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PERSPECTIVE article

Targeting mitochondrial function through clinical foods: Emerging strategies for metabolic diseases

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Abstract: Metabolic problems, aging, and age-related illnesses like insulin resistance, obesity, sarcopenia, and neurodegeneration are all significantly influenced by mitochondrial dysfunction. Foods that have been shown to have physiological or therapeutic benefits are known as clinical foods, and they have gained attention as possible modulators of mitochondrial function. To improve cellular energy homeostasis and lower oxidative stress, bioactive substances as polyphenols, omega-3 fatty acids, sulforaphane, curcumin, and epigallocatechin gallate can affect mitochondrial biogenesis, mitophagy, oxidative phosphorylation, and NAD^+ metabolism. The gut-mitochondria axis, where metabolites originating from the microbiota influence mitochondrial pathways that connect metabolism, immunity, and nutrition, was highlighted by recent research. In addition to discussing molecular mechanisms and signaling pathways, this article highlights the most recent research on clinical diets that target mitochondrial function and briefly describes their potential therapeutic uses.

Introduction

Because of their numerous health advantages, bioactive substances obtained from foods and natural plants have been widely used to prevent and treat metabolic diseases, including obesity, type 2 diabetes, insulin resistance, non-alcoholic fatty liver disease, and cardiovascular disease [1-7]. Therefore, eating foods high in bioactive chemicals is a good way to lower your chance of developing metabolic illnesses [8]. Mitochondria are central to energy metabolism, redox signaling, and immune regulation. Dysfunction leads to chronic diseases such as metabolic syndrome, obesity, type 2 diabetes, neurodegeneration, and sarcopenia. Clinical foods (functional foods with therapeutic effects) are gaining attention as non-pharmacological interventions [9-11]. The pathophysiology of metabolic diseases linked to obesity involves mitochondria. Because they produce adenosine triphosphate (ATP) by oxidizing proteins, lipids, and carbohydrates, mitochondria are crucial for cellular energy metabolism [12, 13]. Mitochondrial dysfunction refers to the incapacity of mitochondria to generate and sustain enough amounts of ATP, which is caused by an imbalance in food signal input, energy generation, and oxidative respiration [14]. Several studies indicate that eating many foods affects mitochondrial function [15-17] and that obesity increases the risk of mitochondrial malfunction [18, 19]. Adopting a healthy diet and lifestyle has positive preventative and therapeutic effects on obesity and metabolic syndrome, according to basic, translational, clinical, epidemiological, and societal principles [20-26]. Grapes and berries, vegetables and fruits, turmeric, salmon, and shrimp are rich sources of dietary antioxidants like resveratrol, quercetin, coenzyme Q10, curcumin, and astaxanthin [27-31]. In addition, these bioactives have shown positive health impacts, such as anti-inflammatory and antioxidant properties that help lessen oxidative damage to mitochondria [32-34].

Mitochondria and its function: Double-membrane organelles called mitochondria are essential for cells to produce energy. The outer membrane, inner membrane, intermembrane gap, mitochondrial cristae, and mitochondrial matrix are their five separate parts. The cytosol and the mitochondrion are separated by the outer membrane. It has voltage-dependent anion-selective channels called porins that let hydrophilic molecules up to 5 kDa pass through while blocking the diffusion of bigger molecules [35, 36]. This membrane makes it easier for different nutrients, ions, and energy molecules to enter and leave. Because it lacks porins, the inner membrane is far more impermeable to most molecules than the outer membrane. As a result, just a few substances, including water, carbon dioxide, oxygen, and ammonia, may flow through the inner membrane. The inner membrane, which surrounds the mitochondrial matrix, contains electron transport chain (ETC) complexes that enable OXPHOS to produce ATP. Additionally, the matrix includes DNA, RNA, and ribosomes, and the inner membrane is divided into many folds known as cristae that expand the inner mitochondrial membrane's surface and promote ATP generation [37-39]. As the location of the tricarboxylic acid (TCA) cycle, fatty acid β-oxidation, and OXPHOS, mitochondria are commonly referred to as the cellular power factories because they generate chemical energy and heat through the metabolism of nutrients. Using reduced β-nicotinamide adenine dinucleotide (NADH) and flavin adenine dinucleotide (FAD) from glycolysis and the TCA cycle, the ETC system transfers electrons through a sequence of redox processes to molecular oxygen, reducing it to generate water. Thermogenesis is the mechanism by which mitochondrial uncoupling proteins discharge the electrochemical gradient as heat [40, 41].

Mitochondrial dysfunction: A reduction in the mitochondria's capacity to generate enough ATP through OXPHOS in response to energy needs is a hallmark of mitochondrial malfunction. Reductions in mitochondrial biogenesis, mitochondrial membrane potential, and the activity of mitochondrial oxidative proteins as a result of reactive oxygen species (ROS) buildup may be the cause of this dysfunction. In mammalian cells, ROS are mostly produced in mitochondria [42, 43]. Natural byproducts of oxygen metabolism during the OXPHOS process, reactive oxygen species (ROS) including hydroxyl radicals (OH^-), hydrogen peroxide (H_2O_2), and superoxide anions ($O2^-$) can harm mitochondria and cellular components like DNA, proteins, lipids, and other molecules [44, 45].

Diet effect on mitochondria: Increased production of ROS and dysregulated bioenergetics are frequently linked and prevalent in human illness [46, 47]. Thus, there may be therapeutic promise in avoiding this imbalance. It's interesting to note that some diets have previously been shown to have an impact on the oxidative state of mammalian cells and mitochondrial bioenergetics. Bruckbauer and Zemel, for instance, discussed how increased dairy intake affects mitochondrial biogenesis and Sirtuin (Sirt) 1 activation. They employed human muscle and adipose tissue as well as an *in vitro*/*in vivo* method to achieve this. For four weeks, participants who were overweight or obese were either fed a diet heavy in dairy or one based on soy. Serum samples were then collected and applied to human muscle cells and adipocytes in culture. According to their findings, the high dairy diet group's serum markedly raised Sirt1 activity and gene expression in both muscle and adipocyte cells. Increased mitochondrial biogenesis was noted in the same samples, as evidenced by the overexpression of important genes (PGC-1α, UCP2, UCP3, and NRF1). These results imply that the control of Sirt1-mediated mitochondrial activity may be influenced by dairy diets [48].

The research by Garcia-Roves and others [47] provides another illustration. This demonstrated increased expression of enzymes involved in fatty acid oxidation, the citrate cycle, and OXPHOS, as well as a markedly increased mitochondrial DNA (mtDNA) copy number, in rats fed a high-fat diet and given daily heparin injections to increase plasma-free fatty acids. enhanced activation of the peroxisome proliferator-activated receptor δ (PPARδ), which is frequently linked to enhanced mitochondrial biogenesis, was noted by the authors [50]. Peroxisome proliferator-activated receptor gamma coactivator-1α (PGC-1α), a master regulator of mitochondrial biogenesis and oxidative metabolism, is expressed via a post-transcriptional mechanism rather than by raising mRNA levels when PPARδ is activated. This results in a gradual increase in

mitochondrial content [51]. Moreover, Leduc-Gaudet et al. demonstrated that juvenile rats fed a high-fat diet for 14 days exhibited no significant changes in muscle mass, body weight, or energy expenditure. But in the animals' muscle areas, this diet greatly increased the mitochondria's ability to oxidize fatty acids, which eventually affected mitochondrial respiration [52]. On the other hand, mitochondrial content and respiration rates with traditional complex I and II substrates did not alter. The same study's authors showed that, in comparison to control samples, certain skeletal muscle tissue from rats given a high-fat diet shows increased mitochondrial fission and decreased fusion. High-fat diet groups did not exhibit any changes in mitochondrial ROS generation and coupling efficiency, despite the observation of elevated mRNA levels of mitochondrial uncoupling proteins (UCP2 and UCP3, which can help reduce ROS generation and regulate energy metabolism) and mitochondrial lipid transport proteins (CPT1b and CPT2) [53]. Another group of investigators looked more closely at the connection between particular diets and the oxidative state of mitochondria. Ribeiro and others [54] showed that mice's gut microbiota is significantly altered by a diet rich in fat and cholesterol. Increased metabolism of short-chain fatty acids, such butyrate and propionate, was seen in the same model. Adult neurogenesis is disturbed as a result of these alterations, which are linked to increased oxidative stress and cell death in parts of the animal's cortex and hippocampus. The authors noted how the observed reduced expression of Sirt3 and adaptive increases in total manganese superoxide dismutase 2 levels may indicate an imbalanced radical scavenger system as the molecular mechanism behind these effects. In the same study, mice given the changed diets showed higher levels of mitochondrial biogenesis than the control group, as evidenced by higher levels of Tfam (mitochondrial transcription factor A) expression and mtDNA copy number. Ribeiro and others [54] came to the conclusion that although this increase in mitochondrial biogenesis supports neurogenesis and energy generation, it is linked to an increase in ROS production.

Clinical aspect: Most experts believe that the mitochondria's primary function is to produce energy for cellular needs. Because of this, the mitochondria can support cellular metabolism, redox signaling, and ion homeostasis. Cellular survival and health depend on each of these factors [55]. The pathogenesis or progression of many human diseases or pathophysiologies, such as diabetes, cancer, neurodegenerative and cardiovascular diseases, sepsis, traumatic injury, inflammation, aging, frailty, and loss of skin elasticity, is suggested by the dysregulation of their function. The precise role and mechanisms through which the mitochondria contribute to human diseases remain unclear, creating new challenges for basic and translational biomedical science, despite the scientific community's intense focus on mitochondrial physiology-one study out of every 154 studies indexed in PubMed since 1998 has examined mitochondrial function [56]. recently, oxidative stress produced by the mitochondria is either the primary cause or a secondary exacerbating factor that drives a number of tissues pathophysiologies [57]. On the other hand, dietary interventions like the ketogenic diet, fasting, or Mediterranean diet can alter the molecular environment of previously malfunctioning mitochondria in a number of conditions, such as cancer, Alzheimer's disease, and heart failure, reducing a number of symptoms and improving quality of life. Fasting and Mediterranean die have been shown to increase longevity because they can keep the mitochondria in a fit state. Clinicians and other scientists are able to maximize their treatments that control cellular powerhouses and affect human health since food is a regular habit.

Conclusion: Using a wide range of models, researchers have examined the possible benefits of dietary interventions on mitochondrial function in recent decades. Furthermore, research has been done on how particular diets affect mitochondrial parameters that are dysregulated in particular disorders. Low-glycemic diets, for instance, have been demonstrated to benefit diabetic and pre-diabetic patients by lowering glycated hemoglobin, fasting glucose, body mass index, and cholesterol; however, they have no effect on other significant indicators, such as triglyceride levels and fasting insulin. Additionally, the Mediterranean diet, which is high in monounsaturated fats and antioxidants, has been associated with neuroprotective benefits that appear to be somewhat related to mitochondrial function.

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