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Factors Of Chemotaxis Of Eosinophil Cells

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Abstract

Eosinophils are an important component of the immune system, and their activity plays a central role in allergic reactions, asthma, parasitic infections, and autoimmune processes [1]. Chemotaxis is the main mechanism by which eosinophils are attracted to inflammatory foci. This article reviews the chemotaxis factors of eosinophils, their associated signaling pathways, and molecular mediators.

Keywords: Eosinophil, chemotaxis, IL-5, eotaxin, CCR3, cytokines, inflammation, immune response.

Introduction

The aim of the study was to identify the main cytokines, chemokines, and their receptors that regulate the chemotaxis of eosinophils and to shed light on their role in immunopathology based on current research in 2015–2025.

Research Methods: More than 20 articles published in PubMed, Scopus, Nature, Frontiers in Immunology, and Journal of Allergy and Clinical Immunology from 2015 to 2025 were reviewed [2]. Selected sources included clinical observations, in vitro and in vivo experimental studies, and meta-analyses.

Eosinophils are a class of granulocytes of the immune system and are important mediators of inflammatory

responses [1]. They are mainly involved in defense against parasitic infections, allergic diseases, and tissue repair [2]. Studies conducted in the last decade have confirmed that chemotaxis is at the heart of eosinophil activity. This process represents the directed movement of cells to the site of inflammation or infection and is regulated by various chemokines and cytokines [3].

The mechanism of chemotaxis is based on complex signaling cascades, in which eotaxins (CCL11, CCL24, CCL26) and their receptors, especially CCR3, play a central role [4]. Through this ligand-receptor interaction, eosinophils adhere to the endothelial surface, rearrange the actin cytoskeleton, and are directed to the site of inflammation [5]. Cytokines such as IL-5, IL-33, and TSLP have been identified as activating or potentiating factors in this process [6]. In particular, IL-5 is a key mediator in the release of eosinophils from the bone marrow, prolonging their lifespan, and enhancing CCR3 expression [7]. In allergic diseases, bronchial asthma, and atopic dermatitis, excessive proliferation of eosinophils and their accumulation in tissues determine the severity of clinical symptoms [8]. Therefore, studying the molecular pathways that control eosinophil chemotaxis is important for a deeper understanding of the pathogenesis of these diseases and for the identification of new therapeutic targets [9]. Scientific studies published between 2015 and 2025 have highlighted the integrated role of the eotaxin–CCR3 axis, the IL-5–IL5Rα signaling pathway, and the IL-33–ST2 system in eosinophil migration [10]. Another important aspect is that eosinophils are not only effector cells that secrete inflammatory mediators, but also active participants in immune regulation [11]. They maintain immune homeostasis through bidirectional communication with T cells, mast cells, and dendritic cells [12]. Against the background of such complex interactions, the process of chemotaxis mobilizes eosinophils as “directed soldiers” of the immune system.

Results

Molecular and immunological studies conducted over the past decade have revealed that the process of chemotaxis of eosinophils is based on multistep, complex, and multicomponent signaling mechanisms [1]. Eosinophil activation, movement, and tissue homing are coordinated by mediators such as IL-5, eotaxin (CCL11), RANTES (CCL5), IL-33, and TSLP [2]. In particular, the IL-5–CCR3 signaling axis has been identified as a key system that enhances eosinophil migration [3]. Clinical observations have shown that patients with allergic asthma and eosinophilic bronchitis have significantly higher levels of eotaxin-1 in the blood [4]. Activation of the eotaxin-CCR3 complex promotes eosinophil adhesion to endothelial cells, their transendothelial migration, and homing to the site of inflammation [5]. In vitro studies have also shown a 3-fold increase in CCR3 receptor expression in eosinophils stimulated with IL-5 [6].

In animal models, blocking the IL-33-ST2 signaling pathway reduced eosinophil recruitment by up to 60%, further demonstrating the importance of this pathway [7]. In contrast, increased levels of TSLP and IL-13 cytokines increased eotaxin synthesis and further activated eosinophil migration [8]. These results suggest the existence of an interconnected cytokine network that enhances eosinophil chemotaxis.

Animal models have shown that the IL-33-ST2 signaling pathway is one of the main mechanisms that direct eosinophils to the site of inflammation [1]. Blocking this pathway not only reduces the number of eosinophils, but also significantly reduces their release of inflammatory mediators, major basic protein (MBP) and eosinophil cationic protein (ECP) [2]. As a result, the level of inflammation in the lung tissue is reduced by up to 60%, which confirms the crucial role of the IL-33–ST2 axis in pathological processes [3].

It has also been found that the cytokines TSLP (thymic stromal lymphopoietin) and IL-13 have a synergistic effect that enhances eosinophil chemotaxis [4]. These cytokines are released from epithelial cells and enhance the synthesis of eotaxin-1, eotaxin-2 and eotaxin-3 (CCL11, CCL24, CCL26), as a result of which eosinophils are actively directed to the inflammatory focus through the CCR3 receptor [5]. In a study conducted by Licona-Limón et al. (2020), it was observed that eosinophil migration increased by 2.3 times when the TSLP-IL-13 axis was activated [6].

In experiments by Cayrol et al. (2018), it was found that blocking the IL-33–ST2 signaling pathway reduced eosinophil recruitment by up to 60% in models of inflammation [7]. When this pathway was blocked, TSLP expression was also reduced, indicating a link between these two pathways. Furthermore, a recent study by Seluk et al. (2025) showed that eosinophil infiltration was reduced by up to 70% using novel biotherapeutics that block IL-5, IL-4/13, and TSLP receptors [8]. These

findings highlight the interconnected nature of the immune system and the existence of a complex cytokine network that activates eosinophil chemotaxis [9]. Thus, the IL-33–ST2, TSLP-IL-13, and eotaxin-CCR3 pathways act in a synergistic manner. This knowledge is important for a deeper understanding of the pathogenesis of diseases such as allergic asthma, atopic dermatitis, and eosinophilic gastroenteritis and for the development of new drug targets [10]. In the 2020s, biotechnological drugs aimed at reducing eosinophil activity are being developed. For example, anti-IL-5 monoclonal antibodies (mepolizumab, reslizumab, benralizumab) have been shown in clinical trials to reduce eosinophil numbers and alleviate asthma symptoms [9]. New generation nanovector-based drugs show promising results in selectively blocking the CCR3 receptor and inhibiting eosinophil migration [10].

Also, in a study by L. Seluk et al. (2025), it was noted that the combined use of novel therapeutic agents that block IL-5, IL-4/13, and TSLP receptors reduced eosinophil infiltration by up to 70% [11]. These results provide a scientific basis for new approaches to the treatment of asthma and other eosinophil-associated inflammatory diseases in the future.

In general, scientific studies conducted between 2015 and 2025 have scientifically confirmed that eosinophil chemotaxis is coordinated by the IL-5–CCR3, IL-33–ST2, and TSLP–eotaxin pathways, and that this process is an important component of the immune response [12].

In recent years, the study of the mechanisms of eosinophil chemotaxis has become an important scientific direction in immunology, since this process plays a crucial role not only in allergic inflammation, but also in the pathogenesis of asthma, atopic dermatitis, and gastroesophageal eosinophilic diseases [1]. Although eosinophils are important in the immune defense and tissue repair processes in the body, their excessive activation causes chronic inflammation [2].

The IL-5–CCR3 signaling pathway is one of the main mechanisms controlling eosinophil migration, and blocking this pathway provides clinically significant relief [3]. In clinical studies, anti–IL-5 drugs (mepolizumab, reslizumab, benralizumab) have been shown to not only reduce eosinophil numbers but also reduce inflammatory mediators in the airways [4]. At the same time, the IL-33–ST2 and TSLP–IL-13 signaling pathways appear to be part of an interconnected cytokine network that enhances eosinophil chemotaxis [5].

IL-33 is released as an “alarmin” produced by epithelial cells when tissue is damaged and activates eosinophils through the ST2 receptor [6]. This process interacts with TSLP, which increases the production of eotaxin and recruits eosinophils to the inflammatory focus through the CCR3 receptor [7]. As Licona-Limón et al. (2020) noted, the combined activity of IL-33 and TSLP cytokines increases the number of eosinophils by 2–3 times, which ensures the continuation of chronic inflammation [8].

In a new clinical trial conducted by Seluk et al. (2025), a combined therapy model that blocks IL-5, IL-4/13 and TSLP receptors was tested, and the results showed a reduction in eosinophil infiltration of up to 70% [9]. This indicates that multi-targeted drug strategies are promising.

Also, research is ongoing on methods to block eosinophil migration by selectively blocking the CCR3 receptor using nanovector-based drugs [10]. Studies in this direction allow us to move to a new stage of pharmacological control of eosinophils.

The results discussed show that eosinophil chemotaxis cannot be explained by a single factor alone — this process is based on a complex interaction of multipathway cytokine networks. The IL-33–ST2, IL-5–CCR3, TSLP–IL-13 axes act in a mutually reinforcing manner, which creates network-like mechanisms of activity in the immune system [11].

Therefore, future studies are aimed at studying the epigenetic control of eosinophil chemotaxis, the interaction of signaling networks with intracellular transcription factors, and its regulation by microRNAs [12]. Based on these approaches, it is expected that new personalized immunotherapy concepts will be developed

Discussion

Eosinophil chemotaxis involves a complex network regulation. IL-5 increases not only proliferation and viability, but also the expression of the CCR3 receptor [11]. This sensitizes eosinophils to eotaxins. IL-4 and IL-13, which are secreted by T cells, also enhance eotaxin production [12].

In an experimental model, it was found that eosinophil migration was significantly reduced when the IL-33-ST2 signaling pathway was blocked [13]. This suggests that the interaction of several cytokine pathways determines eosinophil activity.

Conclusion

Chemotaxis of eosinophil cells is central to the control of inflammatory processes. The eotaxin-CCR3 axis, the IL-5 signaling pathway, and IL-33/TSLP are key drivers of this process [14]. In-depth study of these mechanisms will allow the identification of new drug targets for asthma and other allergic diseases.

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