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Editorial

Metabolic Dysfunction Associated Fatty Liver Disease: A Recent Endemic Due to Pollutants, Adulterants, and Junk Food (PAJ)

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Formerly known as non-alcoholic fatty liver disease (NAFLD), this disorder is characterized by liver damage akin to alcohol induced injury - steatosis, inflammation, and fibrosis - without the causative excessive alcohol intake [1]. Research showed this condition to be a physical manifestation of complex metabolic dysfunction. The heterogeneity of the disease, coupled with its hepatic, cardiovascular, and oncogenic implications, led to its current identifier: Metabolic dysfunction associated fatty liver disease (MAFLD) [2]. The prevalence of MAFLD in Asian countries (33.9%) is higher than the global average (25%) and is on the rise [3]. Ethnic risk factors and lifestyle changes have resulted in an MASLD endemic. The pathogenesis of MAFLD is complex: various pathways have been identified that lead to liver steatosis (or its extreme progression - Non-alcoholic steatohepatitis, NASH). Many more factors are suspected to affect liver health, but a concrete link with the condition is yet to be established.

Rapid industrialization has intensified ambient air pollution, which contributes significantly to the development of metabolic syndromes [4]. Particulate matter penetrates deep into the respiratory tract, crossing into the bloodstream and adsorb heavy metals and organic compounds. This indirectly facilitates hepatic fibrogenesis [5]. Nitrogen dioxide (NO_2) has also been associated with an increased risk of MAFLD, potentially by promoting hepatocyte apoptosis [6].

Prolonged exposure to NO_2, SO_2, and CO interferes with hepatic glucose and lipid metabolism, by the Reactive Oxygen Species (ROS) generated by heavy metals and thus oxidizes LDL, also leading to oxidative stress and persistent inflammatory responses [5]. Consequently, the circulating lipoproteins get altered, particularly LDL, making them harder for the body to clear and more likely to accumulate in the bloodstream [5-7]. At the same time, pollution induced inflammation signals the liver to produce more very-low-density lipoprotein (VLDL), increasing triglyceride and LDL-C levels [8]. Inflammatory mediators also reduce the production of apolipoprotein A-I, a key component of HDL, thereby weakening the body's ability to remove excess cholesterol through reverse cholesterol transport [9].

Even the household air pollution and long-term exposure to cooking fumes can further sustain this low-grade inflammatory state, increasing the release of free fatty acids from adipose tissue and promoting lipid buildup in the liver [8]. Over time, these combined effects shift the lipid profile toward higher levels of atherogenic lipids such as LDL-C and ApoB and lower levels of protective HDL [9]. Such changes not only heighten cardiovascular risk but may also contribute to the development and progression of metabolic dysfunction associated fatty liver disease (MAFLD). Despite the exact mechanisms being unknown, evidence supporting a causative association is rapidly mounting.

Beyond pollutants, diet has a major impact on the progression of MAFLD. Various components of modern diets can increase the risk of liver damage. Fast food diets and ultra-processed foods are known to induce hepatic steatosis through multiple pathways [10]. High intake of refined sugars and trans fats impairs insulin signaling, leading to increased lipogenesis and hepatic fat accumulation [11,12]. In animal models, high-sugar/high-fat diets are shown to induce glucose intolerance, insulin resistance, and elevate oxidative stress markers [13].

MAFLD is also associated with dysbiosis of the gut microbiome. Fat accumulation in the liver of MASLD patients has been shown to improve with improvements in gut microbiota dysbiosis via fecal microbiota transplant, indicating a strong relationship [14]. Processed foods contain a variety of additives to prolong shelf life and enable mass production, transport, and distribution. Many such additives - colorants, preservatives, stabilizers, sweeteners, etc. - are known to alter gut microbiota, indirectly impacting liver health [15]. Some even exhibit slight cytotoxic effects *in vitro* [15].

Microplastics, commonly ingested via food and water, cause glucose intolerance and translocate from the gut to the liver, resulting in inflammation, increased oxidative stress, and fibrosis [16]. Bisphenols, commonly found in polycarbonate plastics and epoxy can linings, have been strongly implicated in each stage of MAFLD. It is not only one pollutant from plastic. Several pollutants can be leached from plastic pollutants and that can ultimately cause MAFLD apart resulting in severe problem to environmental health [17,18].

Soil pollution is also indirectly responsible for the addition of substances that cause liver injury into the food chain. Organochlorine pesticides (OCPs) were widely used in agriculture. Despite being banned in India in 1989, they are still present in the soil in considerable amounts [19]. Additionally, high amounts of heavy metals in soil get absorbed by crops and can be transmitted through the food chain. An increase in liver enzymes is noted after exposure to OCPs, heavy metals, and biphenyls, indicating hepatic inflammation [20-22].

While India has taken steps that are instrumental in increasing awareness about the occurrences, risks, and diagnosis of NAFLD, preventive measures regarding causative environmental factors are an urgent need.

Most Indian cities, particularly metropolitan areas, experience poor air quality, characterized by high $PM_{2.5}$ concentrations and moderate NO_2 levels, drastically exacerbating the risk of NAFLD for residents [23]. To combat deteriorating air quality, the Indian government launched the National Clean Air Program (NCAP), which targets to reduce particulate matter levels by at least 40% by 2026. In the meantime, it is imperative to spread awareness about and increase access to masks and air filters to protect residents. Similarly, regulations regarding the use of pesticides and fertilizers with known or suspected harmful effects from long-term exposure must be tightened. Industry wastewater treatment requirements must also be strictly implemented to reduce toxic chemicals and microplastics in the water supply and food chain.

To mitigate the adverse effects of diet on the liver, measures must be taken to reduce hepatotoxins in food production and processing pipelines. These may include regulations on food packaging practices, such as prohibiting the service of hot food in plastic containers and promoting the use of safer, non-toxic alternatives such as paper, plant-based packaging, and bagasse [24,25]. Acceptable limits of additives may be revised, and regulations must be strengthened to reduce long-term exposure to potential hepatotoxins, especially in foods marketed towards children. While MASLD has a significant genetic component, the effects of environment and lifestyle cannot be disregarded (Figure 1). These factors must be considered when making health decisions on a personal or policy level.

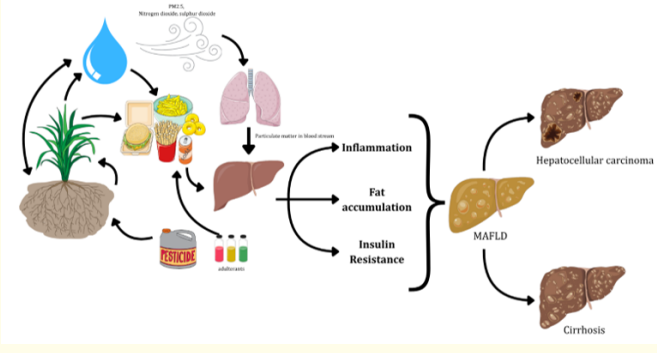

Figure 1: The impact of pollution, diet, and adulterants on liver health Certain diets (high fructose and high fat, ultraprocessed junk food with certain additives) and air pollutants (particulate matter, nitrogen dioxide and sulphur dioxide) introduce hepatotoxic substances into the body through inhalation or consumption. These substances travel through the bloodstream to the liver, where they increase oxidative stress, inflammation, fat accumulation, and cause insulin resistance. This condition is known as MAFLD and may progress to hepatocellular carcinoma or cirrhosis, if left unchecked. (Figure created with BioIcons and Canva).

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