



ISSN: 2456-0057

IJPNPE 2019; 4(1): 2345-2347

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www.journalofsports.com

Received: 22-11-2018

Accepted: 26-12-2018

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Erythrocytes function, production, death and disposal, homeostasis and disorders: An overview

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Abstract

A cell that contains hemoglobin and can carry oxygen to the body. The reddish color is due to the hemoglobin. Erythrocytes have two principal functions: 1) to pick up oxygen from the lungs and deliver it to tissues elsewhere, and 2) to pick up carbon dioxide from the tissues and unload it in the lungs. An erythrocyte lives for an average of 120 days from the time it is produced in the red bone marrow until it dies and breaks up. The production of erythrocyte is known as erythropoiesis. The process of RBCs production normally takes 3 to 5 days and involve 5 major developments: a reduction in cell size, an increase in cell number, the synthesis of hemoglobin, the loss of nucleus, and the loss of other organelles. Any imbalance between the rates of erythropoiesis and RBC destruction may produce an excess or deficiency of red blood cells. Erythrocyte disorders includes: anemia, sickle cell anemia, thalassemia, erythrocytosis.

Keywords: hemoglobin, erythropoiesis, anemia, reticulocyte, hypoxemia, hemolysis, tubules, macrophages

Introduction

1.1 Erythrocyte

Erythrocytes, or red blood cells (RBCs), have two principal functions: 1) to pick up oxygen from the lungs and deliver it to tissues elsewhere, and 2) to pick up carbon dioxide from the tissues and unload it in the lungs. RBCs are the most abundant formed elements of the blood and therefore the most obvious things one sees upon its microscopic examination. They are also the most critical to survival; although a severe deficiency of leukocytes or platelets can be fatal within a few days, a severe deficiency of erythrocytes can be fatal within a few minutes. It is the lack of life-giving oxygen, carried by erythrocytes, that leads rapidly to death in cases of major trauma or hemorrhage.

1.2 Form and Function

An erythrocyte is a discoidal cell with a thick rim and a thin sunken center. It is about 7.5 μm in diameter and 2.0 μm thick at the rim. Although most cells, including white blood cells, have an abundance of organelles, RBCs lose nearly all organelles during their development and are thus remarkably devoid of internal structure. When viewed with the transmission electron microscope, the interior of an RBC appears uniformly gray. Lacking mitochondria, RBCs rely exclusively on anaerobic fermentation to produce ATP. The lack of aerobic respiration prevents them from consuming the oxygen that they must transport to other tissues. Erythrocytes are the only cells in the body that carry on anaerobic fermentation indefinitely. Lacking a nucleus and DNA, RBCs also are incapable of protein synthesis and mitosis.

The plasma membrane of a mature RBC has glycoproteins and glycolipids on the outer surface that determine a person's blood type. On its inner surface are two cytoskeletal proteins, spectrin and actin, that give the membrane resilience and durability. This is especially important when RBCs pass through small blood capillaries and sinusoids. Many of these passages are narrower than the diameter of RBCs to stretch, bend, and fold as they squeeze through. When they enter larger vessels, RBCs spring back to this discoid shape like an air-filled inner tube.

The cytoplasm of an RBC consists mainly of a 33% solution of hemoglobin. This is the red

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pigment that gives an RBC its color and name. It is known especially for its oxygen transport function, but it also aids in the transport of carbon dioxide and the buffering of blood pH. Although the lack of a nucleus makes an RBC unable to repair itself, it has an overriding advantage: The biconcave shape gives the cell a much greater ratio of surface area to volume, which enables oxygen and carbon dioxide to diffuse quickly to and from the hemoglobin.

1.3 The Erythrocyte Life Cycle

An erythrocyte lives for an average of 120 days from the time it is produced in the red bone marrow until it dies and breaks up. In a state of balance and stable RBC count, the birth and deaths of RBCs amount to about 2.5 million cells per second, or a packed cell volume of 20 ml of RBCs per day.

1.4 Erythrocyte Production

The production of erythrocyte is known as erythropoiesis. The process of RBCs production normally takes 3 to 5 days and involve 5 major developments:

- a reduction in cell size
- an increase in cell number
- the synthesis of hemoglobin
- the loss of nucleus
- and the loss of other organelles

It begins when a pluripotent stem cell (PPSC) becomes an erythrocyte colony-forming unit (ECFU), which has receptors for the hormone erythropoietin (EPO). EPO stimulates the ECFU to transform into an erythroblast. Erythroblasts multiply and synthesize hemoglobin. When this task is completed, the nucleus shrivels and is discharged from the cell. The cell is now called a reticulocyte, named for a temporary network (reticulum) composed of ribosome clusters.

Reticulocytes leave the bone marrow and enter the circulating blood. In a day or two, the last of the polyribosomes disintegrate and disappear, and the cell is a mature erythrocyte. Normally, about 0.5% to 1.5% of the circulating RBCs are reticulocytes, but this percentage rises under certain circumstances. Blood loss, for example, stimulates accelerate erythropoiesis and leads to an increasing number of reticulocytes in circulation- as if the bone marrow is in such a hurry to replenish the lost RBCs that it releases many developing RBCs into circulation a little early.

1.5 Erythrocyte Homeostasis

The red blood cells count is maintained in a class is negative feedback manner. If the count should drop (for example, because of hemorrhaging), it may result in a state of hypoxemia- O₂ deficiency in the blood. The kidneys detect this and increase their EPO output. After three or four days, the RBC count begins to rise and reverses the hypoxemia that started the process.

Hypoxemia has many causes other than blood loss. Another cause is a low level of oxygen in the atmosphere. The blood of an average adult has about 5 million RBCs/μL, but people who live at high altitude may have counts of 7 to 8 million RBCs/μL. Another cause of hypoxemia is an abrupt increase in the body's oxygen consumption. If a lethargic person suddenly takes up tennis or aerobics, for example, the muscle consume oxygen more rapidly and create a state of hypoxemia that stimulates erythropoiesis. Endurance trained athletes commonly have RBC counts as high as 6.5 million RBCs/μL.

Not all hypoxemia can be corrected by increasing erythropoiesis. In emphysema, for example, less lung tissue is available to oxygenate the blood. Raising the RBC count cannot correct this, but the kidneys and bone marrow have no way of knowing this. The RBC count continuous to rise in a

futile attempt to restore homeostasis, resulting in a dangerous excess called polycythemia.

1.6 Erythrocyte Death and Disposal

As an RBC ages and its membrane proteins (specially spectrin) deteriorate, the membrane grows increasingly fragile. Without a nucleus or ribosomes, an RBC cannot synthesize new spectrin. Many RBCs die in the spleen, which has been called the 'erythrocyte graveyard'. The spleen has channels as narrow as 3μm that severely test the ability of old, fragile RBCs to squeeze through the organ. Old cells become trapped, broken up, and destroyed. An enlarged and tender spleen may indicate diseases in which RBCs are rapidly breaking down.

Hemolysis, the rupture of RBCs, releases hemoglobin and leaves empty plasma membranes. The membrane fragments are easily digested by macrophages in the liver and spleen, but hemoglobin disposal is a bit more complicated. It must be disposed of efficiently, however, or it can block kidney tubules and cause renal failure. Macrophages begin the disposal process by separating the heme from the globin. They hydrolyze the globin into free amino acids, which become part of the body's general pool of amino acids available for protein synthesis or energy releasing catabolism. Disposing of heme is another matter. First, the macrophage removes the iron and releases it into the blood, where it combines with transferrin and is used or stored in the same way as dietary iron. The macrophage converts the rest of the heme into a greenish pigment called biliverdin, then further converts most of this to a yellow-green pigment called bilirubin. Bilirubin is released by the macrophages and binds to albumin in the blood plasma. The liver removes it from the albumin and secretes it into the bile, to which it imparts a dark green color as the bile becomes concentrated in the gallbladder. Biliverdin and bilirubin are collectively known as bile pigments. The gall bladder discharges the bile into the small intestine, where bacteria convert bilirubin to urobilinogen, responsible for the brown color of the feces. Another hemoglobin breakdown pigment, urochrome, produces the yellow color of the urine. A high level of bilirubin in the blood causes jaundice, a yellowish cast in light-colored skin and the whites of eyes. Jaundice may be a sign of rapid hemolysis or a liver disease or a bile duct obstruction that interferes with bilirubin disposal.

1.7 Erythrocyte Disorders

Any imbalance between the rates of erythropoiesis and RBC destruction may produce an excess or deficiency of red blood cells. An RBC excess is called polycythemia, and a deficiency of either RBCs or hemoglobin is called anemia.

Anemia

Anemia occurs when your body is not producing enough red blood cells or when the red blood cells are not properly functioning. When a problem with red blood cell production or function occurs, the body does not get the oxygen that it needs which could cause on or more following symptoms:

- Weakness
- Shortness of breath
- Fast or irregular heart beat
- Headache
- Cold hands or feet
- Pale or yellow skin
- Chest pain

Chronic disease, infections, poor diet, and intestinal disorders are common cause of anemia. The risk of anemia increases with age and with activity. People who run or engage in other strenuous sports or activities have a greater chance for

developing anemia due to their blood cells breaking down. People with one or more of the following chronic conditions are also at a greater risk for developing anemia:

- Rheumatoid arthritis or other autoimmune disorders
- Kidney disease
- Cancer
- Liver disease
- Thyroid disease
- Inflammatory bowel disease

Sickle Cell Anemia

It is the most common type of inherited blood disorders. Healthy blood cells are fluid in shape so they can easily pass through the small blood vessels in the body. In people with sickle cell, their red blood cells are shaped like a half moon (or a sickle), so they have difficulty flowing into the small blood vessels and often get blocked. The lack of blood flow can cause pain, infection, and problems with organs not receiving the adequate amount of blood.

Thalassemia

Thalassemia is an inherited blood disorder in which the body makes an abnormal form of hemoglobin which results in large numbers of red blood cells being destroyed, which leads to anemia. A blood test is used to diagnose thalassemia and during a physical exam, The doctor will often look for an enlarged spleen, which is often a symptoms. Other symptoms include:

- Fatigue
- Shortness of breath
- Yellow skin (jaundice)
- Bone deformities in the face
- Growth failure

Erythrocytosis

Erythrocytosis is a rare inherited blood disorder where the body produces too many cells. The excess blood cells increase the risk for blood clots which could prevent blood flow to major organs such as the heart, lungs, or brain. There are two types of erythrocytosis. Familial erythrocytosis is where a person inherits the disorder, and non-familial erythrocytosis is where a person acquires the disease later in life. Non-familial erythrocytosis can be caused from long term exposure to high altitude, chronic lung disease, sleep apnea, chronic smoking, or certain types of tumors. Symptoms of erythrocytosis include:

- Headache
- Dizziness
- Nosebleeds
- Shortness of breath

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