



Editorial Note on Non-Alcoholic Fatty Liver Disease

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Abstract

Nonalcoholic fatty liver disease (NAFLD), hepatic manifestation of metabolic syndrome is now the commonest chronic liver disease due to rising obesity and diabetes. NAFLD progresses from simple steatosis (NAFL) to steatohepatitis (NASH) and cirrhosis. In presence of suitable genetic and environmental factors (diet/physical activity/ gut dysbiosis), insulin resistance (IR) and obesity results in adipose dysfunction, which triggers proinflammatory response, decreased lipolysis, increased de-novo lipogenesis and further increased IR. These occasions increment free unsaturated fat (FFA) motion to liver, which prompts triglyceride gathering (NAFL). Toxic levels of FFA in liver trigger increased β -oxidation and mitochondrial dysfunction (MD). Obesity, homocysteine and environmental factors trigger endoplasmic reticulum stress (ERS). MD and ERS result in reactive oxygen species (ROS) production. ROS activates antioxidant mechanisms (consisting of enzymes like Superoxide dismutase, catalase, glutathione peroxidase, glutathione reductase, glutathione transferase; and non-

enzymes like vitamin A, C, E, β -carotene and glutathione) which scavenges them, but over production of ROS results in depletion of antioxidants.

Keywords: Homocysteine, Non-alcoholic fatty liver disease, Gender, Body mass index, Interaction

INTRODUCTION:

Homocysteine adds to ROS production and suppresses antioxidants. Oxidative pressure brings about proinflammatory cytokine creation, lipid peroxidation (estimated by Malondialdehyde) and protein adducts creation prompting cell injury, irritation and cell demise prompting NASH. In addition, it triggers hepatic stellate cell activation leading to fibrosis and subsequently cirrhosis. Oxidative stress also produces DNA damage leading to future hepatocellular carcinoma. Along these lines, oxidative pressure stays key to advancement of NASH and cirrhosis. In clinical practice, differentiating NAFL and NASH requires liver biopsy because non-invasive scoring systems are not sensitive. Measuring homocysteine and enzymes (like glutathione transferase, glutathione peroxidase, catalase, etc.) may prove helpful to define progress to NASH. Also targeting these molecules by newer therapeutic strategies may halt progression of NAFLD.

Non-alcoholic greasy liver illness (NAFLD), which incorporates a range of conditions related with lipid testimony in hepatocytes, is the most widely recognized liver ailment. Around the world, the general predominance of NAFLD determined by imaging was 25.2% to have the most elevated commonness rates were accounted for from South America (30.5%) and the Middle East (31.8%) . NAFLD influences over a fourth of the populace, and its predominance is as yet expanding quickly because of significant changes in way of life and maturing. NAFLD is regularly connected with metabolic hazard factors, for example, corpulence; type two diabetes, dyslipidemia, and insulin obstruction. Notwithstanding hepatic complexities, NAFLD is likewise connected with genuine fundamental outcomes. NAFLD has been generally acknowledged to essentially expand the dismalness and mortality of cardiovascular maladies.

NAFLD is considered as a metabolic disorder that results from complex interaction between genetic, hormonal and nutritional factors.¹ Recent evidence suggests that several genetic risk factors predispose to the development and progression of NAFLD.¹⁸ For example, polymorphisms of PNPLA3, TM6SF2, FTO, LIPA, IFN λ 4 HFE, and HMOX-1 genes have been found to be associated with development/progression of the disease. Hyperinsulinemia and IR lead to increased adipocyte lipolysis and circulating free fatty acids (FFAs) that are taken up by hepatocytes, initiating various complex metabolic pathways that lead to NAFLD

Homocysteine was altogether connected with the pervasiveness of NAFLD, especially in female, hefty or non-smoking grown-ups.

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