REDVET - Revista electrónica de Veterinaria - ISSN 1695-7504
Vol 25, No. 2 (2024)
http://www.veterinaria.org
Article Received: June 2024; Revised: July 2024; Accepted: August 2024

Concept Of Ama In Ayurveda W.S.R. To Mitochondrial Role In Nervous Disorders: A Conceptual Research

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Abstract
These days nervous disorders are a bigger challenge amongst medical field. Various factors are provoking mental health. In this study mitochondrial health is the topic of concern for further studies to restrain nervous disorders. According to Ayurveda, Ama Rasa production is the basic reason for any disease. If , it can be controlled then mitochondrial death can be controlled hence nervous disorders. Article elaborates about inter-relationship among mitochondria & brain activity. Under nutrition of cells is the basic factor which can be taken into account for healthy life. Ayurveda can better explain the process of apoptosis process.

Keywords : Ama Rasa, Apoptosis, Ayurveda, Mitochondria, Neurodegeneration.

Introduction

According to Ayurveda, Ama word means unripe. In Ayurveda it is mentioned that if Rasa Dhatu (plasma) does not metabolise properly with its own Agni (digestive fire), then the end product formed become putrid due to long stay inside body because it does not get converted into nano size so will be unable to cross the cell pores for further pharmacokinetic actions. Hence get stuck, circulating in blood stream causing diseases accordingly. This Ama Rasa is also denoted as Ama Visha (putrid toxic fluids). It happens with all the Dhatus (elements) one by one. So, as a result, inspite of formation of Shudh (healthy) Dhatus , Mala (waste products) become more prominent causing lesser immunity. Vata (air) inside body increases because it is produced after every biochemical reaction. Increased Vata is due to long term decrease in metabolism reactions of cells. Due to lesser rate of metabolism, Kapha Dhatu (adipose tissue) tends to accumulate and blocks the Strotras (channels). Hence frequency of calcium ions to cross cell pores decreases which ultimately leads to production of more Ama Rasa. The reason being, slower metabolism & same pace of food habits. On the long run , it causes hypertrophy of cells & ultimately apoptosis. Throughout all this process , production of air inside body increases which is the cause of hypertrophy. Cell size increases due to trapped air because air has tendency of expansion. As per modern concepts, when there is slower metabolism then permeability of cell membrane tends to increase to cope up with deficit concentration of calcium ions inside mitochondria which ultimately leads to cell death one day but this happens when this process goes for longer time. Neurons also bear the same procedure which leads to decrease in nervous system functions. Symptoms may vary.

Key Role Of Mitochondria In Neurodegeneration

Energy metabolism & Ca^{2+} ion regulation are the functions of mitochondria inside neurons. Axonal and dendritic development, axonal regeneration and synaptic functions are regulated by mitochondria. Dysfunctional mitochondria is the cause of certain neurodegenerative diseases. [1]

As neurons are the primary functional unit of the nervous system, it follows that most of the work on brain metabolism and mitochondria have focused on these cells.[2]

It is reported that damaged mitochondria released from microglia are sensed by astrocytes to propagate inflammatory signals and provoke neurodegeneration.[3]

Astrocytes are specialized star - shaped glial cells in CNS. These communicate with neurons through calcium waves. These comprise around 20% of brain cells. These are of 2 types: fibrous & protoplasmic. They transfer healthy mitochondria to the neighboring neurons to prevent neuronal damage when in need.

Brain has many mitochondria because it is a highly energy-dependent organ. Mitochondria regulate brain function. This is demonstrated in inherited mitochondrial disease, which is a distinct group of conditions caused by mutations in mtDNA (mitochondrial DNA) and nuclear genes that encode for structural mitochondrial proteins or proteins that are involved in mitochondrial function. Many of these disorders are distinguished by atrophy in cortical, brainstem, and cerebellar brain regions as well as a variety of neurological symptoms such as changes in vision, deafness, difficulty swallowing, reduced muscle tone, incoordination, neuropathy, and seizures. Mitochondrial diseases are linked to affective changes. Mitochondrial dysfunction is also found in primary developmental and neuropsychiatric disorders. Neuroendocrine pathways activate actions and mechanisms to control energy usage during acute stress exposure. These include changes in eating habits (shifting toward calorically dense macronutrients) and rapid removal of free fatty acids from central fat stores. These changes in metabolic, endocrine stressors, and stress-mediators affect mitochondria.

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Mitochondria perform a crucial function in maintaining the cell's internal environment, when glucocorticoid exposure is high, mitochondria has reduced their capacity to buffer calcium. Sensitization to cell death is induced by rapid calcium flux or calcium overload in the cell. Acute stress increases circulating levels of vital substrates like glucose and lipids to provide energy. In conditions of severe or prolonged exposure to stress, mitochondria become fragmented, increasing the risk of cell death . Prolonged fragmentation is associated with further oxidative stress and damage to mtDNA . Importantly, these changes impair the bioenergetic functions of mitochondria and are amplified over time . All changes are due to hyperglycemia.

Mitochondria communicate with other parts of the cell, indicating their functional status, according to their symbiotic origins. Crucially, the mitochondrial response to stress is communicated both intracellularly and across the body as a whole. When exposed to stress mediators, mitochondria release signaling molecules called mitokines. Mitochondrial fitness is shown by mitokines, which is particularly important when environmental stressors are present. Mitochondrial metabolites, calcium, and reactive oxygen species (ROS) are mitokines.

At low levels, ROS supports several important cell functions as a byproduct of energy producing processes in mitochondria. ROS increases the cell's antioxidant capacity and causes oxidative stress, which leads to cell death and tissue damage. ROS, like other mitokines, can induce local and systemic pathological processes like inflammation, metabolism, oxidative stress, gene expression, and cell senescence. Nucleus, where mitochondrial regulation, controls the genome, requires these signals. Mitochondria can influence many physiological processes in the body through these mechanisms. Oxidative stress has been suggested to play a role in the pathogenesis of neuropsychiatric disorders, particularly depression and dementia. Among other abnormalities, it is suggested that the accumulation of ROS and mtDNA mutations leads to impaired cell functioning and replication, resulting in neuronal atrophy and apoptosis.[4]

Conclusion

It is evident from the above discussion that mitochondria plays a crucial role in causation of neurodegeneration. Ama Rasa is the factor which can be controlled by healthy eating habits with wise eating gaps between two meals. Food intake after proper digestion of the previous meal is the most important factor to avoid neurodegeneration and hence nervous disorders so that mitochondria may get some time to refresh itself and to avoid overwork along with ultimate exhastion which leads to apoptosis one day.

Conflict Of Interest

None.

Reference

1. Pleiotropic Mitochondria: The Influence of Mitochondria on Neuronal Development and Disease. Vidhya Rangaraju, Tommy L Lewis Jr ,Yusuke Hirabayashi, Matteo Bergami, Elisa Motori, Romain Cartoni, Seok-Kyu Kwon, Julien Courchet, 2019 Oct 16;39(42):8200– 8208. doi: 10.1523/JNEUROSCI.
2. Glial mitochondrial function and dysfunction in health and neurodegeneration . Kevin McAvoy, Hibiki Kawamata. Elsevier Molecular and Cellular Neuroscience Volume 101, December 2019, 103417 Molecular and Cellular Neuroscience.
3. Translational Neurodegeneration Focusing on mitochondria in the brain: from biology to therapeutics . Nanshan Song , Shuyuan Mei, Xiangxu Wang , Gang Hu and Ming Lu. Translational Neurodegeneration (2024) 13:23 https://doi.org/10.1186.
4. Stress and Psychiatric Disorders: The Role of Mitochondria. Teresa Daniels, Elizabeth Olsen, Audrey R Tyrka. 2021 May 7. Published in final edited form as: Annu Rev Clin Psychol. 2020 Feb 24;16:165–186. doi: 10.1146/annurev-clinpsy-082719-104030.