STEM CELL HIERARCHY AND IMMUNE TOLERANCE IN BONE MARROW NICHES: MECHANISMS AND EMERGING PERSPECTIVES

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ABSTRACT

Bone marrow niches play a crucial role in regulating the hierarchy of hematopoietic stem cells (HSCs) and immune tolerance. In this microenvironment, interactions between niche cells and molecular components such as Nitric Oxide (NO) and the CD200 protein influence HSC function. NO acts as a signaling molecule that supports HSC proliferation at low concentrations and promotes differentiation at higher concentrations. HSCs with high NO production are often located near CD200-rich capillaries, providing protection from immune responses. Additionally, the signaling pathway involving IFT20, CD200, eNOS, and autophagy supports cellular homeostasis and the survival of cells under stress, including in cancer. The expression of CD200 in capillaries contributes to a tolerogenic immune environment, protecting tissues from excessive inflammation. However, excessive CD200 expression can be exploited by tumor cells to evade immune detection. This review highlights the critical role of the bone marrow microenvironment in regulating stem cell behavior and immune tolerance, offering insights into how these molecular interactions govern stem cell fate and tissue homeostasis. A deeper understanding of these pathways can pave the way for novel therapeutic strategies in blood disorders, cancer, and inflammatory diseases. By unraveling the intricate molecular interactions within the bone marrow niche, this research provides new perspectives on stem cell regulation and immune tolerance, with implications for improving clinical outcomes in stem cell-based therapies.

Keywords : Bone marrow niche, hematopoietic stem cells, nitric oxide, CD200, IFT 20

INTRODUCTION

The bone marrow is a highly specialized and dynamic microenvironment that serves as a central hub for hematopoiesis—the process of blood cell formation. Within this complex tissue, bone marrow niches provide distinct anatomical and functional compartments that regulate the behavior, maintenance, and differentiation of hematopoietic stem cells (HSCs) (Morrison & Scadden, 2014). These niches are composed of a diverse array of cellular and molecular components, including osteoblasts, endothelial cells, mesenchymal stromal cells, and immune cells, which together establish a finely tuned regulatory system governing stem cell hierarchy.

Somatic stem cells, despite originating from the same lineage, demonstrate considerable functional heterogeneity. This diversity is predominantly governed by their local microenvironment, or niche, which delivers specific molecular cues and cellular interactions that modulate key cellular processes such as proliferation, differentiation, and tissue regeneration (Picoli et al., 2021;

Farahzadi et al., 2024). Certain organs, such as the placenta, testes, and hair follicles, have developed unique mechanisms to protect themselves from immune system attacks. These areas are referred to as 'immune-privileged sites,' where immune responses are suppressed or prevented. This form of protection allows the acceptance of transplants from other individuals or even from different species, without the need for immunosuppressive drugs (Washburn et al., 2022; Kiselev & Park, 2024; Cohen et al., 2024).

Hematopoietic stem cells (HSCs) receive immunological protection through specialized interactions with immune cells within the bone marrow niche. In this microenvironment, HSCs are associated with regulatory T cells (Tregs), which are characterized by FOXP3 expression and high levels of the surface protein CD150. These Tregs play a crucial role in supporting the survival of allogeneic donor stem cells in immunocompetent mice, without the need for prior immunosuppressive therapy (Hirata et al., 2018; Camacho et al., 2020; Huang et al., 2024).

Recent studies have highlighted the dual role of bone marrow niches in not only maintaining the balance between self-renewal and differentiation of HSCs but also in promoting immune tolerance (Granata et al., 2022). Immune tolerance within the marrow environment ensures the prevention of autoimmunity and the elimination of autoreactive clones during early lymphocyte development. Disruption in this balance has been associated with hematological malignancies, immune dysregulation, and poor outcomes in stem cell transplantation (Kwon et al., 2024).

Understanding the interplay between stem-cell hierarchy and immune regulation within bone marrow niches provides crucial insights into both normal physiology and pathological conditions. This research aims to explore how specific cellular constituents and signaling pathways within the bone marrow niche orchestrate stem-cell fate decisions while maintaining immune homeostasis.

MATERIALS AND METHODS

The purpose of this literature review is to summarize and analyze current understanding of how bone marrow niches regulate stem cell hierarchy and immune tolerance. The review focuses on scientific publications published between January 2020 and March 2025, with particular emphasis on studies discussing molecular mechanisms and cellular interactions within the bone marrow microenvironment.

A systematic literature search was conducted using the PubMed and Google Scholar databases. Various keyword combinations were used in the process, including: “bone marrow niche,” “stem cell hierarchy,” “immune tolerance,” “hematopoietic stem cells,” “stem cell regulation,” and “bone marrow microenvironment.” These keywords were combined using Boolean operators to broaden the scope of relevant studies. Additionally, the search was limited to English-language articles and included only peer-reviewed publications, including original research articles, reviews, and meta-analyses.

Once the literature was gathered, a screening process was conducted by reviewing titles and abstracts to assess topic relevance. Articles that specifically addressed the role of the bone marrow niche in regulating stem cell function and immune system tolerance were selected for full-text review. Studies that focused solely on non-marrow tissues, were unrelated to stem cell hierarchy or immune tolerance, or were editorials or conference abstracts, were excluded from the review.

All selected articles were read thoroughly to extract key information regarding the types of niche-forming cells, signaling pathways involved in stem cell maintenance and differentiation, and the mechanisms

underlying immune tolerance within the bone marrow. The results of this analysis were synthesized narratively to illustrate conceptual relationships, current research trends, and future directions in this field.

RESULT AND DISCUSSION

Nitric Oxide (NO) Distinguishes HSCs based on Their Strength and Growth Rate

The NO (nitric oxide) molecule can be used to distinguish between healthy, robust hematopoietic stem cells (HSCs) and those that are slow-growing or poorly developing (Furuhashi et al., 2025). Nitric oxide (NO) plays a crucial role in distinguishing and influencing the function of hematopoietic stem cells (HSCs). HSCs with high levels of NO exhibit enhanced regenerative capacity, greater immune tolerance, and increased longevity, particularly in the context of transplantation and recovery following hematopoietic stress. Understanding the role of NO may pave the way for novel therapies in the treatment of blood disorders and improve the success of stem cell transplantation (Xu et al., 2021; Sinha et al., 2024).

Research shows that Nitric Oxide (NO) plays an important role in regulating the activity of HSCs, particularly in terms of proliferation, differentiation, and regenerative capacity. At low concentrations, NO supports HSC proliferation and maintains the expression of the CD34 marker. However, at high concentrations, NO promotes the differentiation of HSCs toward the myeloid progenitor lineage, reduces the fractions of HSCs and MPPs (Multipotent Progenitors), and decreases CD34 expression after several cell division cycles (Hümmer et al., 2021). At low concentrations, NO supports the proliferation and maintenance of HSCs. However, at high concentrations, NO promotes the differentiation of HSCs towards myeloid progenitors, reduces the HSC and MPP fractions, and decreases CD34 expression after several cell division cycles (Hümmer et al., 2021). NO has different effects depending on the age of HSCs. In young HSCs (6–8 weeks), NO increases CD34 expression and supports self-renewal. In contrast, in adult HSCs (10–12 weeks), NO promotes linear commitment and reduces regenerative capacity (Jalnapurkar et al., 2016). NO has different effects depending on the age of HSCs. In young HSCs, NO increases CD34 expression and supports self-renewal. In contrast, in adult HSCs, NO promotes linear commitment and reduces regenerative capacity. NO enhances the homing and engraftment abilities of HSCs through the cGMP-PKG signaling pathway, which can be utilized to improve the efficiency of HSC transplantation (Xu et al., 2021). NO enhances the homing and engraftment capabilities of HSCs through the cGMP-PKG signaling pathway, which can be utilized to improve the efficiency of HSC transplantation.

HSCs with high NO production are found near capillaries rich in CD200

Blood stem cells that produce high levels of nitric oxide (NO) tend to reside near small blood vessels in the bone marrow that are rich in the CD200 protein. This environment helps protect the stem cells from immune system attacks, allowing them to survive longer and function more effectively (Herbrich et al., 2021).

HSCs with high metabolic activity often produce more Nitric Oxide (NO). NO functions as a signaling molecule that can regulate the cell cycle, differentiation, as well as the homing and regenerative abilities of HSCs. CD200 is a surface protein expressed by various cell types, including capillary endothelial cells. CD200 is known to have an immunosuppressive role and contributes to

maintaining a microenvironment that supports HSC quiescence. In vivo mapping studies using imaging techniques and molecular markers have shown that HSCs with high NO production tend to accumulate near CD200-rich capillaries. This relationship is believed to be linked to the interaction between NO and the CD200–CD200R signaling pathway, which regulates local immunity and niche stability (Furuhashi et al., 2025).

For example, a study by Itkin et al. (2016) in the journal Nature found that sinusoidal blood vessels with high CD200 expression serve as primary sites for the retention of metabolically active HSCs, which exhibit higher NO production profiles compared to HSCs in the arteriolar niche (Itkin et al., 2016).

The relationship between Nitric Oxide production by HSCs and their geographical location within the bone marrow indicates the influence of the vascular niche on hematopoietic stem cell function. Capillaries expressing CD200 not only serve as sources of nutrients and oxygen but also act as "immune gatekeepers" that modulate HSC activation (Hümmer et al., 2021).

CD200 interacts with the CD200R receptor on HSCs or surrounding immune cells to dampen inflammatory signals. In this context, highly active HSCs (characterized by high NO production) are better protected from oxidative stress and excessive immune responses when located near vascular structures with high CD200 expression (Barclay et al., 2002).

This phenomenon suggests that HSC behavior is not governed solely by internal (genetic or epigenetic) factors, but also by spatial microenvironmental interactions, including signaling molecules such as NO and immune-regulatory proteins like CD200.

The IFT20 to CD200 signaling cascade that activates eNOS and autophagy

There is a cooperation pathway between four proteins—IFT20, CD200, eNOS, and autophagy—that play a role in maintaining stem cell health and protecting them from immune system attacks. Recent research indicates that the signaling pathway between IFT20 and CD200 plays a crucial role in regulating eNOS (endothelial Nitric Oxide Synthase) and autophagy, two vital cellular mechanisms. IFT20, a protein involved in intraflagellar transport, serves as an important link in the signaling pathway leading to the activation of CD200. CD200 activation then triggers eNOS activation, which plays a role in the production of nitric oxide (NO), a critical signaling molecule in the regulation of blood vessels and cellular homeostasis. Additionally, this pathway also affects autophagy, a process in which the cell breaks down and recycles its components to maintain cellular balance (Furuhashi et al., 2025).

Studies show that disruption of the IFT20-CD200 pathway can impact both of these mechanisms, potentially contributing to the pathology of various diseases, including cancer and cardiovascular diseases. eNOS activation through this pathway increases NO production, which in turn can influence processes such as cell proliferation and invasion, key characteristics of cancer development. At the same time, the regulation of autophagy through this signaling pathway is essential for maintaining cellular homeostasis and may affect the survival of cancer cells (Zhang et al., 2019).

The IFT20-CD200 pathway plays a crucial role in regulating various cellular physiological aspects, including vasodilation through eNOS and cellular quality control via autophagy. eNOS activation, dependent on this signaling, contributes to increased NO production, which has multiple effects in endothelial cells, including regulation of blood pressure and inflammatory responses. NO also has anti-apoptotic effects, which may explain

its role in supporting cancer cell growth and survival (Kim & Lee, 2025).

Autophagy, activated through this signaling pathway, plays a dual role in cancer. While autophagy can act as a protective mechanism against cellular stress and enhance cell survival, excessive activation of this process can help cancer cells survive under unfavorable conditions, such as nutrient or oxygen deprivation (Yun et al., 2020; Mehta & Shende, 2023; Jalali et al., 2025; Kausar et al., 2025; Tang et al., 2025).

Overall, the signals from IFT20 through CD200, activating eNOS and autophagy, form a complex network of interactions that not only play a role in cellular homeostasis but also in disease development, including cancer. Further research is needed to map the molecular mechanisms behind this pathway in greater detail and to explore potential therapies that could intervene in this signaling for the treatment of cancer or other related diseases (Jin et al., 2022; Jain et al., 2023).

The expression of CD200 in capillaries plays a role in enhancing protection against immune response

Capillaries that contain a high amount of CD200 protein can create an environment that is less likely to be attacked by the immune system. This means that the CD200 protein helps provide “special protection” to keep important cells (such as stem cells) safe and prevent them from being destroyed by the immune system.

The analysis results show that CD200 expression is consistently found in capillary endothelium across various tissues, including the brain, eye, and skin. CD200 expression levels increase in capillaries located in immunologically sensitive environments or those experiencing mild inflammation (Ko et al., 2009; Rütsche et al., 2022).

Several studies have reported that increased CD200 expression correlates with reduced infiltration of CD8+ T cells and activated macrophages around the capillaries. Furthermore, in animal models with genetically induced CD200 expression, there is a decrease in the production of proinflammatory cytokines such as TNF-α and IFN-γ in the tissues containing those capillaries (Hayakawa et al., 2016; Tonecka et al., 2021).

CD200 is a transmembrane glycoprotein that functions as a negative regulator of the immune system through its interaction with the CD200R receptor, which is found on myeloid cells such as macrophages and dendritic cells. The expression of CD200 in capillary endothelium plays a crucial role in creating a tolerogenic immune environment, particularly in tissues that require protection from excessive immune responses, such as the brain (blood–brain barrier) and the eye (immune-privileged site) (Pujol et al., 2024).

Studies have shown that CD200 expression in capillaries can suppress macrophage activation and prevent the transmigration of effector immune cells into the parenchymal tissue. This supports the hypothesis that CD200 serves as a local defense mechanism that prevents tissue damage caused by chronic inflammation or autoimmunity (Copland et al., 2007).

This protective mechanism is especially important in the context of chronic inflammatory diseases, neurodegenerative disorders, or post-transplantation conditions, where immune regulation is necessary to prevent rejection or tissue damage. In several disease models, the loss of CD200 expression has been associated with increased susceptibility to inflammation, reinforcing the idea that the presence of CD200 in capillaries is a key element in maintaining immune homeostasis (Pujol et al., 2024).

However, excessive CD200 expression can also be exploited by tumor cells or pathogens to evade immune detection. Therefore, the role of CD200 in capillaries

must be understood contextually, depending on the physiological or pathological condition involved. Overall, the available data support that CD200 expression in capillaries exerts a protective effect against immune responses by reducing the activity of proinflammatory immune cells (Mukhopadhyay et al., 2010).

SUMMARY

Nitric oxide (NO) plays a crucial role in regulating the function and characteristics of hematopoietic stem cells (HSCs). Low levels of NO support proliferation and maintain stemness, while high levels promote differentiation and reduce regenerative capacity, particularly in adult HSCs.

HSCs with high NO production tend to be located near CD200-rich capillaries, which provide protection against immune responses. The signaling pathway involving IFT20–CD200–eNOS also supports NO production and autophagy processes, helping maintain cellular balance and enabling cells to survive under stress conditions, including in cancer.

CD200 expression in capillaries helps create a tolerant immune environment, protecting tissues from excessive inflammation, although in some cases it can be exploited by tumor cells to evade immune detection.

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