

Based on the above-mentioned points, we hypothesize that exercise may help regulate neuronal polarization which leads to brain development in neurodegenerative disorders such as PD. So we think that exercise would have an essential role in regulation of dopaminergic neurons which are important in reducing PD symptoms. Surely, experimental studies and clinical observations are needed after a specific period of exercising to validate our hypothesis.

References

- Vernier P, Moret F, Callier S, Snapyan M, Wersinger C, et al. (2004) The degeneration of dopamine neurons in Parkinson's disease: insights from embryology and evolution of the mesostriatocortical system. Ann N Y Acad Sci 1035: 231-249.
- Rieker C, Engblom D, Kreiner G, Domanskyi A, Schober A, et al. (2011) Nucleolar disruption in dopaminergic neurons leads to oxidative damage and parkinsonism through repression of mammalian target of rapamycin signaling. J Neurosci 31: 453-460.
- Hardie DG, Hawley SA, Scott JW (2006) AMP-activated protein kinasedevelopment of the energy sensor concept. J Physiol 574: 7-15.
- Richter EA, Ruderman NB (2009) AMPK and the biochemistry of exercise: Implications for human health and disease. Biochem J 418: 261-275.
- Stephen Amato, Xiuxin Liu, Bin Zheng, Cantley L, Rakic P, et al. (2011) AMP-Activated Protein Kinase Regulates Neuronal Polarization by Interfering with PI 3-Kinase Localization. Science 332: 247-251.
- Dasgupta B, Milbrandt J (2009) AMP-activated protein kinase phosphorylates retinoblastoma protein to control mammalian brain development. Dev Cell 16: 256-270.

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