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HISTOGENESIS OF HEMATOPOIETIC AND IMMUNE ORGANS DURING PRENATAL AND EARLY POSTNATAL DEVELOPMENT

Bekjanova Gulnara Marxabaevna
candidate of medical sciences senior teacher of the department of Medical biological sciences, EMU University
Turanova Shoxida Yunusalievna
teacher of the department of Medical biological sciences, EMU University

Abstract. This article reviews how hematopoietic and immune organs are built during prenatal life and how they rapidly adjust after birth. Developmental blood formation does not stay in one place. It shifts through a sequence of anatomical niches that provide distinct cellular signals and structural support. Early embryonic hematopoiesis begins outside the embryo, then definitive hematopoietic stem cells arise within embryonic vessels and seed fetal organs where expansion and lineage specification occur.

Keywords: histogenesis, hematopoiesis, hematopoietic stem cell, fetal liver niche, bone marrow microenvironment, thymus development.

INTRODUCTION

Histogenesis of hematopoietic and immune organs is the story of two systems that must be ready on day one yet are assembled under strict developmental constraints. Blood cell production has to start early to deliver oxygen and sustain growth, while immune development must strike a careful balance: build diversity and surveillance without triggering damaging reactions against self or the maternal environment. The key idea is that development is niche driven. A cell's fate is not decided only by its genes, but also by where it lands, which stromal partners surround it, and what growth factors and chemokines are available in that microenvironment. During prenatal life, hematopoiesis migrates through successive sites, each with characteristic histological features and functional outputs. At the same time, immune organs mature in a staged way: the thymus becomes a factory for T cell education, and secondary lymphoid tissues assemble the scaffolding that later supports antigen driven responses. These processes explain many clinical observations, from the distinctive blood counts of newborns to the higher vulnerability to certain infections in early life and the unique window when immune tolerance is shaped.

MATERIALS AND METHODS

Developmental hematopoiesis begins with an early wave that is optimized for the embryo's immediate needs. In humans, the first recognizable blood formation is associated with the yolk sac blood islands during early embryogenesis. This primitive program mainly produces nucleated erythroid cells and macrophage lineage cells that support oxygen delivery and tissue remodeling. Soon after, a definitive program emerges that can generate long lived hematopoietic stem cells capable of self renewal and multilineage differentiation. A major insight from modern developmental biology is that definitive hematopoietic stem cells arise from specialized endothelium in embryonic vessels through an endothelial to hematopoietic transition. The aorta gonad mesonephros region is a well known site where such stem cells appear, after which they enter circulation and colonize fetal organs. This vascular origin links histogenesis directly to vessel wall biology: hemogenic endothelial cells, surrounding mesenchyme, and local signaling pathways collectively shape the earliest stem cell pool [1].

Once stem cells are produced, expansion and lineage output depend strongly on the fetal liver. Histologically, the fetal liver becomes densely populated by hematopoietic cells that form clusters among hepatoblasts and supportive stromal elements. The fetal liver niche is not merely a storage site; it actively promotes proliferation, allowing stem and progenitor populations to expand far beyond what is typical in adult bone marrow. This high proliferative state is one

reason fetal hematopoietic stem cells behave differently from adult counterparts, including differences in cell cycle dynamics and stress responses. Alongside erythropoiesis, myeloid and early lymphoid progenitors are generated, preparing the organism for the transition to air breathing and microbial exposure. Reviews of fetal liver hematopoiesis emphasize that the niche is a composite of endothelial cells, hepatic stromal cells, and extracellular matrix cues that together regulate homing, retention, and differentiation. In short, the fetal liver acts like a highly productive industrial zone, while adult marrow is closer to a tightly regulated vault [2].

RESULTS AND DISCUSSION

The spleen can participate in prenatal hematopoiesis as well, though its role is more limited and context dependent than the fetal liver. What matters for immune histogenesis is that the spleen is also becoming a secondary lymphoid organ with distinct compartments. Over time, splenic architecture differentiates into red pulp regions involved in blood filtration and white pulp regions that organize lymphoid cells. This structural segregation is crucial because it later supports efficient antigen capture and the initiation of adaptive responses. In early life, however, full functional maturation of splenic immune microdomains depends on postnatal antigen exposure and lymphocyte trafficking patterns, so structure and function do not mature at exactly the same pace [3].

Bone marrow histogenesis is the pivotal transition for long term hematopoiesis. Prenatally, developing bones gradually establish marrow cavities and vascular networks, while stromal populations differentiate into the cell types that will form adult niches. The adult marrow environment maintains stem cells in a controlled balance between quiescence and activation. Classic models emphasize perivascular niches and osteogenic components, supported by chemokine gradients and adhesion mechanisms that retain stem cells and regulate their mobilization. The developmental handover from fetal liver dominance to marrow dominance occurs late in gestation and continues around birth, leading to the postnatal pattern in which marrow becomes the primary site of blood cell production. A key conceptual point is that hematopoiesis does not simply move location; it switches regulatory logic from expansion oriented fetal programs to maintenance oriented adult programs [5].

CONCLUSION

Prenatal and early postnatal histogenesis of hematopoietic and immune organs is best understood as a coordinated migration through niches combined with staged construction of immune microenvironments. Early embryonic hematopoiesis supplies immediate physiological needs, definitive stem cells arise from vascular endothelium, and the fetal liver provides the expansion platform that prepares the organism for birth. Bone marrow then assumes the long term role by building a tightly regulated niche system. In parallel, the thymus establishes T cell selection capacity before birth and relies on changing progenitor supply as fetal bone marrow develops, while secondary lymphoid organs assemble the architecture that becomes fully functional as postnatal antigen exposure increases. The practical takeaway is simple: newborn physiology reflects a system that is structurally present but functionally transitioning, and many hallmark adult immune behaviors are the result of both developmental programming and postnatal experience.

REFERENCES

1. Orkin S H, Zon L I. Hematopoiesis: an evolving paradigm for stem cell biology // Cell. 2008. Vol. 132. No. 4. P. 631-644. DOI: 10.1016/j.cell.2008.01.025.
2. Haddad R, Guimiot F, Six E, Jourquin F, Setterblad N, Kahn E, Yagello M, Schiffer C, Andre-Schmutz I, Cavazzana-Calvo M, Gluckman J C, Delezoide A L, Pflumio F, Canque B. Dynamics of thymus-colonizing cells during human development // Immunity. 2006. Vol. 24. No. 2. P. 217-230. DOI: 10.1016/j.immuni.2006.01.008.

3. Lewis K, Yoshimoto M, Takebe T. Fetal liver hematopoiesis: from development to delivery // Stem Cell Research and Therapy. 2021. Vol. 12. Article 139. DOI: 10.1186/s13287-021-02189-w.
4. Moore K L, Persaud T V N, Torchia M G. The Developing Human: Clinically Oriented Embryology. 11th ed. Saunders, 2019.
5. Abbas A K, Lichtman A H, Pillai S, Henrickson S. Cellular and Molecular Immunology. 11th ed. Elsevier, 2025.