Non-alcoholic fatty liver disease

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Commentary

Non-Alcoholic Fatty Liver Disease (NAFLD) is a serious public health concern across the world. NAFLD (including simple steatosis and steatohepatitis) is defined by changes in hepatic lipid metabolism, which can progress to serious liver consequences such as cirrhosis and hepatocellular cancer. As a result, a thorough assessment of lipid abnormalities in NAFLD patients' livers is critical. Lipidomics systems based on mass spectrometry enable in-depth investigation of lipid changes in a variety of human illnesses, including NAFLD. This review summarises current research on lipid alterations associated with NAFLD and related complications, with a focus on changes in long-chain and short-chain fatty acid levels in both serum and liver tissue, as well as hepatic expression of genes encoding the enzymes that catalyse lipid interconversions.

Chronic liver diseases have become a serious public health concern globally in recent decades. Obesity and metabolic syndrome are well-established risk factors for Chronic Liver Disease (CLD). Obesity affects about 400 million individuals, and it is predicted that 75% of them will develop Non-Alcoholic Fatty Liver Disease (NAFLD). NAFLD refers to a group of liver disorders that range from excessive fat build up in the organ (simple steatosis) to Non-Alcoholic Steato-Hepatitis (NASH). The presence of hepatocyte mortality and inflammation distinguishes NASH from Simple Steatosis (SS), and it is strongly linked to the development of severe liver disorders such as fibrosis, cirrhosis, and even Hepatocellular Cancer (HCC). NAFLD is linked to insulin resistance, which leads to increased lipolysis in adipose tissue. As a result, the levels of serum Free Fatty Acids (FFA) and hepatic Triacylglycerols Rise (TAG). It might also be caused by hereditary abnormalities and/or rapid body mass reduction following bariatric surgery. Furthermore, fatty liver is a typical side effect of several medicines (for example, tamoxifen).

The global prevalence of NAFLD is believed to be 25%, yet it is not evenly spread around the world. In reality, Western nations including the United States have recorded more instances. However, recent data suggest that South America has the greatest frequency of NAFLD. Nonetheless, given there are no precise and non-invasive diagnostics for determining the severity of NAFLD, these findings should be taken with care. NASH is primarily identified in clinical practise by histological examination of liver samples, which is an invasive process. Because of the hazards of the operation, a liver biopsy is typically performed only in cases with severe symptoms suggestive of NASH. Hence, it cannot play a significant role in either prevention or early diagnosis of NASH. As a result, new non-invasive techniques for distinguishing individuals with SS from those with NASH should be developed, as the latter is a significant risk factor for more severe liver consequences such as cirrhosis and HCC. Lipid build up causes large-droplet macrovesicular steatosis as well as irreversible small-droplet steatosis. Another NAFLD-related liver damage is hepatocyte ballooning, which is characterised by enlarged hepatocytes with unusual cytoplasm. Changes in tissue microarchitecture cause greater resistance to blood flow via the liver and portal hypertension. The last stage of cirrhosis is gradual, continuous, and irreversible fibrosis, which renders liver functions ineffective. Cirrhosis of the liver is an excellent state for the formation of HCC, and it is predicted that almost 80% of cirrhotic individuals will develop HCC.

Lipids are a diverse collection of organic molecules that perform a variety of activities, including energy storage and the creation and stability of intra and extracellular membranes. Lipids are also precursors of bioactive chemicals seen in the bloodstream, and they have the ability to influence gene expression. Fatty Acids (FA) are found in almost every lipid structure. FAs can be found in the human body in two forms: freestanding FFAs and esterified (e.g., in TAG and phospholipids (PL)). Notably, the name FFA may be deceptive because these molecules are generally linked to albumin or other FA-binding proteins in the circulation.

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