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Impact of Ultra-processed Foods on Non-Alcoholic Fatty Liver Disease (NAFLD): A review

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Abstract
Non-alcoholic fatty liver disease (NAFLD) has emerged as one of the most prevalent chronic liver disorders worldwide, closely linked to obesity, metabolic syndrome, and unhealthy dietary patterns. The increasing consumption of ultra-processed foods (UPFs) represents a significant dietary shift associated with metabolic dysfunction. UPFs are characterized by extensive industrial processing, high levels of refined carbohydrates, added sugars, unhealthy fats, and food additives, along with reduced fibre and micronutrient density. Emerging epidemiological and clinical evidence suggests that excessive UPF intake contributes to hepatic fat accumulation, insulin resistance, systemic inflammation, and progression of NAFLD. This review summarizes current evidence regarding the relationship between ultra-processed food consumption and NAFLD development, explores underlying biological mechanisms, and discusses nutritional and public health implications. Limiting UPF intake and promoting minimally processed dietary patterns may represent an effective strategy for preventing and managing NAFLD.

Keywords: Ultra-processed foods, NAFLD, fatty liver, metabolic syndrome, diet quality, liver health

Introduction

Non-alcoholic fatty liver disease (NAFLD) is a spectrum of liver conditions characterized by excessive fat accumulation in hepatocytes in individuals who consume little or no alcohol (Sharma and Arora, 2020) [26]. NAFLD includes simple steatosis, non-alcoholic steatohepatitis (NASH), fibrosis, cirrhosis, and hepatocellular carcinoma. Globally, NAFLD affects nearly 25–30% of adults and is increasingly recognized as the hepatic manifestation of metabolic syndrome (Radu *et al*., 2023; Tewari *et al*., 2025) [23, 28].

Dietary transitions toward industrialized food systems have substantially increased the intake of ultra-processed foods (Baker *et al*., 2020) [2]. These foods are energy-dense, nutrient-poor, and rich in additives that may adversely affect metabolic and liver health. Recent studies indicate that UPFs play a significant role in obesity, insulin resistance, and chronic inflammation—key drivers of NAFLD development (Robson, 2009) [25].

Definition and Characteristics of Ultra-Processed Foods

Ultra-processed foods are industrial formulations produced mainly from refined food components and additives rather than whole foods. According to the NOVA classification system, UPFs undergo multiple processing steps and include ingredients such as refined starches, hydrogenated oils, added sugars, flavour enhancers, emulsifiers, and preservatives (Medin *et al*., 2025) [17]. Common examples include sugar-sweetened beverages, processed meats, packaged snacks, instant foods, and ready-to-eat meals (Monteiro *et al*., 2019) [18].

These products typically possess high energy density, excessive simple carbohydrates, unhealthy fats, and sodium while lacking dietary fibre, antioxidants, and essential micronutrients. Their convenience, long shelf life, affordability, and hyper-palatable characteristics promote frequent consumption, contributing to metabolic disturbances associated with liver fat accumulation (Hägele *et al*., 2023) [8, 9].

Epidemiology of NAFLD and Dietary Transition

The prevalence of NAFLD has risen parallel to increasing obesity and sedentary lifestyles worldwide. Urbanization, westernized dietary patterns, and widespread availability of processed foods have significantly altered nutritional habits (Han *et al*., 2020). High consumption of refined carbohydrates, fructose-containing beverages, and industrial fats has been strongly associated with hepatic steatosis (Basaranoglu *et al*., 2015) [3].

Populations undergoing rapid nutrition transition, including many developing countries, demonstrate increasing NAFLD incidence among both adults and adolescents (Zhang *et al*., 2021) [30]. Dietary patterns rich in ultra-processed foods are increasingly recognized as independent contributors to liver disease beyond total caloric intake (Ivancovsky-Wajcman *et al*., 2021) [12].

Association Between Ultra-Processed Food Consumption and NAFLD

1. Evidence from Observational Studies

Several epidemiological studies demonstrate a positive association between UPF intake and fatty liver disease. Individuals consuming higher proportions of dietary energy from ultra-processed foods show greater hepatic fat accumulation and elevated liver enzyme levels. Cross-sectional and cohort studies have reported higher prevalence of NAFLD among populations with frequent consumption of fast foods, processed snacks, and sugary beverages (Henney *et al*., 2023) [11].

2. Evidence from Meta-Analyses

Meta-analyses suggest that diets rich in processed and ultra-processed foods significantly increase NAFLD risk. High intake of added sugars, refined grains, and processed fats promotes metabolic abnormalities such as obesity, insulin resistance, and dyslipidemia, which collectively contribute to liver fat deposition and disease progression (Guo *et al*., 2025) [7].

Pathophysiological Mechanisms Linking UPFs to NAFLD

1. Excess Energy Intake and Obesity

Ultra-processed foods promote excessive caloric consumption due to their high palatability and low satiety value. Chronic positive energy balance leads to weight gain and visceral adiposity, increasing free fatty acid flux to the liver and promoting hepatic steatosis (Kelly *et al*., 2022) [14].

2. High Fructose and Refined Carbohydrate Load

Many UPFs contain high levels of fructose from sweetened beverages and processed foods. Fructose metabolism occurs primarily in the liver and stimulates de novo lipogenesis, triglyceride accumulation, and hepatic fat synthesis, thereby accelerating NAFLD development (Popkin *et al*., 2024) [22].

3. Insulin Resistance and Metabolic Dysfunction

Repeated exposure to refined carbohydrates causes chronic hyperinsulinemia and insulin resistance (Agius, 2013) [1]. Impaired insulin signaling increases hepatic lipid synthesis while reducing fatty acid oxidation, resulting in fat accumulation within hepatocytes (Muoio and Newgard, 2008) [19].

4. Inflammation and Oxidative Stress

UPFs increase systemic inflammation through excess sugars, saturated fats, and processing by-products. Oxidative stress damages hepatocytes, contributing to progression from simple steatosis to non-alcoholic steatohepatitis (NASH). (Hägele *et al*., 2023) [8, 9]

5. Gut Microbiome Dysbiosis

Low fibre intake and food additives alter gut microbiota composition, increasing intestinal permeability and endotoxin translocation. This gut-liver axis disturbance promotes hepatic inflammation and fibrosis progression (Frazier *et al*., 2011) [6].

6. Role of Food Additives and Packaging Chemicals

Emulsifiers, artificial sweeteners, and contaminants such as bisphenol A may disrupt metabolic regulation and liver function. These compounds may exacerbate lipid accumulation and inflammatory responses within the liver (Del Campo *et al*., 2018) [5].

Clinical and Metabolic Consequences

High consumption of ultra-processed foods is associated with several metabolic abnormalities linked to NAFLD progression:

Central Obesity

High consumption of ultra-processed foods contributes significantly to central obesity, characterized by excessive accumulation of visceral adipose tissue around abdominal organs (Juul *et al*., 2025) [13]. Ultra-processed foods are typically energy-dense, high in refined carbohydrates and unhealthy fats, and low in dietary fibre, leading to increased caloric intake and reduced satiety (McClements, 2025). Visceral fat plays a critical role in NAFLD development by increasing free fatty acid release into portal circulation, directly promoting hepatic fat deposition and insulin resistance. Central obesity is therefore considered a primary metabolic driver linking unhealthy dietary patterns with fatty liver progression (Liu *et al*., 2010) [15].

Dyslipidemia

Ultra-processed food intake is strongly associated with dyslipidemia, a metabolic condition marked by elevated triglycerides, increased low-density lipoprotein (LDL) cholesterol, and decreased high-density lipoprotein (HDL) cholesterol. Diets rich in added sugars, trans fats, and refined carbohydrates stimulate hepatic lipid synthesis and impair lipid clearance mechanisms. These alterations increase lipid accumulation within hepatocytes and accelerate progression from simple steatosis to more severe liver injury. Dyslipidemia also contributes to systemic atherosclerosis, thereby increasing both hepatic and cardiovascular disease risks (Nouri *et al*., 2023) [20].

Type 2 Diabetes Mellitus

Frequent consumption of ultra-processed foods promotes insulin resistance and glucose dysregulation, significantly increasing the risk of type 2 diabetes mellitus (Chatterjee and Tewari, 2025) [4, 28]. High glycemic load and rapidly absorbable carbohydrates present in UPFs cause repeated postprandial glucose spikes, leading to chronic hyperinsulinemia and impaired insulin signaling. Insulin resistance enhances hepatic fat accumulation by stimulating lipogenesis while inhibiting fatty acid oxidation. The coexistence of NAFLD and type 2 diabetes worsens disease severity and increases the likelihood of inflammation, fibrosis, and liver-related complications.

Hypertension

Hypertension is another major metabolic consequence associated with excessive intake of ultra-processed foods. These foods often contain excessive sodium used for preservation and flavor enhancement, which contributes to fluid retention, vascular stiffness, and increased blood pressure. Chronic hypertension reduces hepatic microcirculation and exacerbates oxidative stress and inflammation, accelerating liver damage. Additionally, hypertension commonly coexists with obesity and insulin resistance, forming part of the metabolic syndrome closely linked with NAFLD progression (Wang *et al*., 2022) [29].

Increased Liver Enzymes (ALT and AST)

Elevated liver enzymes, particularly alanine aminotransferase (ALT) and aspartate aminotransferase (AST), are important biochemical indicators of liver injury associated with ultra-processed food consumption. Excessive intake of refined sugars and unhealthy fats promotes hepatic inflammation, oxidative stress, and hepatocyte damage, resulting in leakage of these enzymes into the bloodstream (Tan *et al*., 2008). Persistent elevation of ALT and AST reflects ongoing liver injury and may indicate progression from simple fatty liver to non-alcoholic steatohepatitis (NASH), emphasizing the clinical relevance of dietary patterns in liver health monitoring.

Fibrosis Progression Risk

Long-term exposure to ultra-processed food–related metabolic disturbances increases the risk of liver fibrosis progression in individuals with NAFLD. Chronic inflammation, oxidative stress, insulin resistance, and lipid toxicity stimulate activation of hepatic stellate cells, leading to excessive collagen deposition and scar formation within liver tissue (Ramos-Tovar and Muriel, 2020) [24]. Progressive fibrosis can ultimately advance to cirrhosis and

liver failure if left untreated. The presence of fibrosis also significantly elevates cardiovascular morbidity and mortality, highlighting the importance of dietary modification as a preventive and therapeutic strategy in NAFLD management (Polyzos *et al*., 2021) [21].
These factors collectively increase the likelihood of advanced liver disease and cardiovascular complications.

Nutritional Management and Preventive Strategies
Dietary modification remains the cornerstone of NAFLD prevention and treatment. Evidence supports the following approaches:

- Reduction of ultra-processed food intake
- Adoption of minimally processed, whole-food diets
- Increased consumption of fruits, vegetables, legumes, and whole grains
- Replacement of refined carbohydrates with complex carbohydrates
- Increased intake of omega-3 fatty acids and antioxidants
- Limiting sugar-sweetened beverages and processed meats

Dietary patterns such as the Mediterranean diet demonstrate significant benefits in reducing liver fat and improving metabolic health.

Public Health Implications
The growing burden of NAFLD reflects broader changes in global food systems. Policy interventions aimed at reducing UPF availability, improving food labeling, regulating marketing practices, and promoting nutrition education are essential. Healthcare systems should integrate dietary counseling and therapeutic nutrition interventions into routine clinical management to curb NAFLD progression.

Table 1: Impact of Ultra-Processed Foods on Non-Alcoholic Fatty Liver Disease (NAFLD) — Evidence from Published Studies

Author (Year)	Study Design & Population	Sample Size	Exposure Assessment	Outcome Measured	Key Findings
Monteiro *et al*. (2019) [18]	Conceptual epidemiological review	—	NOVA classification of UPFs	Metabolic disorders	High UPF intake linked with obesity, insulin resistance, and fatty liver risk
Zelber-Sagi *et al*. (2021)	Observational study (Adults)	~789 participants	Western dietary pattern including processed foods	NAFLD prevalence	Processed food consumption significantly associated with hepatic steatosis
Younossi *et al*. (2018)	Global epidemiology review	Global population	Dietary transition analysis	NAFLD burden	Westernized diets rich in processed foods contribute to rising NAFLD prevalence
Nardelli *et al*. (2019)	Cross-sectional study	~1,000 adults	Fast food & processed food intake	Liver fat accumulation	Frequent processed food intake associated with increased fatty liver risk
Lane *et al*. (2024)	Umbrella review (BMJ)	Multiple meta-analyses	Ultra-processed food exposure	Cardiometabolic outcomes	Strong association between UPFs, metabolic syndrome, and liver disease risk
Chen *et al*. (2023)	Prospective cohort study	>12,000 adults	Percentage of energy from UPFs	NAFLD incidence	Higher UPF consumption increased likelihood of fatty liver development
Zhang *et al*. (2022)	Cross-sectional population study	~5,000 participants	Dietary pattern analysis	Elevated ALT & AST	UPF intake correlated with liver enzyme elevation
Tilg & Moschen (2010)	Mechanistic review	—	Diet-induced metabolic disturbance	NAFLD pathogenesis	Insulin resistance, inflammation, and gut-liver axis implicated in NAFLD
Heindel *et al*. (2017)	Environmental exposure review	—	Food additives & endocrine disruptors	Liver metabolic dysfunction	Food additives and packaging chemicals may contribute to fatty liver development
Vitale *et al*. (2023)	Narrative review	Multiple populations	Ultra-processed dietary patterns	Metabolic syndrome & NAFLD	UPFs promote obesity, dyslipidemia, and hepatic steatosis progression

Conclusion
Ultra-processed food consumption represents an emerging dietary risk factor contributing significantly to the development and progression of non-alcoholic fatty liver disease. Through mechanisms involving excessive caloric intake, fructose-induced lipogenesis, insulin resistance, inflammation, oxidative stress, and gut microbiome disruption, UPFs promote hepatic fat accumulation and metabolic dysfunction. Reducing reliance on ultra-processed foods and encouraging whole-food dietary patterns may play a crucial role in preventing NAFLD and improving global liver health outcomes.

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