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Biochemical Foundations Of Energy Metabolism Pathologies

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Abstract: The biochemical foundations of energy metabolism pathologies analyze the mechanisms of disturbances in catabolic and anabolic processes that lead to cellular energy deficiency. Defects in the Krebs cycle, oxidative phosphorylation, and β-oxidation of fatty acids underlie many metabolic disorders. Energy deficiency manifests as impaired function of highly energy-dependent organs and systems, particularly the nervous and cardiovascular systems. Disorders of energy metabolism are characterized by the accumulation of toxic metabolites, which exacerbate the pathological process. The study of these biochemical mechanisms is of particular importance for the development of methods for diagnosing, treating, and preventing metabolic diseases.

Keywords: Bioenergetics, energy metabolism, ATP, thyroxine, triiodothyronine, hyperthyroidism, cerebral hypoxia, Krebs cycle, antioxidants.

INTRODUCTION:

Energy metabolism (bioenergetics) is the fundamental process that provides the organism with energy required to maintain homeostasis, muscular activity, secretion, and the synthesis of biomolecules. A central role in energy metabolism is played by biological oxidation coupled with phosphorylation, resulting in the formation of ATP — the universal energy equivalent of the cell.

Disturbances in these processes may occur due to genetic defects in enzymes, toxic damage to mitochondria, endocrine disorders, or infectious diseases. Under normal conditions, oxidation and phosphorylation processes are tightly coupled; however, under pathological conditions, their uncoupling may occur, leading to a significant decrease in the efficiency of food energy utilization. Thus, pathologies of energy metabolism represent a key factor in the development of numerous diseases and functional disorders.

Experimental data demonstrate that under pathological uncoupling of oxidation and phosphorylation, the functioning of various organs deteriorates — for example, antibody production decreases in experimental hypothyroidism, and muscular activity is reduced under α-dinitrophenol intoxication.

Pronounced alterations in energy metabolism occur during bacterial intoxication. Diphtheria and staphylococcal toxins, particularly Staphylococcus aureus, exert a disorganizing effect, leading to a significant increase in actual heat production.

More severe deviations in energy metabolism are observed in cases of extensive burns. The initial cause of these changes is a reduction in the level of protein sulfhydryl (SH) groups, accompanied by a loss of mitochondrial enzymatic activity and disinhibition of oxidative phosphorylation. As a result, ATP synthesis decreases, leading to dysfunction of internal organs. In burn pathology, the reduction in SH-group levels is associated with their inhibition by under-oxidized metabolic intermediates and burn toxins.

Cellular energy processes depend not only on the concentration and activity of specific substrates (metabolic intermediates) but also on the condition of systems that regulate energy metabolism—primarily the nervous and endocrine systems. For instance, heat production may markedly increase during emotional excitation or in the phase of traumatic shock.

The key regulators of mitochondrial membrane permeability, and consequently of energy

metabolism, are the thyroid hormones thyroxine and triiodothyronine. When thyroxine secretion is elevated (as in hyperthyroidism), both free and phosphorylative oxidation intensify, leading to increased heat production. However, due to its disorganizing effect, thyroxine induces mitochondrial swelling, which activates phosphorylation and respiration enzymes and shifts metabolism toward predominantly free oxidation. Conversely, when thyroxine secretion is reduced (as in hypothyroidism), heat production decreases.

Further Mechanisms of Energy Metabolism Regulation and Hypoenergetic States

Energy metabolism increases in pituitary tumors associated with excessive secretion of growth hormone (GH), which promotes heat production through stimulation of free oxidation.

In conclusion, under pathological conditions, the reduction of coupled oxidative and phosphorylative processes may also result from a variety of other factors that influence the conversion of food energy into heat. Energy metabolism varies depending on the level of excretion of under-oxidized metabolites, which are more energy-rich than the usual end products of metabolism, as well as on accelerated tissue degradation and other metabolic processes occurring in the organism.

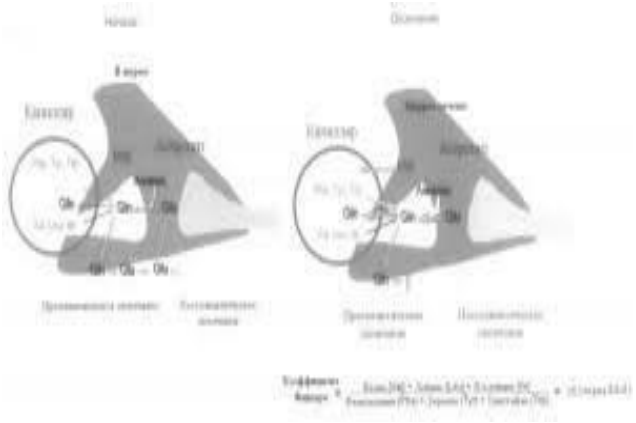
Hypoenergetic States

A living cell continuously requires ATP, since numerous ATP-dependent processes are constantly taking place. For example, approximately 15 % of the total basal metabolic energy (that is, the energy expended at rest) is used for protein renewal, while about 30 % is consumed to maintain the transmembrane gradients of sodium and potassium ions.

During the transition to muscular activity, the demand for ATP increases significantly. ATP reserves in the cell are virtually nonexistent. For instance, in cardiac muscle, ATP is depleted within a few seconds if its synthesis is inhibited. Therefore, the cell must continuously receive nutrients (hydrogen donors) and oxygen to maintain ATP synthesis.

During starvation, endogenous tissue components serve as energy substrates. Under such conditions, the rate of energy metabolism decreases: after two weeks of fasting, oxygen consumption drops by approximately 40 %, which represents an alimentary form of hypoenergetic state. Nutrient reserves are limited, and prolonged energy deficiency leads to progressive tissue catabolism and systemic dysfunction.

The body's nutrient reserves are sufficient to sustain complete starvation for several weeks; however, there are no reserves of oxygen. Therefore, in the absence of oxygen, death occurs within 2–3 minutes. Hypoxia is the most common cause of hypoenergetic states, while cerebral hypoxia represents the most frequent immediate (terminal) cause of death.



Therefore, among resuscitation measures, priority is given to interventions aimed at restoring the oxygen supply to organs. The molecular basis of energy metabolism disorders is mitochondrial dysfunction, which includes alterations in membrane permeability, inhibition of respiratory chain enzymes, and oxidation of protein sulfhydryl (SH) groups. These processes lead to the uncoupling of oxidation and phosphorylation and result in ATP deficiency.

In genetic mitochondrial diseases (such as Leigh syndrome and MELAS), the energy deficit is particularly pronounced in tissues with high oxygen demand — the brain, myocardium, and skeletal muscles. Under conditions of chemical, drug-induced, or bacterial intoxication, energy metabolism is disrupted not only due to mitochondrial damage but also because of the accumulation of toxic metabolites that inhibit enzymes of the Krebs cycle and the respiratory chain.

Disturbances of energy metabolism are consistently accompanied by secondary changes, including metabolic acidosis, impaired ionic homeostasis, reduced protein synthesis, and decreased immune activity. The clinical manifestations of such pathologies are diverse — ranging from rapid fatigue and muscle weakness to severe organ dysfunctions such as heart failure, neurological syndromes, and endocrine disorders.

In medical practice, an important area of research and therapy is the search for methods to correct these disturbances. These include the use of antioxidants, metabolic agents (such as coenzyme

compounds, riboxin, and succinic acid), hormone therapy, and modern approaches of mitochondrial medicine.

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2. Recent studies confirm that molecular defects in enzymatic systems, cofactor deficiencies, impairments in antioxidant defense mechanisms, and excessive formation of free radicals play a major role in the pathogenesis of metabolic disorders. The investigation of biochemical mechanisms underlying energy disturbances provides promising opportunities for early diagnosis, the development of targeted therapies, and personalized approaches to the correction of metabolic dysfunctions.
3. Thus, understanding the biochemical foundations of energy metabolism pathologies not only deepens fundamental knowledge of cellular metabolism but also has significant practical relevance in clinical medicine, enabling the development of novel strategies for the prevention and treatment of diseases associated with energy deficiency.
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