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The Effects of Fructose and Food Preservatives on Steatotic Liver Disease Associated with Metabolic Dysfunction (MASLD)

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Introduction

Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD), previously referred to as Non-Alcoholic Fatty Liver Disease (NAFLD), is a growing public health concern, with a global prevalence that has seen a dramatic increase over the past few decades. MASLD encompasses a spectrum of liver conditions, ranging from simple hepatic steatosis to more severe stages of liver injury, including Non-Alcoholic Steatohepatitis (NASH), cirrhosis, and hepatocellular carcinoma. The disease is strongly linked to various metabolic disorders such as obesity, insulin resistance, and type 2 diabetes, with underlying mechanisms involving inflammation, oxidative stress, and disrupted lipid metabolism [1].

Two major contributors to the rise in MASLD are dietary factors, specifically the excessive intake of fructose and the consumption of processed foods containing various food preservatives. These factors exacerbate metabolic dysfunction and influence liver pathology in ways that have garnered increasing attention from researchers. Fructose, a simple sugar predominantly consumed through High-Fructose Corn Syrup (HFCS) in sugary beverages and processed foods, has been implicated in promoting hepatic steatosis, insulin resistance, and inflammation, all of which are hallmarks of MASLD. Likewise, food preservatives such as artificial sweeteners, synthetic antioxidants, and emulsifiers have been shown to disrupt gut microbiota, alter metabolic pathways, and increase inflammation, all of which may contribute to the pathogenesis of MASLD [2].

Description

The liver plays a crucial role in metabolic regulation, including the storage and synthesis of lipids, glucose, and proteins. In the context of MASLD, the liver becomes overwhelmed with the accumulation of excess triglycerides, resulting in hepatic steatosis. This accumulation is primarily driven by an imbalance between lipid synthesis and clearance. The liver synthesizes fatty acids from carbohydrates and proteins, and when energy intake exceeds the body's needs, these fatty acids are stored as triglycerides in hepatic cells. In healthy individuals, the liver maintains a balance between fat storage and export, but in the context of metabolic dysfunction, this equilibrium is disrupted, leading to fat accumulation in hepatocytes. The primary sources of excess calories contributing to steatosis are dietary carbohydrates, particularly fructose, and the overconsumption of processed foods rich in preservatives. These dietary factors not only increase fat synthesis but also impair the liver's ability to clear lipids effectively, promoting fat storage [3].

Fructose is metabolized predominantly in the liver, where it bypasses key regulatory steps in glycolysis and is readily converted into triglycerides. Unlike glucose, which undergoes a regulated metabolic pathway, fructose is metabolized by the enzyme fructokinase, leading to the rapid production of intermediates that promote De Novo Lipogenesis (DNL), a process in which fatty acids are synthesized from non-lipid precursors. This promotes the accumulation of triglycerides in hepatocytes, setting the stage for hepatic steatosis. The metabolism of fructose also leads to an increase in oxidative stress. The rapid conversion of fructose generates reactive oxygen species (ROS), which can damage cellular structures and promote inflammation, both of which are key contributors to liver injury and progression from simple steatosis to Non-Alcoholic Steatohepatitis (NASH). ROS also activate signaling pathways such as the nuclear factor-kappa B (NF-κB) pathway, which is involved in promoting inflammatory responses in the liver. Chronic oxidative stress can, therefore, exacerbate liver injury and promote fibrosis, a precursor to cirrhosis. Moreover, fructose consumption is associated with an increase in insulin resistance. Elevated fructose intake has been shown to impair insulin signaling in peripheral tissues, which further exacerbates the dysregulated lipid metabolism seen in MASLD. Insulin resistance not only affects glucose metabolism but also leads to increased lipogenesis in the liver, contributing to steatosis [4].

The interplay between fructose and food preservatives in the context of MASLD is a complex and underexplored area of research. However, it is likely that these dietary factors work synergistically to exacerbate liver injury. For example, the consumption of high-fructose diets can lead to insulin resistance and liver fat accumulation, creating an environment in which food preservatives, particularly emulsifiers and artificial sweeteners, can further promote inflammation and oxidative stress. The combined effect of excessive fructose and food additives on gut microbiota composition may also amplify the inflammatory response, accelerating the progression from simple steatosis to more severe forms of liver disease, such as NASH. Furthermore, both fructose and food preservatives can disrupt normal metabolic processes, leading to alterations in lipid metabolism, insulin sensitivity, and inflammatory pathways. This creates a vicious cycle in which diet-induced metabolic dysfunction exacerbates liver damage, increasing the risk of liver fibrosis and other complications associated with MASLD [5].

Conclusion

MASLD is a multifactorial liver disease associated with metabolic dysfunction, and it is strongly influenced by dietary factors such as fructose and food preservatives. Fructose, primarily consumed through sugary beverages and processed foods, contributes to liver fat accumulation, oxidative stress, insulin resistance, and inflammation, all of which promote the progression of MASLD. Food preservatives, including emulsifiers, artificial sweeteners, and synthetic antioxidants, can also disrupt metabolic pathways and contribute to liver injury by altering gut microbiota and increasing systemic inflammation. Given the growing prevalence of MASLD and its potential to progress to more severe forms of liver disease, it is critical to consider dietary modifications and interventions aimed at reducing fructose and food preservative consumption. By promoting healthier eating habits, improving gut health, and utilizing pharmacological approaches, it may be possible to mitigate the effects of these dietary factors on liver health. More research is needed to better understand the complex interactions between diet and liver disease, but

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current evidence underscores the importance of addressing dietary factors as part of a comprehensive approach to preventing and treating MASLD.

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Conflict of Interest

None.

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