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Opinion Article

Role of Combined Metabolic Stimulators in Reducing the Liver Fat

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DESCRIPTION

Hepatic steatosis, steato hepatitis, hepatic fibrosis, and cirrhosis are among the pathologies that make up Nonalcoholic Fatty Liver Disease (NAFLD), which is defined as the accumulation of less than 5% of hepatic fat that is unrelated to alcohol consumption and other liver illnesses. The metabolic syndrome and insulin resistance are directly related to NAFLD. The incidence of obesity and diabetes is rapidly increasing, and this is the main cause of chronic liver disease. Hepatic steatosis affects at least one in four people worldwide. The effectiveness of current management options, such as lifestyle change, increased physical activity, and dietary intervention, is limited. There are immidately needed effective therapy alternatives with long-term safety for NAFLD, for which no medications have been approved. Although research is providing a basis for treatments, early clinical trials of pharmaceuticals that target specific pathways have often failed. For this complex condition, combining drugs that lessen lipid buildup and liver damage has been suggested as a more effective therapy approach. Insufficient hepatic fat elimination through fatty acid oxidation is a major factor in the pathophysiology of NAFLD. Limited serine availability, decreased *de novo* glutathione synthesis, and altered Nicotinamide Adenine Dinucleotide (NAD+) metabolism were found to be three key metabolic features of hepatic steatosis in our prior research using in-depth multi-omics profiling and hepatocyte-specific integrated networks.

In this study, Combined Metabolic Activators (CMA) was used to treat NAFLD. These CMAs included L-carnitine tartrate to help mitochondria take up fatty acids from the cytosol, nicotinamide riboside to help induce hepatic mitochondrial -oxidation and help fatty acids transfer through the mitochondrial membrane, and L-serine and N-acetyl-l-cysteine to help lower oxidative stress. The injection of these metabolic stimulators would increase the absorption and oxidation of fatty acids in the mitochondria, decrease hepatic fat, and inflammation. The term "Non-Alcoholic Fatty Liver Disease" (NAFLD) describes the buildup of too much fat in the liver. By administering Combined Metabolic Activators (CMA) stimulates fat oxidation, reduces the oxidative stress that results, activates mitochondria, and eventually excreat out extra fat from the liver in animal trials and human kinetic studies. Here, a 10-week, placebo-controlled research in NAFLD patients to evaluate the safety and effectiveness of CMA is carried out. After adjusting for weight loss, the CMA dramatically reduced hepatic steatosis and levels of aspartate aminotransferase, alanine aminotransferase, uric acid, and creatinine while finding no differences on these variables in the placebo group. This study identifies the main participants in the host-microbiome interactions as well as the underlying molecular mechanisms driving the decreased hepatic fat and inflammation in NAFLD patients by merging clinical data with plasma metabolomics, inflammatory proteomics, oral and gut metagenomics data. The CMA can be used to create a pharmaceutical therapy plan for NAFLD patients.

A CMA based on four components has been developed based on data-driven modelling and systems biology to raise the liver's fat oxidation and promote mitochondrial function in the cells, hence stimulating hepatic mitochondrial fatty acid absorption and -oxidation and decreasing oxidative stress. A randomised, placebo-controlled phase 2 trial to look at the effectiveness and safety of CMA in NAFLD patients was carried out in order to test this theory in a clinical environment. In this study, the key outcome variable, measured by MRIPDFF after 70 days, was found to be decreased by CMA (10%). Reductions in the levels of the secondary endpoints, serum ALT (39%) and AST (30%), were also significantly improved.

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