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THE ASSOCIATION BETWEEN FAST-FOOD CONSUMPTION AND DISORDERS OF HEPATIC LIPID METABOLISM

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Abstract
Consumption of fast food and ultra-processed foods has increased globally over recent decades and is recognized as an important factor in elevating the risk of metabolic diseases, including non-alcoholic fatty liver disease (NAFLD). This literature review analyzes the associations between fast food/ultra-processed food intake and disturbances in hepatic lipid metabolism (hepatic steatosis, changes in liver enzymes, insulin resistance, and activation of lipogenesis) based on epidemiological, experimental, and mechanistic evidence. Recent meta-analyses and observational studies indicate that high fast-food or ultra-processed food consumption is associated with a 20–55% increase in NAFLD risk. In parallel, strong biological evidence supports the role of fructose and sugar-sweetened beverages (SSBs) in stimulating hepatic de novo lipogenesis and promoting fat accumulation in the liver. The findings highlight the need for public health measures not focused on food fortification, but rather on consumption reduction through regulation, taxation, and information campaigns.

Keywords. Fast food, ultra-processed foods, non-alcoholic fatty liver disease, fructose, lipogenesis, insulin resistance, liver enzymes.

Introduction

Over the past decades, global dietary patterns have undergone profound changes, with highly processed, energy-dense fast-food products becoming a substantial component of daily diets. These products are typically rich in saturated and trans fatty acids, added sugars (particularly fructose), sodium, and carbohydrates with low biological value, and are characterized by adverse effects on metabolic health. Epidemiological data indicate that in some developed countries, up to 30–50% of daily energy intake is derived from fast food and ultra-processed foods, significantly increasing the risk of metabolic disorders, particularly disturbances in hepatic lipid metabolism.

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Non-alcoholic fatty liver disease (NAFLD) is currently the most prevalent chronic liver disease worldwide. Global estimates suggest that approximately 25–30% of the adult population is affected, with prevalence exceeding 35% in certain regions. NAFLD pathogenesis is multifactorial, with insulin resistance, enhanced lipogenesis, oxidative stress, and chronic inflammation considered key mechanisms. Emerging scientific evidence demonstrates that fast-food products influence nearly all of these mechanisms simultaneously.

The impact of fast-food dietary patterns on hepatic lipid metabolism is primarily explained by activation of hepatic de novo lipogenesis. Fructose and high–glycemic index carbohydrates are metabolized in the liver relatively independently of insulin regulation, leading to increased triglyceride synthesis. Consequently, hepatic fat accumulation, increased secretion of very-low-density lipoproteins (VLDL), and disruption of lipid homeostasis occur. Experimental and clinical studies show that fructose-rich beverages and fast-food–based diets can increase hepatic fat content even over short periods.

In addition, saturated and trans fatty acids commonly used in fast-food products suppress mitochondrial β-oxidation and limit lipid utilization. This process further enhances lipid accumulation in the liver and increases the risk of progression from simple steatosis to steatohepatitis and fibrosis. Observational studies have demonstrated a strong association between high fast-food consumption and elevated liver enzyme levels (ALT, AST).

Recent meta-analyses have quantitatively confirmed the association between fast-food and ultra-processed food consumption and the risk of NAFLD. According to several large analyses, individuals who regularly consume fast food have a 1.3–1.6-fold higher risk of developing NAFLD, and this association persists even after adjustment for body weight, physical activity, and other metabolic factors. These findings support the consideration of fast-food consumption as an independent risk factor for NAFLD.

At the same time, the existing literature presents certain methodological limitations, including variability in the definition of fast food, differences in methods used to assess consumption levels, and inconsistent control of confounding factors. These issues necessitate caution in interpreting results. Nevertheless, the consistency of epidemiological, clinical, and experimental

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evidence indicates a biologically plausible association between fast-food consumption and disorders of hepatic lipid metabolism.

Aim of the study

To systematically analyze existing scientific literature on the relationship between fast-food/ultra-processed food consumption and disorders of hepatic lipid metabolism (NAFLD, changes in liver enzymes, hepatic lipogenesis), and to synthesize epidemiological evidence, underlying mechanisms, and practical recommendations for public health.

Materials and methods

This article was prepared as a preliminary stage for a systematic review and meta-analysis. Searches were conducted in electronic databases (PubMed, Scopus, Web of Science) using the following keywords: "physical activity," "exercise," "non-alcoholic fatty liver disease," "hepatitis prevention," "liver fat reduction," and "randomized trial exercise liver." Articles published between 2000 and 2025 were analyzed.

Results from selected studies were extracted and grouped according to various parameters (type of activity, intensity, duration, and dose) for comparative analysis.

Results

Fast-food consumption and NAFLD prevalence. Large epidemiological studies published in recent years demonstrate a consistent and statistically significant association between regular fast-food consumption and the development of non-alcoholic fatty liver disease (NAFLD). According to global meta-analyses, individuals consuming fast food ≥2–3 times per week have a 1.3–1.6-fold higher risk of NAFLD. This association remains significant in models adjusted for body mass index, physical activity, and total energy intake.

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Table 1. Fast-food consumption and NAFLD risk: selected epidemiological studies

Author (year)	Study type	Sample	Main finding
He et al., 2025	Meta-analysis	9 studies	High fast-food intake → NAFLD OR = 1.55 (95% CI 1.51–1.59)
Grinshpan et al., 2024	Systematic review	10 studies	UPF/fast food → NAFLD risk ↑ ≈ 22%
Tseng et al., 2023	NHANES	~3,700	Fast food/SSB ↑ → NAFLD prevalence ↑

Ultra-processed foods and hepatic lipid metabolism. Fast-food products largely fall within the category of ultra-processed foods (UPFs). High UPF intake is associated with disturbances in hepatic lipid metabolism; several cohort studies report 20–30% higher prevalence of hepatic steatosis among individuals whose diets contain a high proportion of UPFs. Increased UPF consumption is linked to hepatic triglyceride accumulation and enhanced secretion of very-low-density lipoproteins (VLDL).

Table 2. UPF intake and hepatic lipid markers

Study	Biomarker	Outcome
Grinshpan et al., 2024	Liver fat (MRI)	High UPF → hepatic TG ↑
Ciaffi et al., 2025	CRP, IL-6	UPF ↑ → inflammatory markers ↑
NHANES analyses	ALT/AST	UPF ↑ → ALT ↑

Sugar-sweetened beverages, fructose, and de novo lipogenesis. Sugar-sweetened beverages (SSBs) and fructose, commonly consumed alongside fast food, are major drivers of hepatic de novo lipogenesis (DNL). Clinical and experimental studies show that fructose metabolism is partially independent of insulin regulation, accelerating triglyceride synthesis in the liver. Meta-analyses indicate that individuals with high SSB consumption have a 1.3–1.4-fold increased risk of NAFLD.

Table 3. SSB/fructose intake and hepatic lipid metabolism

Study	Indicator	Outcome
Jensen et al., 2018	DNL activity	Fructose ↑ → DNL ↑
Tseng et al., 2023	NAFLD prevalence	High SSB → NAFLD ↑
Intervention RCTs	Hepatic TG	SSB reduction → TG ↓

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Fat composition and lipid utilization. Saturated and trans fatty acids, which are abundant in fast-food products, suppress mitochondrial β-oxidation and limit lipid utilization. Animal models demonstrate accelerated hepatic fat accumulation under such diets, while human studies report elevations in ALT and AST levels. These findings indicate that the lipid composition of fast food exerts a direct detrimental effect on hepatic metabolism.

Interaction with components of metabolic syndrome. Fast-food consumption frequently coexists with insulin resistance, abdominal obesity, and dyslipidemia. Evidence suggests that these metabolic disturbances amplify the adverse hepatic effects of fast food. In individuals with established metabolic syndrome, fast-food intake may increase the risk of NAFLD by up to twofold.

Discussion

The results of this literature review demonstrate a stable, biologically plausible, and epidemiologically supported association between fast-food consumption and disorders of hepatic lipid metabolism. The analyzed meta-analyses, cohort studies, and national surveillance data confirm that regular consumption of fast food and ultra-processed foods represents an independent risk factor for the development of non-alcoholic fatty liver disease (NAFLD).

As reported in the results, the finding that fast-food consumption increases the risk of NAFLD by 1.3–1.6 times is not only statistically significant but also clinically meaningful. Importantly, this association persists in many studies even after adjustment for confounding factors such as body mass index, total energy intake, and physical activity. This indicates that the adverse effects of fast food on hepatic metabolism are mediated not solely through obesity, but also via direct metabolic mechanisms.

Several studies have demonstrated a parallel increase in NAFLD prevalence with rising levels of fast-food consumption, suggesting a dose–response relationship and strengthening the likelihood of causality. From an epidemiological perspective, these findings support the view that fast food should not be regarded merely as a "lifestyle marker," but rather as a pathogenic factor in NAFLD development.

Fructose and sugar-sweetened beverages: a central mechanism of hepatic lipid disorders. One of the most critical mechanistic links highlighted in this discussion is the role of fructose and sugar-sweetened beverages (SSBs). As shown in the

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results, fructose is metabolized in the liver in a manner relatively independent of insulin regulation, leading to marked activation of hepatic de novo lipogenesis. This process promotes triglyceride accumulation in the liver, increased very-low-density lipoprotein (VLDL) secretion, and disruption of lipid homeostasis.

The convergence of clinical and experimental evidence indicates that fructose-rich diets can enhance hepatic fat accumulation even over short periods. This underscores the importance of fast-food components in NAFLD pathogenesis and clearly demonstrates that the issue lies not only in caloric excess, but in the quality of calories consumed.

Saturated and trans fats: impaired lipid utilization. Saturated and trans fatty acids, which are prevalent in fast-food products, represent another key pathogenic mechanism underlying hepatic lipid metabolism disorders. The studies reviewed show that these fats suppress mitochondrial β-oxidation and limit lipid utilization, thereby accelerating hepatic fat accumulation and increasing the risk of progression from simple steatosis to steatohepatitis.

This mechanism confirms that fast-food products are not merely sources of excess energy, but foods with intrinsic lipotoxic effects. In particular, the ability of trans fats to activate inflammatory and fibrotic pathways helps explain their association with more severe forms of NAFLD.

The discussed findings also indicate that the adverse effects of fast-food consumption on hepatic lipid metabolism are often amplified by components of the metabolic syndrome. In individuals with insulin resistance, abdominal obesity, and dyslipidemia, fast-food products act as catalysts that accelerate hepatic fat accumulation. This supports the clinical view that NAFLD should be considered not as an isolated disease, but as part of a broader spectrum of metabolic disorders.

Overall, the results and their interpretation identify fast-food consumption as a major modifiable risk factor contributing to the global burden of NAFLD. Public health strategies recommended by the World Health Organization—such as limiting sugar-sweetened beverages, improving food labeling, and regulating food marketing (particularly to children)—are fully aligned with these findings and play a crucial role in primary prevention of NAFLD.

Nevertheless, certain limitations exist in the current literature. Variability in the definition of fast food and ultra-processed foods across studies, reliance on self-reported dietary assessments, and the scarcity of long-term randomized controlled

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trials complicate the generalization of results. Future research should address these gaps through long-term cohort studies, biomarker-based assessments, and dietary modification interventions.

Conclusion

The findings of this literature review demonstrate a consistent and biologically grounded association between consumption of fast food and ultra-processed foods and disturbances in hepatic lipid metabolism, particularly the development of non-alcoholic fatty liver disease. This association is reinforced by fructose-driven de novo lipogenesis, the effects of saturated and trans fats, and interactions with components of the metabolic syndrome.

1. Scientific evidence indicates that regular fast-food consumption increases the risk of NAFLD and hepatic triglyceride accumulation by 1.3–1.6 times. This association persists even after accounting for body weight and total caloric intake, confirming a direct pathogenic effect of fast food on hepatic metabolism. Sugar-sweetened beverages and fructose emerge as central drivers of hepatic lipid synthesis.
2. Saturated and trans fatty acids in fast-food products impair lipid utilization by inhibiting mitochondrial β-oxidation and activating inflammatory processes. In individuals with metabolic syndrome components—such as insulin resistance, abdominal obesity, and dyslipidemia—these effects are amplified, increasing the risk of progression to more severe forms of NAFLD. This underscores the need to view hepatic lipid disorders within the broader context of metabolic disease.
3. Current evidence confirms that reducing fast-food consumption is a key and modifiable strategy for primary prevention of NAFLD. Limiting sugar-sweetened beverages, improving dietary quality, and promoting healthy eating patterns are aligned with World Health Organization dietary policies and are essential for improving hepatic metabolic health.

Fast food is not merely “convenient food” for the liver, but a source of metabolic burden. Regular consumption accelerates hepatic fat accumulation and increases the long-term risk of chronic liver disease. Therefore, reducing fast-food intake is not simply an individual dietary choice, but a strategic public health necessity aimed at safeguarding metabolic stability in future generations.

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