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Perspective

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Physical Exercise Aims to Mitigate Fatty Liver Disease

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Introduction

Exercise not only strengthens muscles but also helps to prevent fatty liver disease.

The most common hepatic pathology linked to the global obesity epidemic is non-alcoholic fatty liver disease (NAFLD). The major cause of non-alcoholic steatohepatitis (NASH) and liver cirrhosis is non-alcoholic fatty liver disease (NAFLD). It has a strong link to hepatocellular carcinoma development. NAFLD is also linked to an increased risk of death.

The condition is thought to be caused by a mismatch between energy intake and consumption. This causes fat deposits in the liver and decreases mitochondrial function over time, both of which are risk factors for the development of hepatic insulin resistance and inflammation. NAFLD is currently one of the most serious health problems, with far-reaching implications for health-care systems and people's quality of life. It affects nearly one-quarter of the world's population [1].

Increased regular physical activity is promoted by health organisations around the world as a potential treatment for NAFLD, as well as the prevention of steatohepatitis and related metabolic comorbidities.

NAFLD can be efficiently treated with a variety of exercise therapies (e.g., endurance and resistance exercise, a mix of both, or an unstructured increase in daily physical activity) [2].

Highlights of Study

- Independent of exercise, a high-energy diet improves mitochondrial respiration in the liver.
- When a high-energy diet is paired with exercise, substrate oxidation is separated from fat synthesis.
- A high-energy diet mixed with exercise lowers the production of complex I in the liver.
- A well-trained skeletal muscle relieves the liver of its burden of excessive substrates.
- A comprehensive database of mitochondrial adaptations to a high-energy diet and exercise regimen.
- Exercising can help avoid fatty liver disease, which is caused by overeating.

Mice were fed a high-energy diet in a study done by experts. Treadmill training was also given to some of the mice. The researchers looked for changes in the transcriptome, mitochondrial proteome, lipid content, and mitochondrial activity in the animals' livers and muscles after the six-week intervention [3].

Training regulated essential glucose and fructose breakdown enzymes in the liver, as well as mitochondrial pyruvate metabolism, according to the findings. The substrate load for mitochondrial respiration and lipid production can be lowered in this way. As a result, the liver stores less fat, and particular lipids such diacylglycerol species are reduced [4-5].

Furthermore, in exercise-trained mice, glucose management improves. Furthermore, enhanced skeletal muscle respiratory capacity decreases metabolic stress on the liver.

The systems biology data provide a thorough understanding of the liver and muscle's molecular response to a high-energy diet, training, and combinatorial effects [6].

The findings are consistent with current clinical studies in which inhibitors are being tested against some of the targets identified here, such as the mitochondrial pyruvate transporter; they also demonstrate that regular physical activity regulates multiple targets at the same time, key nodes of metabolic pathways, and an effect that cannot be achieved with monotherapy.

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