Conclusions

From the results of the survey obtained, it can be concluded that not many people are aware of glycogen storage diseases. Though people are aware about diabetes mellitus, there are other diseases that are related to the metabolism of carbohydrate that are not considered. Not many acknowledge the fact of the types of diabetes and types of glycogen storage diseases. People know more about type 2 diabetes rather than type 1 diabetes mellitus. Further studies are needed to be done on the awareness on the pathology of carbohydrate metabolism and to help spread this knowledge to all study institutes.

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LIPID METABOLISM IN MITOCHONDRIAL AND METABOLIC DISEASES

Introduction

Metabolic diseases, such as obesity and type 2 diabetes, are prevalent across all age groups, with lipid metabolism playing a key role in their development. [2]. Mitochondria, the cellular powerhouses responsible for generating energy, are crucial for maintaining energy balance in metabolic tissues. [6]. Adipose tissue, comprising white (WAT) and brown (BAT) types, stores and expends energy, respectively. [6]. Mitochondrial dysfunction in adipocytes has been linked to obesity and type 2 diabetes, highlighting the importance of mitochondrial health in preventing metabolic disorders. [6]. By regulating mitochondrial biogenesis and dynamics in adipocytes, it is possible to mitigate the risk of obesity and associated conditions. Mitochondria play a vital role in metabolism by converting food into energy, producing essential molecules, and maintaining redox equilibrium. reactive oxygen species, highly reactive molecules, can contribute to cellular damage and the development of metabolic disorders when produced excessively [5].

Goal

To investigate the regulation of mitochondrial function in order to develop treatments for lipid metabolic diseases. The study aims to identify physiological and biochemical factors that contribute to these conditions.

Material and methods of research

PubMed, Google scholar, Wikipedia and other databases were referred to collect proper information. I used some keywords like “metabolism” OR “homeostasis” OR “syndrome” OR “dysfunction” to gather and assess information regarding the topic. Any primary or secondary information related to this topic was included in the article.

The results of the research and their discussion

The lifestyle of a person can highly affect the controlling of metabolic disorders [2]. And mitochondria are important organelles that affect the physiological role of differentiation and lipid regulation. Specifically, an increase in mitochondrial metabolism is indicative of adipocyte development. Targeting mitochondria as a therapeutic methodology, metabolic disease includes brown adipose tissue thermogenesis and white adipose tissue browning, mitochondrial targeted antioxidants, Exercise and caloric restriction, and natural dietary components [1].

1. Brown adipose tissue thermogenesis and white adipose tissue browning:

By raising energy expenditure, mitochondrial activation in brown adipose tissue (BAT) has become a safe means of managing and preventing metabolic diseases. An alternative to BAT thermogenesis has been suggested recently; WAT browning. Numerous environmental variables including hormones, prolonged cold exposure, physical activity and environmental enrichment, affect the browning process. Exposure to cold accelerates the clearance of plasma lipid by triglyceride absorption into BAT, hence improving dyslipidemia and insulin resistance. PGC1-α is an ideal target because of its important function in BAT adaptive thermogenesis. [1]. [6]. Relevant PGC1-α activation mechanisms are silent information regulator sirtuin1 and the upstream stimulation of estrogen-related receptor α.

2. Mitochondrial targeted antioxidants:

The antioxidant redox system which gets rid of a lot of different kinds of oxidants such as metals, lipid peroxides and reactive oxygen species oversees maintaining redox equilibrium. One etiology of metabolic is known to be oxidative stress brought on by malfunctioning mitochondria. Because metabolic and neurological disorders are linked to mitochondrial dysfunction. Treatment of adipocytes with the mitochondria targeted antioxidant lipoic acid increases oxygen consumption rate and fatty acid oxidation and promotes the expression of genes related to mitochondrial biogenesis, including PGC1-α, nuclear respiratory factor A and mitochondrial transcription factor A. [5]. [6].

3. Exercise and caloric restriction.

By enhancing mitochondrial biogenesis, respiration and density in skeletal muscles of diabetes, exercise improves systemic insulin sensitivity. Caloric restriction without starvation is a potential nongenetic dietary methodology that helps prevent metabolic disorders and lengthen life in addition to exercise. According to current guild lines, increasing physical activity levels at a moderate intensity is crucial for improving muscular, cardiorespiratory and fat mass reduction, all of which lower the risk of developing metabolic disorders [2].

4. Natural dietary compounds.

Many metabolic organs including the liver, adipose tissue and skeletal muscles have diminished mitochondrial activity and oxidative capability in metabolic disorders. Numerous natural substances found in food including polyunsaturated fatty acids have been linked to the prevention and even treatment of metabolic illnesses, according to recent research. In rats and humans for example α-linoleic acid, a polyunsaturated fatty acid obtained from plants promotes mitochondrial density and fatty acid oxidation which has anti-obesity properties [1, 7].

Conclusion

The optimal approach to addressing metabolic disorders involves prevention, starting from infancy through the adoption of a healthy lifestyle that includes adequate sleep, exercise, and

diet. Extensive research has explored the crucial role of mitochondria in energy provision and the maintenance of metabolic balance. Mitochondria in adipocytes regulate insulin sensitivity, adipocyte differentiation, and white adipose tissue browning, with mitochondrial dysfunction contributing to various metabolic disorders such as obesity and type 2 diabetes.

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SYSTEMIC JUVENILE RHEUMATOID ARTHRITIS: INCIDENCE DUE TO MIF-173C MUTATION

Introduction

Systemic juvenile rheumatoid arthritis (SJRA), also known as juvenile chronic or idiopathic arthritis, is a disease that affects approximately 11% of patients with this condition and is characterized by clinical homogeneity. Despite advances in treatment, many children with this disease still face early joint destruction, leading to the need for surgical interventions.Additionally, a significant portion of patients (48%) continue to experience symptoms even after 10 years.

Research has shown that certain genetic variations, specifically polymorphisms in the IL-6 and MIF genes, are associated with an increased susceptibility to this disorder. Macrophage migration inhibitory factor (MIF) has emerged as a novel cytokine that may play a crucial role in linking rheumatoid arthritis to atherosclerosis, highlighting the complex interplay between these diseases.

JRA is present in all over the world approximately 3 million children and young adults are sufferingfrom JRA some countries have (see table 1).

Table 1 – Percentage of systemic juvenile rheumatoid arthritis

Countries	Percentage
UK	1
US	0.5–1
INDIA	0.9
AFRICA	0.16