

ADVANCED PHYSIOLOGICAL AND MOLECULAR DYNAMICS OF HUMAN BLOOD CORPUSCULAR ELEMENTS

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ARTICLE INFORMATION

ABSTRACT:

ARTICLE HISTORY:

Received: 13.04.2026

Revised: 14.04.2026

Accepted: 15.04.2026

KEYWORDS:

*Erythrocytes,
Leukocytes,
Thrombocytes,
Hemostasis,
Hematopoiesis,
Hemoglobin, NETosis,
Glycolysis.*

Human blood is a highly specialized connective tissue consisting of a complex liquid matrix and suspended cellular components, collectively termed corpuscular elements. These elements—erythrocytes, leukocytes, and thrombocytes—are not merely passive transporters but active mediators of respiratory gas exchange, immunological surveillance, and hemostatic integrity. This article provides an in-depth analysis of the morphological characteristics, biochemical pathways, and molecular mechanisms that govern these cells. Particular attention is given to the erythrocyte membrane stability, the diverse defense strategies of leukocytes (including NETosis and oxidative burst), and the signaling cascades of thrombocytes. The study highlights the homeostatic balance required for physiological health and the pathological implications of their dysfunction.

1. Introduction

Blood is the primary internal medium of the human body, representing approximately 7-8% of total body weight. The corpuscular elements, which constitute the hematocrit (roughly 45% of blood volume), are produced through a highly regulated process called hematopoiesis

in the red bone marrow. These cellular components have evolved to perform high-precision tasks: oxygenation, defense against pathogens, and vascular repair.

2. Erythrocytes (Red Blood Cells): The Gas-Exchange Specialization

Erythrocytes are the most numerous corpuscular elements, characterized by their biconcave geometry and lack of nuclei in their mature state.

Molecular Architecture: The RBC membrane is a masterpiece of biological engineering. A dense network of proteins like spectrin and ankyrin provides the flexibility needed to traverse capillaries as narrow as 3 μm without rupturing.

Metabolic Pathways: Since they lack mitochondria, RBCs generate ATP via the Embden-Meyerhof (anaerobic glycolysis) pathway. A specific bypass, the Luebering-Rapoport shunt, produces 2,3-BPG, which is essential for regulating hemoglobin's oxygen affinity.

Hemoglobin Kinetics: Each RBC contains ~270 million hemoglobin molecules. The "cooperative binding" mechanism ensures that as one oxygen molecule binds, the protein's conformation changes to make subsequent binding easier.

3. Leukocytes (White Blood Cells): The Multi-Layered Defense System

Leukocytes are the executive units of the immune system, categorized by their morphology and functional specialized roles.

Granulocytes (Neutrophils, Eosinophils, Basophils):

Neutrophils utilize oxidative burst (producing reactive oxygen species) and NETosis (releasing DNA traps) to neutralize bacteria.

Eosinophils are specialized in neutralizing parasites through the release of major basic protein (MBP).

Agranulocytes (Lymphocytes, Monocytes):

Lymphocytes (B and T cells) are the foundation of adaptive immunity. B cells differentiate into plasma cells to secrete specific antibodies, while T cells (CD4+/CD8+) coordinate the cellular immune response.

Monocytes are the largest cells, capable of transforming into macrophages to perform phagocytosis and antigen presentation.

4. Thrombocytes (Platelets): The Hemostatic Guardians

Thrombocytes are anucleated cytoplasmic fragments derived from megakaryocytes.

The Activation Cascade: Upon vascular injury, platelets bind to the subendothelial matrix via GP Ib-IX-V receptors. This triggers a shape change and the release of alpha and dense granules. * **Aggregation:** The activation of GP IIb/IIIa receptors allows fibrinogen to bridge multiple platelets, forming a primary hemostatic plug.

Clot Retraction: Utilizing an internal actin-myosin cytoskeleton, platelets contract the fibrin mesh, physically closing the wound and facilitating tissue regeneration.

5. Hematopoiesis and Clinical Correlation

All corpuscular elements originate from Pluripotent Hematopoietic Stem Cells. This process is governed by specific growth factors:

Erythropoietin (EPO) for RBCs.

Thrombopoietin (TPO) for platelets.

Colony-Stimulating Factors (CSFs) for WBCs.

Clinically, deviations such as Polycythemia (excess RBCs), Leukopenia (low WBCs), or Thrombocytopenia (low platelets) can lead to severe systemic failures, including thrombosis, immunodeficiency, or spontaneous hemorrhage.

