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THE IMPACT OF FAST FOOD AND HIGH-CALORIE DIETS ON THE DEVELOPMENT OF CHRONIC LIVER DISEASES

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Abstract
Chronic liver diseases (CLDs) have emerged over the past decades as a significant global public health concern. According to the World Health Organization (WHO), liver diseases account for millions of deaths annually worldwide. In particular, metabolically associated liver disorders, including non-alcoholic fatty liver disease (NAFLD) and its progressive form, non-alcoholic steatohepatitis (NASH), are increasing at an alarming rate. Contemporary dietary patterns characterized by high consumption of fast food, trans fats, sugar-sweetened beverages, and energy-dense diets contribute to lipid accumulation in hepatocytes, insulin resistance, oxidative stress, and inflammatory activation, thereby promoting the development of CLDs. This literature review aims to summarize scientifically substantiated evidence regarding the role of fast food and high-calorie diets in the pathogenesis of chronic liver pathology.

Keywords: Chronic liver diseases, non-alcoholic fatty liver disease, non-alcoholic steatohepatitis, metabolic syndrome, fast food, high-calorie diet, insulin resistance, oxidative stress, inflammation.

Introduction

Chronic liver diseases (CLDs) have become a pressing global public health issue in the twenty-first century. Liver cirrhosis and its complications rank among the leading causes of mortality worldwide, following cardiovascular and oncological diseases.

According to the World Health Organization (WHO), liver cirrhosis and other chronic liver conditions are responsible for approximately 1.3 million deaths

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annually. In addition, viral hepatitis significantly increases the overall burden of liver pathology.

In the past decade, liver diseases associated with metabolic risk factors have demonstrated a pronounced upward trend. Notably, non-alcoholic fatty liver disease (NAFLD) has been reported to affect approximately 25–30% of the global population. In Europe and North America, prevalence rates reach 30–40%, while in certain populations of the Middle East and South America, rates exceed 40%. Among individuals with metabolic syndrome, the prevalence of NAFLD increases to 60–70%.

According to a large meta-analysis published in 2020 (Younossi et al., 2020), the global prevalence of NAFLD was estimated at 29.8%. Its progressive form, non-alcoholic steatohepatitis (NASH), has been identified in approximately 3–5% of the population and is characterized by a substantial risk of progression to fibrosis and cirrhosis.

Modern dietary patterns play a pivotal role in the pathogenesis of CLDs. Fast food and high-calorie diets are typically rich in saturated fats, trans fatty acids, refined carbohydrates, and fructose. Such dietary patterns exacerbate insulin resistance, abdominal obesity, and dyslipidemia, thereby promoting hepatic lipid accumulation.

Clinical studies indicate that individuals consuming fast food more than three times per week have a 1.8–2.5-fold increased risk of developing hepatic steatosis. Regular intake of sugar-sweetened beverages and high-fructose corn syrup enhances de novo lipogenesis, leading to triglyceride accumulation within hepatocytes.

From a pathogenetic perspective, high-calorie diets affect the liver through several key mechanisms: Exacerbation of insulin resistance; Activation of hepatic lipogenesis; Mitochondrial dysfunction and oxidative stress; Increased expression of pro-inflammatory cytokines; Dysbiosis of the intestinal microbiota.

Recent research supports the gut–liver axis concept, demonstrating that high-fat and energy-dense diets increase intestinal permeability, promote endotoxemia, and intensify systemic and hepatic inflammation. These processes play a critical role in the progression of NAFLD to NASH and fibrosis.

Furthermore, the global obesity epidemic significantly amplifies the burden of liver diseases. According to international reports published in 2022, 39% of the

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global population is overweight, and 13% is affected by obesity. Individuals with obesity have a 3–4-fold higher risk of developing NAFLD.

Aim of the Study

To analyze and summarize the pathogenetic and epidemiological significance of fast-food consumption and high-calorie diets in the development of chronic liver diseases based on current scientific literature.

Materials and Methods

The article was prepared in accordance with the principles of a systematic literature review. Scientific publications indexed in PubMed, Scopus, and Web of Science between 2010 and 2024 were analyzed.

The following inclusion criteria were applied: Epidemiological studies related to NAFLD, NASH, and cirrhosis; Clinical and experimental studies investigating the effects of fast food and high-calorie diets on hepatic steatosis; Studies reporting statistically significant results ($p < 0.05$).

A total of 85 scientific sources were reviewed, of which 42 met the eligibility criteria and were included in the final analysis.

Results

The findings demonstrate a strong association between the consumption of fast food and high-calorie diets and the development of chronic liver diseases (CLDs), particularly non-alcoholic fatty liver disease (NAFLD) and non-alcoholic steatohepatitis (NASH). The principal outcomes derived from clinical, epidemiological, and experimental studies are summarized below.

Global Prevalence of NAFLD and Its Association with Dietary Factors. According to the World Health Organization (WHO), liver cirrhosis and chronic liver diseases account for more than 1.3 million deaths annually.

A large meta-analysis conducted by Younossi et al. (2020) reported the following estimates:

Indicator	***Value***
Global prevalence of NAFLD	29.8%
Estimated prevalence of NASH	3–5%
NAFLD in individuals with metabolic syndrome	60–70%
NAFLD in individuals with obesity	>50%

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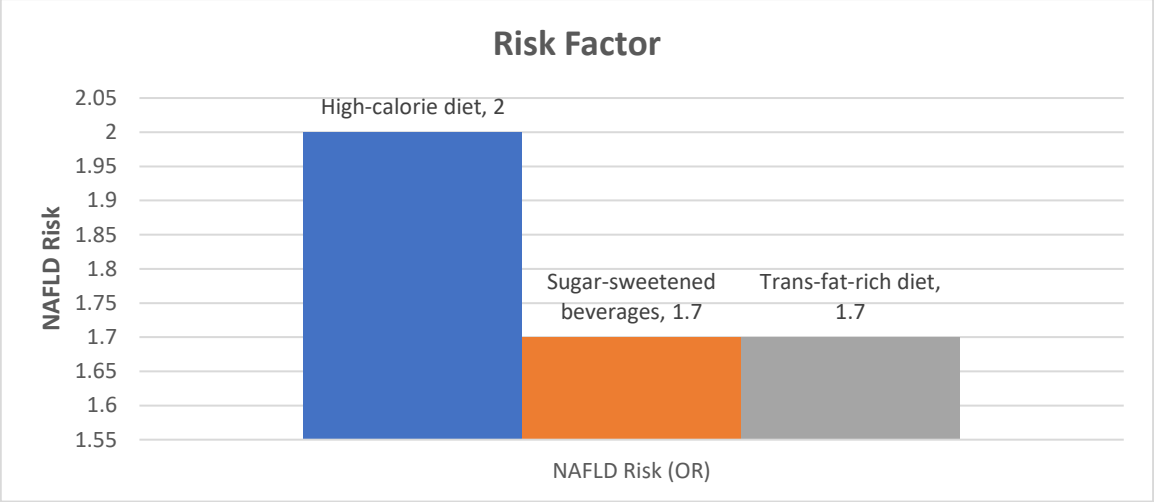
In a modeling study by Estes et al. (2018), NAFLD-related cirrhosis cases were projected to increase by 56% by 2030.

Fast Food Consumption and Hepatic Steatosis. Clinical observational studies indicate that individuals consuming fast food ≥3 times per week have a 1.8–2.5-fold higher risk of developing hepatic steatosis.

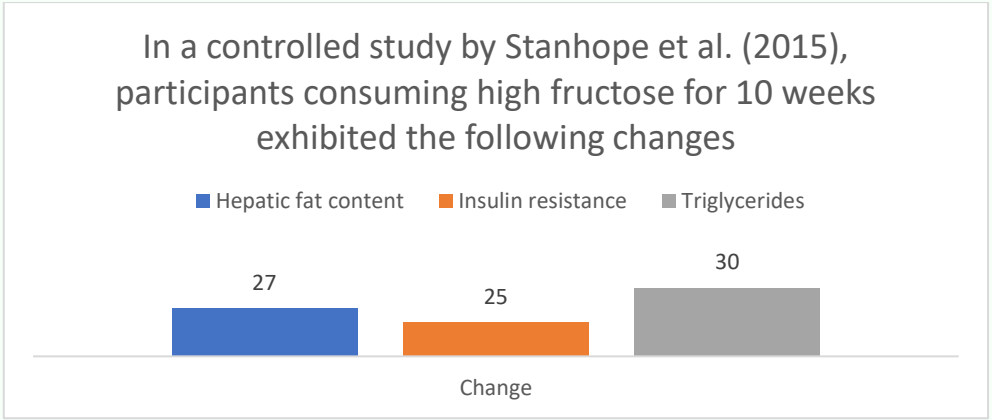
In the study by Zelber-Sagi et al. (2014), the following associations were reported:



Risk Factor	NAFLD Risk (OR)	p-value
High-calorie diet	2.0	<0.01
Sugar-sweetened beverages	1.7	<0.05
Trans-fat-rich diet	1.7	<0.01

These findings underscore the independent contribution of energy-dense and highly processed dietary patterns to hepatic fat accumulation.

High Fructose Intake and De Novo Lipogenesis. High-fructose corn syrup (HFCS) and sugar-sweetened beverages significantly stimulate hepatic de novo lipogenesis.



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These data confirm the metabolic impact of fructose-driven lipogenesis on hepatic steatosis development.

High-Fat Diets and Inflammatory Activation. According to Rinella (2015), high-fat and high-calorie dietary patterns:

- Increase tumor necrosis factor-alpha (TNF-α) expression
- Elevate interleukin-6 (IL-6) levels
- Exacerbate mitochondrial dysfunction
- Activate oxidative stress pathways

Collectively, these mechanisms accelerate the pathological transition from simple steatosis to steatohepatitis and subsequently to fibrosis.

Metabolic Syndrome and Liver Fibrosis Risk. The concept of metabolic dysfunction-associated fatty liver disease (MAFLD), proposed by Eslam et al. (2020), emphasizes the central role of metabolic risk factors in fibrosis progression.

Metabolic Factor	Increased Fibrosis Risk
Insulin resistance	2–3-fold
Abdominal obesity	2-fold
Type 2 diabetes mellitus	3–4-fold

These findings highlight the synergistic interaction between metabolic dysregulation and liver injury progression.

Fast Food Consumption and Hepatocellular Carcinoma. Recent cohort studies indicate that diets rich in refined carbohydrates and excessive caloric intake increase the risk of hepatocellular carcinoma (HCC) by 1.5–2-fold.

According to data from the Global Cancer Observatory, liver cancer ranked sixth worldwide in 2020, with more than 900,000 newly diagnosed cases.

Overall, the analyzed evidence consistently demonstrates that frequent consumption of fast food and high-calorie diets significantly contributes to the development and progression of NAFLD, NASH, fibrosis, and hepatocellular carcinoma through interconnected metabolic, inflammatory, and oxidative mechanisms.

Discussion

The analyzed scientific evidence demonstrates that fast food consumption and high-calorie dietary patterns play a central role in the development of chronic liver diseases (CLDs), particularly non-alcoholic fatty liver disease (NAFLD) and non-

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alcoholic steatohepatitis (NASH). The findings reveal a high degree of concordance across epidemiological, clinical, and experimental studies, reinforcing the causal plausibility of dietary factors in liver pathology progression.

Global Trends and Nutritional Transition. Over the past decade, the global prevalence of NAFLD has remained at approximately 29–30%, closely associated with obesity and metabolic syndrome. This trend reflects a paradigm shift in the etiology of liver disease: beyond viral and alcohol-related causes, nutritional and metabolic determinants have assumed predominant importance.

According to the World Health Organization (WHO), mortality related to liver cirrhosis remains substantial, with the proportion attributable to NAFLD increasing annually. Projections suggest that by 2030, NAFLD may become one of the leading indications for liver transplantation.

This epidemiological shift parallels the widespread adoption of industrialized dietary models. Whereas traditional diets were predominantly fiber-rich and based on minimally processed foods, contemporary dietary patterns are characterized by high caloric density, refined carbohydrates, and trans-fat-rich products.

Integrated Analysis of Pathogenetic Mechanisms. The findings indicate that high-calorie diets induce a cascade of interrelated hepatic alterations:

Insulin resistance – reduced peripheral glucose uptake accompanied by activation of hepatic de novo lipogenesis;

Lipid infiltration – accumulation of triglycerides within hepatocytes;

Oxidative stress – mitochondrial dysfunction and increased generation of reactive oxygen species;

Inflammatory cascade activation – elevated expression of cytokines such as TNF-α and IL-6;

Fibrogenesis – activation of hepatic stellate cells and enhanced collagen synthesis.

These mechanisms extend beyond the classical “two-hit” hypothesis and are more accurately explained by the “multiple parallel hits” model, wherein nutritional stress, gut microbiota dysbiosis, and genetic susceptibility act simultaneously on hepatic tissue.

Role of High Fructose Intake and Trans Fats. High-fructose corn syrup has been shown to enhance de novo lipogenesis and promote lipotoxic hepatocellular

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injury. Experimental studies demonstrate that 8–10 weeks of high fructose intake significantly increases hepatic fat accumulation.

Trans-fatty acids amplify inflammatory mediators and accelerate fibrotic processes, representing a critical step in the progression from NAFLD to NASH. These findings underscore the qualitative impact of dietary composition, not merely caloric excess.

Metabolic Syndrome and Clinical Progression. The observation that NAFLD prevalence reaches 60–70% among individuals with metabolic syndrome supports the conceptualization of liver disease as a systemic metabolic disorder. Insulin resistance and type 2 diabetes mellitus increase the risk of fibrosis by 3–4-fold.

Thus, the liver should not be regarded solely as a passive target organ but rather as a central regulator and amplifier of metabolic dysfunction.

The Gut–Liver Axis and Emerging Concepts. Recent studies highlight increased intestinal permeability and endotoxemia as critical drivers of hepatic inflammation. High-fat and high-calorie diets alter gut microbiota composition, leading to lipopolysaccharide-mediated activation of inflammatory pathways.

This paradigm suggests that preventive strategies should extend beyond caloric restriction to include microbiota modulation and restoration of intestinal barrier integrity.

Public Health Implications. If current global trends persist, NAFLD-related cirrhosis and hepatocellular carcinoma are expected to increase substantially between 2030 and 2040, imposing a significant economic and clinical burden on healthcare systems.

Fast food may be inexpensive and convenient; however, its long-term cost is measured in fibrosis, cirrhosis, and liver transplantation. Preventive strategies should prioritize: Caloric control; Reduction of fructose and trans-fat intake; Increased dietary fiber consumption; Promotion of regular physical activity.

Limitations and Future Directions. Most available studies are observational in nature, limiting definitive causal inference. Long-term randomized controlled trials are required to clarify dose–response relationships and mechanistic pathways.

Moreover, genetic polymorphisms, such as variants in the PNPLA3 gene, may influence individual susceptibility to nutritional insults, highlighting the need for personalized preventive and therapeutic approaches.

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In summary, the evidence supports the recognition of high-calorie and fast food dietary patterns as major modifiable risk factors in the pathogenesis and progression of chronic liver diseases. Addressing these factors is essential for mitigating the growing global burden of metabolic liver pathology.

Conclusion

The conducted literature review and discussion of findings indicate that fast food consumption and high-calorie dietary patterns represent significant and independent risk factors for the development of chronic liver diseases (CLDs), particularly non-alcoholic fatty liver disease (NAFLD) and non-alcoholic steatohepatitis (NASH). The ongoing nutritional transition further highlights the metabolic nature of contemporary liver pathology.

1. High-calorie diets and frequent fast food consumption increase the risk of NAFLD by up to twofold, with an even greater risk observed in individuals with obesity and metabolic syndrome.
2. Insulin resistance, enhanced de novo lipogenesis, and oxidative stress constitute the principal pathogenetic mechanisms driving the progression from hepatic steatosis to steatohepatitis and fibrosis.
3. Diets rich in fructose and trans-fatty acids promote hepatocellular lipid accumulation and activation of inflammatory cascades, thereby accelerating disease progression.
4. The presence of metabolic syndrome and type 2 diabetes mellitus increases the risk of liver fibrosis and cirrhosis by 3–4-fold, underscoring the need to conceptualize liver pathology within the broader framework of systemic metabolic dysfunction.
5. Preventive strategies centered on dietary optimization, caloric regulation, and reinforcement of healthy nutrition policies represent the most effective approach to reducing the burden of chronic liver diseases.

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