**Review Article**

**The Interplay Between Iron Metabolism and metabolic dysfunction-associated steatotic liver disease (MASLD): A review on Implications of Iron Supplementation in Disease Progression and Management**

**Abstract**

Iron is a vital micronutrient involved in oxygen transport, mitochondrial respiration, and cellular metabolism, with the liver serving as the primary regulator of iron homeostasis through hepcidin-mediated control of absorption and storage. However, excessive iron intake—whether from diet or supplements—can lead to hepatic iron overload, triggering oxidative stress, lipid peroxidation, and chronic inflammation. Dysregulated iron metabolism, particularly in the presence of high-fat diets, has been strongly associated with the progression of metabolic dysfunction-associated steatotic liver disease (MASLD) to its more severe form, metabolic dysfunction-associated steatohepatitis (MASH). Animal studies have demonstrated that iron accumulation in Kupffer cells and macrophages amplifies hepatic inflammation, exacerbates metabolic dysfunction, and accelerates fibrosis, increasing the risk of cirrhosis and hepatocellular carcinoma. Epidemiological evidence further supports a link between high dietary iron intake and liver damage, particularly in populations with genetic predispositions to altered iron metabolism. Despite these findings, the impact of excessive iron intake on liver health remains underrecognized. This review explores the mechanisms by which excess supplemental iron contribute to liver injury, highlighting the need for further research to establish safe iron intake levels and potential therapeutic strategies for mitigating iron-induced liver damage.

Key words: Iron metabolism, hepcidin regulation, oxidative stress, fatty liver disease.

**Introduction**

Iron is essential for mammalian metabolism, playing a key role in mitochondrial respiration, oxygen transport, the citric acid cycle, and DNA biosynthesis. It supports erythropoiesis and various cellular functions, but excess iron can trigger oxidative stress through the Fenton reaction. To prevent damage, cells sequester iron within ferritin under normal conditions.[1] Hepcidin regulates iron release from enterocytes and macrophages to maintain homeostasis. Iron overload saturates transferrin, increasing toxic non-transferrin bound iron (NTBI), which accumulates in the liver and heart, causing oxidative stress, lipid peroxidation, and organ dysfunction.[2] Metabolic dysfunction-associated steatotic liver disease (MASLD) ranges from simple steatosis to metabolic dysfunction-associated steatohepatitis (MASH), potentially progressing to cirrhosis. Its prevalence is increasing with metabolic disorders, particularly in Western countries. While the role of iron overload in MASLD remains debated, dysmetabolic iron overload syndrome (DIOS) affects about one-third of individuals with MASLD and metabolic syndrome.[3]

Iron overload commonly results from hereditary hemochromatosis, iron-loading anemias (such as thalassemia), and parenteral iron accumulation due to repeated blood transfusions. The liver plays a central role in iron metabolism, regulating its storage, utilization, and systemic homeostasis. Given the increasing prevalence of fatty liver disease across all age groups, it is essential to investigate the interplay between iron metabolism and liver pathology.[4]

Iron metabolism dysregulation plays a crucial role in the development of non-obese or lean MASLD. Disruptions in iron homeostasis, such as increased serum ferritin levels, elevated transferrin saturation, and excessive hepatic iron accumulation, contribute to oxidative stress, mitochondrial dysfunction, and hepatocellular injury. Studies have shown that lean MASLD patients often exhibit abnormal iron metabolism markers, even in the absence of traditional metabolic risk factors. This suggests that iron dysmetabolism may independently drive liver inflammation, fibrosis, and lipid accumulation, promoting the progression of MASLD in individuals without obesity.[5]

Excessive iron intake, whether through supplements, has been implicated in liver injury by promoting hepatic iron accumulation, oxidative stress, and inflammation, which may accelerate the progression of MASLD to MASH. Experimental and clinical studies suggest that iron overload, particularly in the presence of a high-fat diet, exacerbates liver damage, highlighting the need to understand its role in metabolic dysfunction and chronic liver disease.

This review explores the liver’s function in iron metabolism, the impact of liver disease progression on iron overload, and the associated consequences.

**Iron Metabolism and Its Regulation in the Human Body**

Iron is an essential nutrient that the body cannot produce on its own and must be obtained through diet or supplements. According to the US Institute of Medicine, the recommended daily intake of iron is 8 mg for men and postmenopausal women, while premenopausal women require 18 mg per day.[6] Dietary iron is primarily absorbed in the duodenum and upper jejunum, but this intake alone is often insufficient to meet the body’s needs. To maintain adequate iron levels, the body relies on the recycling of iron from aging or damaged red blood cells, a process that takes place in the spleen, liver, and bone marrow.

The liver plays a crucial role in iron metabolism by producing hepcidin, a hormone that regulates iron absorption and distribution. Hepcidin controls iron levels by inducing the degradation of ferroportin, a protein responsible for exporting iron from cells into the bloodstream. By limiting iron release from dietary absorption and stored reserves, hepcidin helps prevent excessive iron accumulation, which can be toxic. Since the liver serves as a major iron storage site, conditions such as MASLD may be particularly vulnerable to iron imbalances.[7]

Iron is transported throughout the body primarily by transferrin, a protein that binds iron in a soluble and non-toxic form. Each transferrin molecule can carry up to two iron atoms, ensuring safe transport and delivery to cells. The majority of serum iron (SI) exists in transferrin-bound form, while a small amount remains as non-transferrin-bound iron (NTBI), which is highly reactive and contributes to oxidative stress and tissue damage.[8]

Ferritin stores iron within cells and circulates in small amounts as serum ferritin (SF), while transferrin saturation (TSAT) reflects iron availability. Soluble transferrin receptor (sTfR) rises with iron deficiency, and together with SF, TSAT, and serum iron (SI), these biomarkers help assess iron metabolism and diagnose iron-related disorders.[9,10]

**Role of the Liver in Iron Metabolism**

The liver regulates iron metabolism through transferrin and hepcidin, controlling absorption, storage, and distribution. Dietary iron is absorbed in the duodenum, transported into enterocytes, and either stored in ferritin or exported by ferroportin. In circulation, ferroxidases convert iron to its ferric form for transferrin binding, with most iron used for hemoglobin synthesis and the rest stored in hepatocytes for systemic balance.[11]

Hepcidin, mainly produced by the liver, regulates iron homeostasis by binding to ferroportin, reducing iron absorption and recycling. Its expression is controlled by the bone morphogenetic protein (BMP) signaling pathway, where BMP6 and hepatic hemojuvelin (HJV) promote hepcidin synthesis, while matriptase-2 (MT2) inhibits it by degrading HJV.[12]

Iron homeostasis is influenced by hypoxia, erythropoiesis, and inflammation, with hypoxia and increased erythropoiesis suppressing hepcidin to enhance iron release, while interleukin-6 (IL-6) stimulates hepcidin, reducing iron availability. Liver dysfunction, as seen in MASLD and hereditary hemochromatosis, can lead to iron accumulation, oxidative stress, and fibrosis, highlighting the need for targeted therapies for iron-related liver diseases.[13]

**Regulation of Hepcidin in Liver Injury**

Hepcidin regulation during liver injury varies with inflammation and oxidative stress. Acute liver injury increases hepcidin via IL-6/STAT3 signaling, lowering blood iron levels, while chronic injury often suppresses hepcidin due to CHOP-mediated inhibition of C/EBPα. However, oxidative stress may also elevate hepcidin through BMP6 activation, making its role in iron balance complex.[14] (Fig.1)

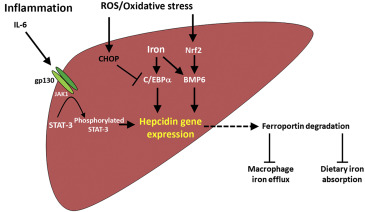


Fig.1 Hepcidin regulation in liver injury

**Serum Ferritin Levels and MASLD**

High serum ferritin levels (hyperferritinemia) are linked to liver damage in MASLD. Studies by Bugianesi, Kowdley, and Sumida indicate that while ferritin does not directly reflect liver iron content, elevated levels correlate with severe liver injury, advanced fibrosis, and steatohepatitis risk.[17]

However, some studies have not confirmed a direct link between ferritin levels and liver fibrosis. Valenti et al. found no strong correlation between serum ferritin and liver fibrosis in MASLD, suggesting ferritin levels may be driven by inflammation rather than iron overload, particularly when iron is stored in macrophages rather than liver cells.[18] Similarly, Chitturi et al., in a study of 93 patients with MASH (33% of whom had advanced fibrosis), found that serum ferritin was not an independent predictor of severe liver disease.[19]

A large prospective study in South Korea followed 2,410 healthy men aged 30 to 59 for over 7,500 person-years. Among them, 24.3% developed fatty liver disease, and their baseline ferritin levels were found to be a strong predictor of the condition. This suggests that elevated ferritin levels may contribute to the development of MASLD rather than simply being a consequence of liver disease. [20]

Despite large studies, serum ferritin is an imprecise marker of total body iron stores. Its link to MASLD and type 2 diabetes likely reflects metabolic disturbances like inflammation or insulin resistance rather than direct iron overload.[4]

**Hepatic Iron in MASLD: Metabolism and Consequences of Iron Overload**

Iron contributes to liver damage in MASLD by promoting oxidative stress, a key driver of MASH. This depletes vital molecules like ATP, NAD, and glutathione, damages cellular components, and triggers inflammation and fibrosis, worsening liver disease.[21]

Iron-induced oxidative stress disrupts fat metabolism by degrading apolipoprotein B100 (ApoB100), reducing VLDL secretion, and promoting liver fat accumulation. Studies show that iron chelation, dietary restriction, or phlebotomy can lower oxidative stress and restore ApoB100, improving VLDL export.[22]

Liver iron overload is linked to lipid peroxidation and oxidative DNA damage, as seen in elevated 8-oxodG and thioredoxin levels, which decrease after venesection. Experimental studies also show that excess iron impairs insulin signaling, contributing to metabolic dysfunction.[4] Iron promotes liver inflammation by activating macrophages and hepatic stellate cells, triggering NF-κB signaling and fibrosis. In MASLD, iron accumulation in immune cells is linked to inflammation, while animal studies show dietary iron overload activates inflammasomes, leading to liver damage.[23] Iron may further contribute to liver damage through endoplasmic reticulum (ER) stress. Studies in mice have shown that iron overload can trigger ER stress and an unfolded protein response, which disrupts normal cell function. Additionally, iron accumulation in the liver has been linked to increased cholesterol biosynthesis, providing another potential mechanism for liver injury in MASLD.Research on the relationship between hepatic iron concentration (HIC) and MASLD severity has yielded mixed results. Some studies have found that higher iron levels are associated with more severe fibrosis, while others have not confirmed this link. In a study of 587 Italian MASLD patients, [18] hepatocellular iron was associated with a higher risk of significant fibrosis, while iron in immune cells (reticulo-endothelial iron) appeared to have a protective trend. However, a U.S. study of 849 MASLD patients [24] found the opposite—reticulo-endothelial iron was linked to a higher risk of advanced fibrosis, lobular inflammation, and liver cell ballooning. The reasons for these discrepancies remain unclear but may involve genetic and demographic differences between study populations.

Iron overload contributes to liver injury in MASLD through oxidative stress, inflammation, and metabolic disruption, but its overall impact appears moderate. Further research is needed to assess the effectiveness of iron reduction strategies in treating MASLD-related liver damage.[4]

**Iron Metabolism in MASLD**

In NASH, hepcidin levels are high, but iron still accumulates due to disrupted metabolism. Studies show a high-fat diet increases transferrin receptor-1 (Tfr1) in liver cells, while MASH patients exhibit increased divalent metal transporter 1 (DMT1) activity, enhancing iron absorption despite elevated hepcidin.[25]

A high-fat diet increases red blood cell fragility, leading to breakdown and iron accumulation in liver immune cells, possibly explaining elevated iron in MASH patients. Hepcidin levels in MASH may rise due to inflammation, iron overload, or insulin regulation, while fat tissue also produces hepcidin, linking obesity to altered iron metabolism. The complex interplay between iron and fat metabolism suggests maintaining balanced iron levels is crucial for metabolic health.[26]

If iron contributes to MASH, dietary intake may influence iron accumulation. While no direct link exists in humans, excess iron, particularly from red meat, is associated with increased insulin resistance and type 2 diabetes risk.[4]

**The Role of Iron in MASLD Progression to MASH**

Iron overload contributes to MASLD progression to MASH and cirrhosis through the "multi-hit" hypothesis. It promotes oxidative stress, inflammation, and mitochondrial dysfunction, generating reactive oxygen species (ROS) that activate NF-κB and hepatic stellate cells, leading to fibrosis and liver damage.[3] (Fig. 2)

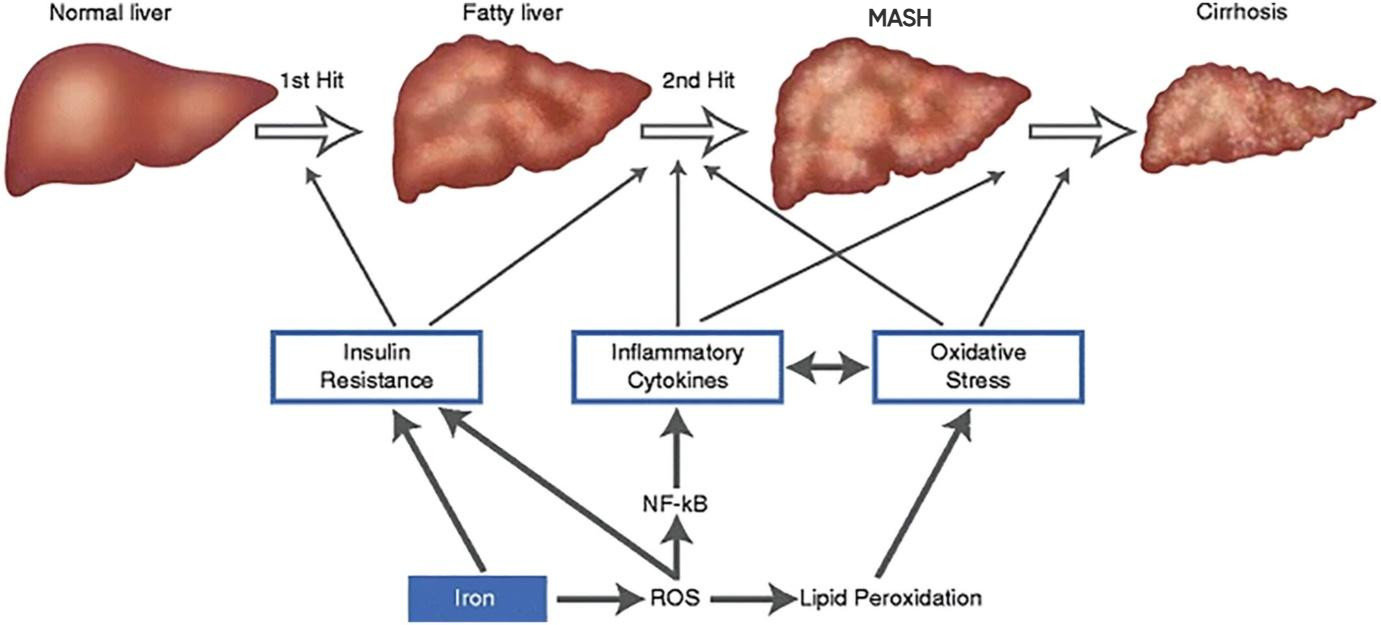


Fig. 2 Multi hit hypothesis

**Iron, Insulin Resistance, and Fatty Liver**

Iron may influence insulin resistance and fat metabolism in the liver. Insulin resistance is a key factor in the development of MASLD, and iron can interfere with insulin signalling in liver cells, reducing their sensitivity to insulin. Dysfunctional fat tissue alters the production of hormones and inflammatory molecules that promote fat buildup in the liver. Excess iron in visceral fat may worsen this dysfunction, leading to increased fat breakdown, more free fatty acids reaching the liver, and higher inflammation. In MASLD, excess fat in liver cells increases lipid peroxidation, which may contribute to liver damage, regardless of iron levels. Additionally, iron buildup has been shown to reduce the release of apolipoprotein E, a protein that helps protect against liver inflammation, leading to higher oxidative stress.[3] (Fig.3)

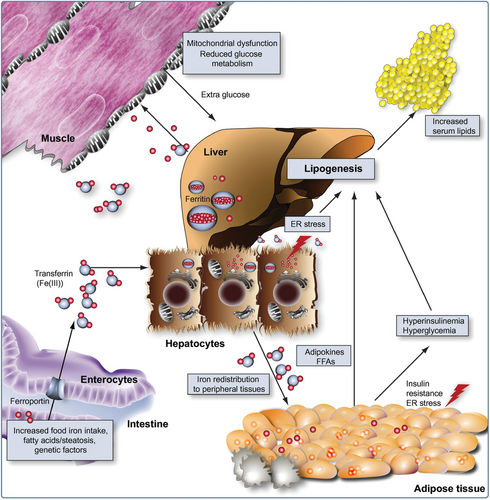


Fig. 3. Relation of iron overload, insulin resistance and fatty liver.

**Iron Overload in the Progression of MASH**

Excess intake of iron has been linked to an increased risk of MASLD, particularly in men, as demonstrated by a large cross-sectional study of 5,445 middle-aged and elderly Chinese individuals. The study found a dose-dependent relationship between iron consumption and MASLD prevalence, with individuals in the highest quintiles of iron intake showing significantly higher odds of developing the disease. Excess iron may contribute to MASLD by promoting insulin resistance, increasing hepatic oxidative stress, and triggering inflammatory pathways that lead to liver damage. Additionally, iron overload may impair copper metabolism, further exacerbating mitochondrial dysfunction and fat processing in the liver. Prior research in animal models and small human studies supports the role of high iron intake in worsening liver inflammation and fibrosis.[27]

These findings emphasize the risks of excessive or improper use of iron supplements, which may contribute to the development of MASLD.

MASH is one of the most prevalent chronic liver diseases (CLD) and has become a leading cause of cirrhosis and hepatocellular carcinoma. Iron is an essential micronutrient for living organisms; however, excessive iron accumulation in vital organs can lead to dysfunction due to the generation of reactive oxygen species. Hepatic iron overload is frequently observed in CLD patients, and in individuals with MASH, liver iron accumulation is positively associated with histological severity. This suggests that iron overload may contribute to the progression of MASLD to MASH.

In a rat model of MASH, a diet high in both fat and iron exacerbates hepatic inflammation and increases cytokine expression in the liver compared to a high-fat diet alone. In this model, iron is predominantly accumulated in Kupffer cells/macrophages within the affected areas, indicating that iron-laden Kupffer cells/macrophages may play a crucial role in intensifying hepatic inflammation under MASH conditions. Conversely, in a rat model of liver cirrhosis, dietary iron overload significantly inhibits the development and progression of cirrhosis induced by repeated administration of thioacetamide (TAA).

These findings suggest that the impact of iron overload on chronic liver diseases can vary depending on the tissue microenvironment.[28]

**Excess Iron supplements leading to MASLD and MASH**

A study conducted on rats investigated the effects of excessive iron supplementation on liver health, revealing its potential role in exacerbating liver diseases such as MASLD and MASH. In this study, rats were fed a diet supplemented with 0.5% iron, resulting in a modest increase in liver iron content. Despite consuming approximately 75 mg of iron per day—well above the upper intake limit for adult humans—hepatic iron accumulation remained lower than expected. However, previous findings from a four-week feeding study with the same iron concentration showed a two-fold increase in liver and serum iron levels, suggesting that prolonged iron intake can lead to progressive hepatic iron accumulation. Notably, iron-laden macrophages in an active inflammatory state were observed, indicating that excess iron may contribute to hepatic inflammation. These findings suggest that excessive dietary iron intake, especially in the presence of a high-fat diet, can promote liver injury and inflammation, increasing the risk of MASLD progression to MASH. [29]

While the impact of mild to moderate hepatic iron excess on MASLD remains debated, growing evidence suggests that excess iron may contribute to liver damage, increasing the risk of MASH and fibrosis progression. Iron promotes oxidative stress through reactive oxygen species (ROS) generation, which is exacerbated by lipid accumulation in liver cells.

Overuse of iron supplements can contribute to the development of MASLD and its progression to MASH by promoting hepatic iron accumulation and inflammation. Studies in rats have shown that excessive dietary iron intake leads to increased liver iron content, even at levels exceeding the upper intake limit for humans. Prolonged iron supplementation has been linked to a rise in serum and liver iron levels, suggesting a cumulative effect over time. Additionally, the presence of iron-laden macrophages in an active inflammatory state indicates that excess iron may play a key role in hepatic inflammation. These findings suggest that excessive iron intake, especially when combined with a high-fat diet, can exacerbate liver injury and increase the risk of MASLD progressing to MASH.[28,29]

Therefore, excessive iron supplementation may contribute to liver diseases such as MASLD and MASH, highlighting the need for further research to better understand its long-term impact and optimal management strategies.

**CONCLUSION**

Liver injury is closely linked to disruptions in iron metabolism, with hyperferritinemia often serving as a marker of iron overload and metabolic dysfunction. The overuse or misuse of iron supplements can lead to hepatic iron accumulation, triggering oxidative stress, inflammation, and fibrosis, which contribute to the progression of MASLD and MASH. Excess iron can contribute to insulin resistance by disrupting insulin signaling in liver cells, promoting inflammation, and exacerbating metabolic dysfunction in MASLD. These findings highlight the need for careful regulation of iron intake to prevent its harmful effects on liver health.

**List of abbreviations:**

1. **ATP** – Adenosine Triphosphate
2. **ApoB100** – Apolipoprotein B100
3. **BMP** – Bone Morphogenetic Protein
4. **BMP6** – Bone Morphogenetic Protein 6
5. **CHOP** – C/EBP Homologous Protein
6. **C/EBPα** – CCAAT Enhancer Binding Protein Alpha
7. **CRN** – Clinical Research Network
8. **DcytB** – Duodenal Cytochrome B
9. **DMT1** – Divalent Metal Transporter 1
10. **ER** – Endoplasmic Reticulum
11. **HFE** – Hemochromatosis Gene
12. **HIC** – Hepatic Iron Concentration
13. **HJV** – Hemojuvelin
14. **IL-6** – Interleukin-6
15. **MT2** – Matriptase-2
16. **MASH**– Metabolic dysfunction-associated steatotic liver disease
17. **MASLD**– metabolic dysfunction-associated steatotic liver disease
18. **NF-κB** – Nuclear Factor Kappa B
19. **NTBI** – Non-Transferrin Bound Iron
20. **ROS** – Reactive Oxygen Species
21. **SF** – Serum Ferritin
22. **SI** – Serum Iron
23. **sTfR** – Soluble Transferrin Receptor
24. **STAT3** – Signal Transducer and Activator of Transcription 3
25. **Tfr1** – Transferrin Receptor 1
26. **TMPRSS6** – Transmembrane Serine Protease 6
27. **TRX** – Thioredoxin
28. **TSAT** – Transferrin Saturation
29. **VLDL** – Very Low-Density Lipoprotein

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