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Targeting Hepcidin in Anemia: A New Frontier for Precision Medicine?

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ABSTRACT

Anemia is a common and multifactorial condition, often resulting from iron deficiency, chronic disease, or genetic disorders. One key regulator of iron homeostasis is hepcidin, a liver-produced hormone that plays a pivotal role in iron metabolism. Elevated or dysregulated hepcidin levels can lead to iron-restricted anemia, while reduced hepcidin expression can contribute to iron overload conditions. This review aims to explore the role of hepcidin in anemia and its potential as a target for precision medicine. We discuss the molecular mechanisms underlying hepcidin regulation, its interaction with iron transporters like ferroportin, and the clinical implications of hepcidin dysfunction in various types of anemia. Moreover, we examine the emerging therapeutic approaches aimed at modulating hepcidin expression or activity to treat iron-related disorders. Understanding the intricate regulation of hepcidin offers exciting prospects for precision-based therapies in anemia management, with potential applications in both iron-deficiency anemia and anemia of chronic disease.

Keywords: Hepcidin, Anemia, Iron metabolism, Precision medicine, Iron deficiency, Chronic disease anemia, Ferroportin

INTRODUCTION

Anemia is one of the most prevalent and significant global health concerns, affecting millions of people across diverse age groups, regions, and socioeconomic backgrounds[1–3]. The condition is characterized by a deficiency in the number or quality of red blood cells, leading to reduced oxygen delivery to tissues and organs, thereby impairing overall physiological function[4, 5] Anemia can arise from a wide array of causes, ranging from iron deficiency and chronic diseases to genetic disorders and inflammatory conditions. The severity and underlying etiology of anemia can vary significantly, often requiring personalized diagnostic and therapeutic strategies[6–8].

Iron metabolism plays an essential role in the process of erythropoiesis (the production of red blood cells), as iron is a crucial component of hemoglobin, the protein responsible for oxygen transport in the blood[9]. The dysregulation of iron homeostasis—whether through inadequate iron intake, improper iron storage, or defects in iron absorption or utilization—can lead to various forms of anemia[10]. Hepcidin, a 25-amino acid peptide hormone primarily produced in the liver, is a central regulator of iron metabolism. It plays a critical role in maintaining iron balance by controlling the amount of iron available for erythropoiesis and other physiological processes[11, 12]. Disruptions in hepcidin expression or activity can result in iron-related disorders, including iron deficiency anemia (IDA) and anemia of chronic disease (ACD)[13]. This review aims to delve into the role of hepcidin in the pathogenesis of anemia and explore its potential as a therapeutic target for the development of precision medicine approaches to treat anemia.

Hepcidin and Its Role in Iron Metabolism

Hepcidin is a small but potent peptide hormone that is central to the regulation of iron absorption, distribution, and storage within the body[14]. The hormone is predominantly synthesized and secreted by hepatocytes in the liver in response to various physiological signals, including changes in iron levels, inflammation, erythropoietic activity, and hypoxia[15]. Hepcidin regulates iron homeostasis by controlling the ferroportin protein, which is the only known cellular exporter of iron. Ferroportin is found on the surface of enterocytes (intestinal cells), macrophages, and hepatocytes, and its function is critical for releasing stored or absorbed iron into the bloodstream[16]. Under normal conditions, hepcidin binds to ferroportin, leading to its internalization and subsequent degradation, thereby preventing the export of iron from these cells into the circulation. As a result, circulating iron levels are reduced, and iron is retained within the cells[17]. This mechanism helps to prevent iron overload, which can be toxic to tissues, while also regulating iron availability for vital processes

such as erythropoiesis. Several factors regulate the synthesis and activity of hepcidin, making its expression a highly dynamic and tightly controlled process. These include:

Iron levels: The most important regulator of hepcidin production is the body's iron status. When iron levels are high, either due to increased dietary intake or the mobilization of stored iron, the liver responds by producing more hepcidin to reduce iron absorption from the intestine and iron release from storage sites (such as macrophages and hepatocytes[18]. Conversely, when iron levels are low, hepcidin production decreases, allowing for increased iron absorption and mobilization to meet the body's demands.

Inflammation: Inflammatory conditions, particularly those associated with chronic diseases such as infection, autoimmune disorders, and cancer, lead to an increase in pro-inflammatory cytokines such as interleukin-6 (IL-6)[19]. These cytokines stimulate hepcidin production, contributing to a state of iron sequestration in the reticuloendothelial system and reduced iron availability for erythropoiesis. This mechanism is thought to play a significant role in the development of anemia of chronic disease (ACD), a common form of anemia observed in inflammatory or chronic conditions[20].

Erythropoiesis: Erythropoiesis, or the production of red blood cells, is a process that requires substantial amounts of iron for the synthesis of hemoglobin[21]. To ensure that there is adequate iron available for red blood cell production, erythropoietic activity suppresses hepcidin expression. This downregulation of hepcidin allows for increased iron mobilization from storage sites and facilitates greater iron absorption from the gastrointestinal tract, ensuring that iron is available for hemoglobin production and new red blood cell formation[21].

Hypoxia: Low oxygen levels, or hypoxia, also influence hepcidin expression. In response to hypoxic conditions, hepcidin production is typically downregulated, allowing for increased iron availability to support enhanced hemoglobin synthesis in red blood cells. This is particularly important in tissues such as the bone marrow, where erythropoiesis is occurring at higher rates to compensate for oxygen deficiency[22]. Through these complex regulatory pathways, hepcidin maintains the delicate balance of iron homeostasis in the body, ensuring that iron is available when needed while preventing the toxic effects of iron overload. However, disruptions in hepcidin regulation can lead to various forms of anemia, including iron deficiency anemia, anemia of chronic disease, and disorders associated with iron overload, such as hemochromatosis.[22] Understanding the precise mechanisms by which hepcidin regulates iron metabolism is critical for developing targeted therapies aimed at correcting iron imbalances in individuals with anemia. This review will further explore the potential of hepcidin as a therapeutic target for precision medicine in the treatment of anemia and related disorders.

Hepcidin Dysregulation in Anemia

Hepcidin, a liver-produced peptide hormone, plays a critical role in regulating iron homeostasis by controlling iron absorption from the intestines, iron release from storage sites (e.g., the liver, macrophages), and the transport of iron into circulation[23]. Dysregulation of hepcidin expression or activity contributes significantly to various forms of anemia, each of which has unique underlying mechanisms, but all involve disruptions in iron availability for erythropoiesis (red blood cell production). Below are the major forms of anemia associated with hepcidin dysregulation[23]

Iron-Deficiency Anemia (IDA)

In Iron-Deficiency Anemia (IDA), the primary issue is insufficient iron availability for the synthesis of hemoglobin, a key component of red blood cells. The causes of IDA are varied, including inadequate dietary intake, impaired absorption (due to gastrointestinal disorders like celiac disease), or chronic blood loss (e.g., gastrointestinal bleeding, heavy menstruation)[24]. The hallmark of IDA is reduced iron stores, resulting in a shortage of available iron for erythropoiesis. Hepcidin dysregulation plays a critical role in exacerbating iron deficiency in IDA. Despite the low systemic iron levels characteristic of IDA, hepcidin levels may paradoxically be elevated in some cases. Elevated hepcidin can be driven by underlying inflammatory conditions, such as chronic disease or infection, which activate the acute-phase response. Inflammatory cytokines, particularly interleukin-6 (IL-6), stimulate hepcidin production in the liver, even when iron stores are low[25]. Elevated hepcidin reduces the intestinal absorption of dietary iron and limits the release of stored iron from macrophages and hepatocytes by inhibiting ferroportin, the iron export protein. This further exacerbates iron deficiency by preventing iron from entering circulation[25]. Additionally, mutations in genes that regulate hepcidin expression, such as those involving hemojuvelin (HJV) or matriptase-2, may cause inappropriate upregulation of hepcidin even in the absence of inflammation, contributing to the iron-restricted environment in IDA.

Anemia of Chronic Disease (ACD)

Anemia of Chronic Disease (ACD), also known as anemia of inflammation, is commonly observed in patients with chronic infections, autoimmune diseases, cancer, or other inflammatory conditions. ACD is characterized by a functional iron deficiency, where iron is present in the body but is sequestered in storage sites, rendering it unavailable for erythropoiesis[26].

In ACD, hepcidin levels are typically elevated due to the inflammatory response. Pro-inflammatory cytokines, such as IL-6, stimulate the liver to produce excessive amounts of hepcidin[26]. The upregulated hepcidin inhibits ferroportin, leading to a reduction in the mobilization of iron from macrophages and hepatocytes to the

bloodstream. This sequestration of iron in storage cells, combined with reduced intestinal absorption of iron, causes iron to become functionally unavailable for red blood cell production, even though total body iron may be normal or increased[27]. This phenomenon contributes to the characteristic low serum iron and transferrin saturation seen in ACD, despite the presence of normal or even high total body iron levels. ACD is often present in conditions such as rheumatoid arthritis, chronic kidney disease, and cancer, where ongoing inflammation and cytokine production maintain elevated hepcidin levels. The role of hepcidin in ACD highlights its importance as a mediator of anemia in the context of inflammation and suggests that targeting hepcidin or its regulatory pathways may offer therapeutic potential for treating ACD.

Hemochromatosis and Iron Overload

In contrast to IDA and ACD, hereditary hemochromatosis and other forms of iron overload are characterized by reduced hepcidin levels[28], leading to excessive iron absorption and accumulation. Hereditary hemochromatosis is primarily caused by mutations in the HFE gene, which encodes a protein that interacts with the transferrin receptor to regulate hepcidin expression. Mutations in HFE (e.g., C282Y and H63D) impair the sensing of body iron levels, resulting in reduced hepcidin production, even in the presence of iron overload. Without adequate hepcidin to downregulate ferroportin activity, iron absorption from the gut is unrestrained, and excess iron is absorbed and deposited in various organs, particularly the liver, heart, and pancreas[28].
This iron overload leads to tissue damage and fibrosis, especially in the liver (cirrhosis), heart (cardiomyopathy), and pancreas (diabetes), as excessive iron accumulation generates reactive oxygen species that cause oxidative damage to cells. Over time, untreated hemochromatosis can lead to severe organ dysfunction[29]. Other genetic mutations, such as those in the HAMP gene (encoding hepcidin itself) or TFR2 gene (encoding transferrin receptor 2), can also contribute to reduced hepcidin levels and the development of iron overload. Iron overload can also result from secondary causes such as repeated blood transfusions (e.g., in thalassemia), where iron intake exceeds the body's ability to store and utilize it. The dysregulation of hepcidin in these contexts also exacerbates iron accumulation[29]. In sum, hepcidin dysregulation plays a central role in a variety of anemic conditions, from iron deficiency to iron overload. The balance of hepcidin expression is crucial for maintaining iron homeostasis, and disruptions to this balance can lead to either insufficient iron availability for red blood cell production or harmful iron accumulation in tissues, both of which contribute to distinct pathophysiological processes in anemia. Understanding the molecular mechanisms underlying hepcidin dysregulation offers potential avenues for therapeutic intervention in anemia-related disorders [30, 31, 32, 33].

Targeting Hepcidin in Precision Medicine

The modulation of hepcidin represents a promising and evolving therapeutic avenue in the management of various forms of anemia and iron-related disorders. Hepcidin, a key regulator of iron homeostasis, influences the absorption, distribution, and storage of iron in the body. Its effects on iron metabolism make it a central target for therapeutic interventions aimed at correcting both iron overload and deficiency [34, 35].
Hepcidin Agonists: Hepcidin agonists, or mimetics, are designed to mimic or enhance the natural action of hepcidin, especially in conditions characterized by iron overload or dysregulated iron metabolism, such as anemia of chronic disease (ACD) or anemia associated with inflammation [36,37]. In these conditions, high levels of hepcidin are often responsible for limiting iron availability by sequestering iron in macrophages and reducing its absorption in the gut. Hepcidin agonists can potentially reduce iron absorption and increase iron sequestration, which is beneficial in controlling iron accumulation in tissues, preventing further organ damage[18]. These agents work by either increasing the endogenous production of hepcidin or directly activating hepcidin's effects on its receptor, ferroportin. Ferroportin is a transmembrane protein that facilitates iron efflux from cells, and its inhibition by hepcidin results in decreased iron release from stores. By enhancing hepcidin signaling, agonists could effectively reduce iron levels in conditions where excess iron causes toxicity, thereby improving clinical outcomes in diseases such as anemia of chronic disease and certain types of iron overload.
Hepcidin Antagonists: Conversely, hepcidin antagonists are being developed to counteract the inhibitory effects of hepcidin on iron absorption, particularly in patients suffering from iron deficiency anemia [38,39,40]. In conditions like anemia of inflammation or chronic disease, hepcidin levels are often elevated, leading to iron retention within macrophages and reduced iron availability for red blood cell production[31]. Hepcidin antagonists would function by blocking hepcidin's action, enabling more efficient iron absorption from the intestines and mobilization from iron stores, thus addressing the underlying iron deficiency. Several strategies are being explored for the development of hepcidin antagonists, including monoclonal antibodies that specifically target and neutralize hepcidin, as well as small molecules that inhibit hepcidin's binding to ferroportin. These antagonists could potentially restore normal iron metabolism in patients with conditions such as anemia due to chronic disease, inflammatory anemia, or anemia in individuals with suppressed hepcidin levels caused by various pathological factors [41, 42]. Iron Chelators and Hepcidin Modulators: Iron chelation therapy, which involves the use of agents to bind excess iron and promote its excretion, is already a cornerstone in the treatment of iron overload disorders, such as thalassemia, hereditary hemochromatosis, and transfusional iron overload [41,42]. However, the integration of hepcidin modulation with iron chelation therapy offers the potential for more refined control over iron levels, resulting in better management of these conditions. By

modulating hepcidin levels in combination with chelation, clinicians can enhance the effectiveness of iron removal therapies, making it possible to reduce iron overload while avoiding complications related to iron deficiency. Hepcidin modulators could help fine-tune iron metabolism in a way that reduces the risk of organ damage, particularly in the liver, heart, and pancreas, which are commonly affected by iron toxicity in conditions of iron overload. This dual approach could offer more personalized and effective treatment strategies, especially in complex cases where both iron overload and iron deficiency coexist [33]. In sum, the modulation of hepcidin offers an innovative approach to managing various iron-related disorders, with hepcidin agonists, antagonists, and combination therapies showing considerable promise in clinical trials. These therapies could significantly improve patient outcomes by targeting the root causes of iron dysregulation, whether it is excess iron accumulation or iron deficiency, providing more tailored and effective treatments for anemia and iron disorders.

Clinical Implications and Future Directions

The potential for hepcidin-targeted therapies in precision medicine is vast. By tailoring treatments to an individual's iron status and underlying pathology, clinicians could offer more personalized and effective therapies for anemia and iron-related disorders. However, the development of hepcidin-based therapies faces challenges, including the need for further research into the long-term safety and efficacy of these treatments. Additionally, understanding the molecular mechanisms governing hepcidin regulation could lead to the identification of novel biomarkers for early diagnosis and more accurate monitoring of treatment responses. The integration of hepcidin modulation into clinical practice will require careful consideration of patient-specific factors, including the type and cause of anemia, genetic factors, and coexisting medical conditions.

CONCLUSION

Hepcidin is a central regulator of iron metabolism, and its dysregulation plays a pivotal role in various forms of anemia and iron overload disorders. Targeting hepcidin holds promise as a new frontier in precision medicine for managing these conditions. Emerging therapeutic strategies to modulate hepcidin activity offer exciting potential for more personalized treatments that address the root causes of anemia and iron imbalance. As research continues, hepcidin-targeted therapies may become an integral part of anemia management, improving outcomes for patients worldwide.

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