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Review Article

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A Mitochondrial Function is Forming Sexual Constitution of Men

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Abstract

The article provides a brief overview of the functions of mitochondria and factors leading to the development of mitochondrial dysfunction. The human sexual constitution is formed in the prenatal period and early ontogenesis. The reasoned data make it possible to suggest the influence of mitochondrial function on the formation of the sexual constitution.

Keywords: Mitochondria; Mitochondrial dysfunction; Sexual constitution

Introduction

Energy exchange in cells is carried out due to universal cellular organelles-mitochondria. Mitochondria are organelles of energy supply, in which metabolic processes of the cell also occur. Their number in the cell is from 50 to 1000 or more. It is distinguished from other cellular organelles of mitochondria by the presence of its own well-studied mitochondrial DNA, which determines the ability of mitochondria to auto produce.

In specialized cells, mitochondria are concentrated in those areas where there is the greatest need for energy. For example, in muscle cells, large numbers of mitochondria are concentrated along the working elements - contractile fibrils. In cells whose functions are associated with particularly high energy costs, mitochondria form multiple contacts, uniting in a network, or clusters (for example, cardio myocytes and symplasts of skeletal muscle tissue). In the cell, mitochondria perform the function of respiration.

It was found that some components of the respiratory chain (coenzyme Q, cytochrome oxidase) along with electron transfer along the chain also carry out the transfer of protons from the mitochondrial matrix into the intermembrane space, resulting in the formation of a proton gradient. In the process of reverse proton flow into the mitochondrial matrix, the energy released in the respiratory chain is utilized by phosphorylation of adenosine diphosphate (ADP) to adenosine triphosphate (ATP) and other

macroergic phosphates, and a reserve of biological oxidation energy is created.

In addition to electron transport, oxidative phosphorylation, mitochondria provide another process involving redox reactions - b-oxidation of fatty acids. Free fatty acids are transformed into Acetyl-CoA and then form esters with carnitine. Carnitine-Acetyl-CoA is transferred through the mitochondrial membrane, Acetyl-CoA is released and participates in b-oxidation [1].

Also in the mitochondria there is a regulation of the intracellular distribution of calcium, the formation of steroids, the regulation of apoptosis. In the mitochondria, the pathways of metabolism of proteins, fats and carbohydrates are integrated, the main energy processes are carried out. In this regard, changes in mitochondria can cause a complex chain of pathological processes at the level of the cell and the whole organism as a whole [2]. Also, mitochondria play an important role in such processes as: aging and cell death, the development of certain diseases and pathological processes, and the physiological adaptation of the body to endurance exercises [3].

There is no doubt that violations of the bioenergetics systems of the cell play a paramount role in the realization of the damaging effects of various factors. Mitochondrial dysfunction leads to insufficient energy supply of cells, disruption of many other important metabolic processes, further development of cell damage, up to cell death [4,5]. Therefore, the energy state of the organism as a whole depends on the state of mitochondria.

Currently, the effect of impaired ATP synthesis in

mitochondria on the functional activity of cells and tissues has been studied to the greatest extent. It was found that when the content of ATP in the cell is reduced by 10-20%, the activity of all energy-dependent processes decreases by 70-80%. The effects of an insufficient amount of ATP include the suppression of anabolic processes, disruption of the operation of ion pumps and, accordingly, ion homeostasis in the cell, inhibition of the functions of the cytoskeleton [6].

Inhibition of energy generation processes in mitochondria is accompanied by a weakening of lipid β -oxidation, which results in impaired lipid homeostasis in the cell and accumulation of Acyl-CoA-thioesters, acyl-carnitines, ceramides and triglycerides, which have potential toxicity to the cell. An exception is mitochondria of nerve tissue, the biochemical feature of which is the lack of ability to carry out β -oxidation of lipids [7].

Mitochondria have an exceptional role both in maintaining the vital functions of the cell and in high destructive potential. Violation of any of the functions of mitochondria - energetic, thanatogenic, or activation of the production of free radicals by them can cause the development of functional and morphological disorders in various tissues and organs [8].

Many environmental factors and drugs are a significant cause of pathological changes in mitochondria. These factors include the action of alkylating agents (e.g., nitrosamines from the environment), hydroxyl radicals, high doses of ultraviolet and ionizing radiation, drugs (bryostatin, azido thymidine), other chemical agents (alloxane, cyanides, carbon monoxide, etc.).

The cause of mitochondrial damage may be the insufficiency of certain trace elements, such as selenium [9]. In many cases, the sensitivity of mitochondrial DNA to the action of environmental damaging factors is several times higher than the sensitivity of the nuclear genome.

It is known that, taking into account the numerous metabolic functions of microflora, a violation of its colonization resistance is considered as a trigger factor in the development of a number of different diseases. Thus, studies conducted in recent years demonstrate a significant association of dysbiotic disorders of intestinal microflora with the pathogenesis of non-alcoholic fatty liver disease (NAFLD), resulting from damage to mitochondria. The presented data demonstrate the significance of dysbiotic changes in the development of mitochondrial dysfunction, the formation of steatosis in NAFLD with its subsequent transformation into steatohepatitis and progression to fibrosis and cirrhosis [10]. A number of publications report an increasing amount of evidence that impaired mitochondrial function can have a significant effect on mood and psychotic disorders.

Discussion

Recently, data have been obtained from a wide range of research studies involving animals and humans that support the hypothesis that impaired mitochondrial functions can disrupt neural plasticity pathways and reduce cellular elasticity, which in turn potentially contributes to the development or progression of mood disorders, such as severe depression or bipolar disorder, as well as other mental illnesses-schizophrenia or autism [11].

A connection was found between mitochondrial dysfunction and Alzheimer's disease. The results are consistent with data from previous work, which established that the accumulation of beta-amyloids in neurons (a hallmark of Alzheimer's disease) is directly involved in mitochondrial dysfunction [12].

It has been experimentally established in animals that the prenatal stress of the female during early maturity leads to the birth of cubs with a higher level of androstenedione and a lower level of testosterone than in normal mice.

This may mean that males have less masculinization. In addition, mice, although they were born with normal weight, but in the future their growth began to slow down. Their body weight was 10-20% lower than in control mice [13]. Researchers have identified several sets of genes associated with the structure and function of mitochondria that are responsible for energy production. Indeed, in mice born to stressed mothers, mitochondrial function in the hypothalamus is sharply reduced compared to normal mice. It is known that the sexual constitution is formed under the influence of hereditary factors and development conditions in the prenatal period and early ontogenesis.

Therefore, factors (psycho-emotional overload, xenobiotic, dysbiosis of the intestinal microflora) that influence the body in the prenatal period and early ontogenesis can lead to mitochondrial dysfunction in the central nervous system (CNS), in particular the hypothalamus.

In this case, various molecular, biochemical and cellular disorders occur, leading to the development of hypothalamic dysfunction. As a result of which there is an imbalance of hormones impaired blood flow in the body, body or part there of metabolic impairment etc., a "vicious circle" occurs.

Consequently, mitochondrial dysfunction in the hypothalamus will contribute to the formation of weak and weakened variants of sexual constitution. This will be manifested by the early detection of vascular endothelial dysfunction. One of the first studies in which a relationship was established between low birth weight (intrauterine growth retardation) and early vascular endothelial dysfunction was the work [14.15].

Conclusion

Thus, we suggest that the mitochondrial function of the central nervous system affects the formation of the human sexual constitution. Mitochondrial dysfunction is a standard pathological phenomenon that develops when exposed to one or most often a combination of pathological factors. These are psychosomatic overloads; xenobiotic burden of the body, intestinal microflora dysbiosis and trauma, including various radiation.

Mitochondrial dysfunction in the central nervous system leads to impaired energy metabolism in the hypothalamic cells. The integration and regulatory functions of the hypothalamus are disrupted - these are vegetative, metabolic, endocrine and trophic functions, the immunological reactivity of the body. Elimination or minimization of the effects of harmful factors on the body of a pregnant woman and a child after birth (2-3 years), it is possible to achieve the formation of the desired sexual constitution.

Each type of human sexual constitution has its own energy balance. The energy balance of the body affects the body's resistance to pathological factors. If the energy balance of the body is below average, the body will not be able to resist painful aggressions and will become hopelessly ill," wrote Russian physician and philosopher A.S. Zalmanov more than 50 years ago [16].

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