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Mechanisms Of Phagocytosis Of Neutrophils

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Abstract: This article analyzes the mechanisms of phagocytosis of neutrophil granulocytes, their role in the defense processes against microorganisms, their molecular basis, and new mechanisms identified in recent years. Neutrophils are the most important component of the immune system, which eliminate pathogens through phagocytosis, degranulation, and NETosis. Studies conducted during 2015–2025 have further revealed the mechanisms of oxidative burst, receptor-mediated signal transduction, and endosomal activity in phagocytosis.

Keywords: Neutrophil, phagocytosis, immune response, oxidative burst, NETosis, ROS, pathogen elimination, endosome.

Introduction: Research objective: The main objective of the study is to analyze the mechanisms of phagocytosis in neutrophil cells, their molecular basis, and changes in protective function based on modern scientific sources.

Research methods: Systematic review and comparative methods were used in the work. During the preparation of the article, more than 20 articles published in PubMed, Nature, ScienceDirect, and Cell Press databases between 2015 and 2025 were analyzed.

[1] Neutrophils (neutrophil cells) belong to the class of granulocytes that constitute the first line of defense of the human immune system, and they play an important role in the recognition, phagocytosis, and elimination of microorganisms. Studies conducted in recent decades (2015–2025) show that the mechanisms of phagocytosis of neutrophils have been studied more deeply at the molecular level [2]. This mechanism is of fundamental importance not only in eliminating bacterial infections, but also in regulating inflammatory processes [3].

[2] The process of phagocytosis carried out by neutrophils consists of several stages: recognition of microbes by chemical signals (chemotaxis), recognition of pathogens by opsonization, formation of phagosome, and their destruction by oxidative burst [4]. During this process, the NADPH oxidase complex, mitochondrial ROS production, and lysosomal enzymes are actively involved [5]. Modern molecular studies have revealed that mitochondria are not only a source of energy, but also a regulator of phagocytosis [6]. [3] Advanced biomedical research conducted in the 2020s has shown that the phagocytic ability of neutrophils is closely related to their metabolic state [7]. In particular, the anaerobic breakdown of glucose (glycolysis) is actively used as a source of energy necessary for phagocytosis. At the same time, metabolic exchanges between immune cells through mitochondrial transfer mechanisms have been observed [8].

[4] Neutrophil activation is an important pathophysiological factor not only in infections but also in chronic inflammation, cancer, and autoimmune diseases [9]. For example, a study published in 2025 in the journal Cell Communication and Signaling found that mitochondrial signaling enhances neutrophil phagocytic activity [10]. Other studies have elucidated

the molecular role of the Syk-ERK signaling pathway, the FcγRIII receptor system, and calcium ions in phagocytosis [11].

[5] Additionally, functional food components in the diet, particularly β-glucans and vitamin C, have been reported as biological modulators that promote neutrophil phagocytosis [12]. This suggests the need to combine pharmacological and nutraceutical approaches in supporting a healthy immune response.

[6] Thus, the mechanisms of phagocytosis of neutrophils are one of the central directions of modern immunology, and their study contributes to a better understanding of infectious diseases, autoimmune syndromes, and oncological processes. Studies conducted between 2015 and 2025 have linked the mechanisms of phagocytosis to intracellular signaling, energy metabolism, and genetic regulation, paving the way for the development of new therapeutic strategies [13]. Neutrophils are the most active component of the innate immune system, destroying bacteria, fungi, and viruses that have entered the body through phagocytosis [6]. The process of phagocytosis consists of the stages of pathogen recognition, adhesion, ingestion (internalization), and degradation [7]. Reactive oxygen species (ROS) generated by the NADPH oxidase system of neutrophils increase the efficiency of the phagocytosis process [8].

RESULTS

[1] Studies have shown that the phagocytic capacity of neutrophils is based on complex molecular and intracellular mechanisms. Experiments conducted between 2015 and 2025 revealed that the production of ROS (reactive oxygen species) in neutrophils is a key factor determining the efficiency of phagocytosis. Studies published in the journal Cell Reports have shown that mitochondrial ROS (mtROS) act as a signal to activate the phagocytosis process, and its reduction reduces the ability to eliminate microbes [2].

[2] Another factor that enhances the phagocytic response of neutrophils is the FcγRIII receptor system, which transmits signals through the Syk and ERK kinases. A study published in the journal Biomedicines in 2021 showed that activation of these receptors, in addition to phagocytosis, also activates the process of NETosis [3]. These results indicate that the protective mechanisms of neutrophils function as an interconnected network.

[3] Molecular observations since 2022 have shown that the energetic role of mitochondria in phagocytosis is not limited to their function in ATP production. They also coordinate phagosome-mitochondrial communication through calcium ions and oxidative signals [4]. This mechanism ensures efficient pathogen degradation. [4] According to new data published in Nature Immunology (2023), the ATP/ADP ratio in neutrophils has been identified as an important bioenergetic indicator of the intensity of phagocytosis [5]. Reduced mitochondrial function or impaired glycolytic flux reduces the rate of phagocytosis, which leads to chronic inflammatory states.

[5] In a 2025 article in Cell Communication and Signaling, direct transfer of mitochondria between neutrophils was observed, a phenomenon termed “mitochondrial transfer.” This mechanism plays an important role in restoring phagocytosis and maintaining metabolic cooperation between immune cells [6].

[6] Studies on the effects of food components on immune activity have also yielded interesting results. In a 2025 article in Carbohydrate Polymers, it was shown that β-glucans significantly increase neutrophil phagocytosis, stimulate ROS production, and accelerate phagosome maturation [7]. Similarly, antioxidants (e.g., vitamin C and polyphenols) balance oxidative stress during phagocytosis and support long-term cellular defenses [8].

[7] Clinical observations conducted in 2019–2024 have shown that the phagocytic activity of neutrophils is closely related to age, metabolic status, and chronic diseases [9]. For example, in diabetic patients, high glucose levels reduce ROS production, reducing the efficiency of phagocytosis. Therefore, metabolic stability is emphasized as an important factor to support immune activity.

[8] In conclusion, scientific works published in 2015–2025 have revealed several new layers of phagocytosis mechanisms:

Mitochondrial ROS act as a necessary signal for phagocytosis.

The FcγRIII/Syk-ERK pathway plays a key role in the integration of phagocytosis and NETosis.

Mitochondrial transfer and glycolytic flux ensure intercellular immune synchrony.

Biologically active food components modulate phagocytic activity.

These results open up new pharmacological approaches in immunology, such as mitochondrial-supporting therapies or drugs based on natural modulators

1. Differential role of receptors: FcγR and CR3 receptors activate different signaling pathways depending on the type of pathogen

[1] The efficiency of neutrophil phagocytosis largely depends on the functional differentiation of surface

receptors. In particular, FcγR (Fc gamma receptor) and CR3 (Complement receptor 3) receptors activate different signaling pathways in the recognition of microorganisms and phagosome formation. According to an article published in the journal Clinical Reviews in Allergy & Immunology in 2020, FcγR receptors mainly recognize opsonized pathogens (bacteria coated with immunoglobulin G) and activate the Syk-ERK-MAPK signaling pathway, which ensures the rapid initiation of phagocytosis [2].

[2] In contrast, CR3 receptors (CD11b/CD18 integrin complex) mainly recognize microorganisms opsonized with complement C3b or iC3b. These receptors, through Rac1 and PI3K, rearrange the cytoskeleton and gradually but steadily form phagosome [3]. Therefore, while the FcγR pathway induces a rapid but short-term response, the CR3 pathway provides a slower but more sustained and profound protective response.

[3] Studies have shown that these two receptors can function in an antagonistic or synergistic manner. According to Nature Communications (2021), ROS (reactive oxygen species) generated by the FcγR act as a modulator that stimulates the activity of the CR3 pathway [4]. This increases the ability of neutrophils to respond adaptively to various pathogens.

[4] A study published in Frontiers in Immunology in 2023 identified the bacterial species as a factor that determines which receptor is dominant in phagocytosis. For example, while FcγR signaling is dominant in Staphylococcus aureus infection, the CR3 pathway is dominant in complement-coated pathogens such as Escherichia coli and Candida albicans [5].

[5] The functional status of the neutrophil and the tissue environment in which the microbes are exposed also alter the functional ratio of these two receptors. According to Cell Communication and Signaling (2025), the CR3 pathway is activated under hypoxia, as oxygen deprivation reduces FcγR signaling [6].

[6] Thus, the differential role of FcγR and CR3 receptors determines the adaptive and context-dependent nature of neutrophil phagocytosis. This mechanism is important not only for the effective elimination of infections, but also for maintaining control of the inflammatory process. New molecular therapies are able to restore the balance of the immune system by selectively modulating these two receptor pathways [7]

2. ROS and mitochondrial signaling: ROS are not only involved in microbial killing, but also in signal transduction processes

[1] Although reactive oxygen species (ROS) produced by neutrophils have long been considered primarily as toxic molecules that kill microbes, in recent years they have been shown to play an important role in intracellular signal transduction processes [2]. Superoxide radicals generated by the NADPH oxidase complex kill microbes within the phagosome, but these molecules also activate various signaling cascades in the cytoplasm [3].

[2] A study published in Nature Communications in 2021 demonstrated that ROS activates signaling pathways such as MAPK, NF-κB, and HIF-1α in neutrophils, leading to the production of inflammatory mediators [4]. In particular, activation of HIF-1α enhances the phagocytic response under hypoxic conditions, which makes neutrophils more susceptible to infection [5].

[3] However, mitochondria are not the only source of ROS. According to Cell Reports (2023), mitoROS, which are produced by complexes II and III of the mitochondrial electron transport chain, act as a signal to stimulate phagosome formation and actin polymerization [6]. This process regulates cytoskeletal dynamics through the PKCζ and p38 MAPK pathways [7].

[4] A new study published in Cell Communication and Signaling (2025) has shown that mitochondrial signaling also interacts with immune cells. Researchers have shown that when phagocytic neutrophils are activated, they transfer mitochondria to other neutrophils via extracellular vesicles, synchronizing the immune response through metabolic cooperation [8].

[5] ROS, particularly H_2O_2, act as “second messengers” within the cell. They oxidize cysteine residues in proteins and inhibit the activity of phosphatases (e.g., SHP-1, PTEN), resulting in prolonged signaling cascades [9]. Thus, ROS are important molecules that regulate the duration and efficiency of phagocytosis [10].

[6] In addition, excessive ROS production can lead to cell damage and apoptosis. Therefore, antioxidant systems (e.g., glutathione, superoxide dismutase, catalase) in neutrophils balance ROS levels [11]. According to Frontiers in Immunology (2024), when this balance is disrupted, an “oxidative stress cycle” occurs, leading to chronic inflammation and tissue fibrosis [12].

[7] In general, ROS and mitochondrial signaling are considered to be integrated mechanisms that not only control the elimination of pathogens, but also the duration, direction, and intensity of the inflammatory response of neutrophil phagocytosis. In this regard, they are recognized as central signaling nodes in the maintenance of immune homeostasis [13]

3. The relationship between NETosis and phagocytosis

Recent studies have demonstrated a complex but coordinated relationship between the NETosis process

of neutrophils (i.e., “neutrophil extracellular traps” — NETs) and the mechanisms of phagocytosis [1]. Initially, NETosis was considered only as a mechanism for capturing pathogens and immobilizing them with DNA structures. However, scientific developments in 2020–2025 have shown that the process of NET formation promotes increased phagocytosis, and conversely, phagocytosis triggers signals that activate NETosis [2].

The initiation of NETosis in neutrophils is closely linked to the production of ROS (reactive oxygen species), which occurs through the activation of the NADPH oxidase complex [3]. ROS are not only involved in microbial clearance, but also in the enhancement of NETosis through mitochondrial signaling and the Syk-ERK signaling pathway [4]. In studies by Lu et al. (2021), a mutually reinforcing signaling mechanism was observed between NET formation and phagocytosis due to the activation of FcγRIII receptors [5]. In a study published in Biomedicines in 2021 by Bohländer et al., it was found that the interaction of NETosis with IgG and IgA immunoglobulins plays an important role in the phagocytosis process, and in particular, blocking FcαRI and FcγRI receptors reduces the efficiency of phagocytosis [6]. This confirms that NETosis and phagocytosis processes work as a coordinated immune response system, not just in parallel.

Also, analyses by Lopez-Pedrera and Barbarroja (2020) showed that excessive activation of NETosis in combination with phagocytosis in rheumatoid arthritis and other inflammatory diseases leads to tissue damage [7]. This situation indicates the need to consider NETosis not only as a protective, but also as a pathological mechanism.

2025 studies (Changaei et al., Cell Communication and Signaling) showed that mitochondrial transfer and energy metabolism during NETosis increase phagocytosis activity, which revealed a new role of mitochondria in the immune response as the “energy base” of neutrophils [8]. Thus, the interaction between NETosis and phagocytosis is considered not only a protective mechanism, but also as an important part of cellular signal transduction.

4. Auto-destruction mechanism: After phagocytosis, neutrophils are eliminated by apoptotic pathway, which reduces inflammation

Neutrophils are inherently short-lived phagocytes, which constitute the first line of defense in the inflammatory process. However, their excessive activity or long-term survival can cause chronic inflammation that damages tissues. Therefore, after completing the phagocytosis process, neutrophils undergo physiological auto-destruction by apoptosis, which ensures the controlled termination of the inflammatory process [1].

Studies in 2025 have shown that neutrophil apoptosis is closely related to the production of mitochondrial ROS. ROS not only kill microbes, but also activate intracellular signal transduction, activating pro-apoptotic proteins of the Bcl-2 family (Bax, Bak), thereby reducing the mitochondrial membrane potential [2]. This triggers the release of cytochrome c and the caspase-9/3 pathway, which are key steps in the completion of the apoptotic process.

A 2025 study published in Frontiers in Immunology (Zhang et al., 2025) [3] showed that neutrophil apoptosis is regulated by the TLR4/MyD88/NF-κB pathway. Overactivation of these pathways can delay apoptosis and prolong inflammation. Therefore, TLR4 blockers or NF-κB inhibitors have been proposed as potential therapeutic agents to reduce inflammation.

Another important aspect is that apoptotic neutrophils are engulfed by macrophages through a process of “efferosis”, which reduces the secretion of inflammatory mediators (IL-1β, TNF-α) and increases the production of anti-inflammatory cytokines (IL-10, TGF-β) [4]. At the same time, it was noted in an article published in the journal Cell Communication and Signaling in 2025 that mitochondrial functions play an important role not only for energy supply, but also in the coordination of apoptosis and immune signaling [5].

The auto-liquidation of neutrophils ensures a “clean” termination of the inflammatory process, i.e. tissue regeneration and restoration of immune balance. On the contrary, impaired apoptosis has been observed in chronic diseases, such as rheumatoid arthritis, chronic lung diseases and autoimmune syndromes [6]. Therefore, modulation of the apoptotic process by drugs is one of the promising directions in the management of inflammation.

DISCUSSION

Current analyses confirm that neutrophil phagocytosis is a complex networked process. Neutrophil activity, ROS production, and NETosis are closely linked [13]. Studies in 2022–2024 have shown that mitochondrial ROS production stimulates phagocytosis [14]. In addition, new data highlight the importance of endosome-mitochondrial communication [15].

CONCLUSION

Neutrophils are one of the most important cells of the immune system, which protect the body from pathogens through mechanisms of phagocytosis, NETosis, ROS production, and apoptotic auto-destruction [1]. Scientific studies in 2015–2025 have shown that these mechanisms work in an

interconnected and coordinated manner [2].

Phagocytosis is the main defense mechanism of neutrophils, which involves the recognition, engulfment, and elimination of pathogens. In this process, receptors such as FcγR and CR3 activate different signaling pathways depending on the pathogen type, which ensures the efficiency of phagocytosis and the specificity of the immune response [3]. In addition, reactive oxygen species (ROS) are not only molecules that kill microbes, but also act as important second messengers that activate intracellular signaling cascades [4]. In particular, mitochondrial ROS are considered to be a central molecular node that controls the processes of phagocytosis, inflammation, and cell death [5].

New scientific developments in 2020–2025 have shed light on the dynamic relationship between NETosis and phagocytosis. The DNA networks (NETs) formed during NETosis function synchronously with phagocytosis, which significantly enhances protection against pathogens [6]. However, excessive activation of this process can cause tissue damage in inflammatory diseases, such as rheumatoid arthritis or sepsis [7]. Therefore, maintaining a physiological balance between NETosis and phagocytosis is essential for immune homeostasis.

The life cycle of neutrophils ends with apoptosis at the end of phagocytosis, which naturally terminates the inflammatory process and is eliminated by macrophages through efferocytosis [8]. Delayed or impaired apoptosis can lead to chronic inflammatory states, and therefore therapeutic modulation of this process is one of the promising directions of modern immune pharmacology [9].

In conclusion, neutrophil phagocytosis is not only a defense system against pathogens, but also a complex biological network that coordinates energy, signaling, and inflammation. Recent studies indicate that the role of neutrophils in the immune response should be considered not only at the level of “microbial killers”, but also as a regulatory and homeostatic center of the immune system [10]. This knowledge will serve as the basis for the development of new diagnostic and therapeutic strategies for autoimmune diseases, chronic inflammatory syndromes, and infectious pathologies in the future.

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