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The Mechanism of Action of Infliximab in Inflammatory Diseases

Geevan Sagar*

Department of Pharmacy, Al-Nasser University, Sana'a, Yemen

Introduction

Inflammatory diseases, particularly autoimmune disorders such as rheumatoid arthritis, Crohn's disease, and ulcerative colitis, are characterized by persistent, dysregulated inflammation, leading to tissue damage and a reduction in quality of life. The underlying mechanism in many of these diseases involves an overactive immune response, where pro-inflammatory cytokines, such as Tumor Necrosis Factor-Alpha (TNF-α), play a crucial role in mediating inflammation and disease progression. Infliximab, a monoclonal antibody against TNF-α, is one of the most widely used biologic therapies for treating various inflammatory conditions. Since its approval in the late 1990s, infliximab has revolutionized the management of diseases that are resistant to traditional therapies, offering patients significant improvements in disease control, symptom relief, and quality of life.

Infliximab binds specifically to TNF-α, neutralizing its activity and preventing its interaction with cell surface receptors. By inhibiting TNF-α, infliximab can halt the cascade of inflammatory responses that contribute to disease progression in several autoimmune and inflammatory conditions. This article will explore the mechanism of action of infliximab in treating inflammatory diseases, focusing on how it impacts immune system signaling, inflammation, and tissue damage. Furthermore, it will discuss the clinical outcomes and potential limitations associated with its use [1].

Description

Tumor Necrosis Factor-Alpha (TNF-α) is a cytokine that plays a critical role in regulating immune responses and inflammation. It is produced primarily by activated macrophages, but it can also be secreted by T cells, neutrophils, and other immune cells. TNF-α exerts its effects through the binding of two receptors, TNFR1 and TNFR2, both of which activate different signaling pathways. In a typical immune response, TNF-α is involved in pathogen defense, inflammation, and tissue repair. However, in chronic inflammatory diseases, excessive or prolonged production of TNF-α leads to sustained inflammation and tissue damage, which is a hallmark of diseases such as rheumatoid arthritis, Crohn's disease, psoriasis, and ankylosing spondylitis. In these conditions, TNF-α contributes to the inflammatory process by promoting the activation of immune cells, increasing the production of other cytokines, and inducing the release of proteases and matrix metalloproteinases (MMPs) that degrade tissues. TNF-α also enhances vascular permeability, facilitating the infiltration of inflammatory cells into affected tissues, which further exacerbates the inflammatory cycle. As a result, inhibiting TNF-α signaling is an effective strategy for managing these diseases [2].

***Address for Correspondence:** Geevan Sagar, Department of Pharmacy, Al-Nasser University, Sana'a, Yemen; E-mail: sagar.geevan@gmail.com*

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Infliximab is a chimeric monoclonal antibody composed of both human and murine components. Its primary action is to bind to TNF-α and neutralize its activity, preventing TNF-α from binding to its receptors (TNFR1 and TNFR2) on the surface of cells. By doing so, infliximab disrupts the downstream signaling that leads to inflammation and tissue damage. Neutralization of Soluble TNF-α: Infliximab binds directly to soluble TNF-α, preventing it from interacting with TNF receptors. This blocks the activation of the NF-κB pathway, a major transcription factor responsible for the expression of pro-inflammatory genes.

Prevention of TNF Receptor Activation: By binding to TNF-α, infliximab prevents the receptor-mediated signaling pathways that would otherwise lead to the production of inflammatory cytokines (e.g., IL-1, IL-6) and enzymes that degrade tissue, such as matrix Metalloproteinases (MMPs) [3]. This helps reduce the inflammatory response and protects tissues from further damage. Induction of Apoptosis: Infliximab can also induce apoptosis (programmed cell death) in certain cells, such as activated T lymphocytes, which contribute to inflammation. This helps reduce the overall immune cell burden in inflamed tissues.

By neutralizing TNF-α, infliximab decreases the recruitment of immune cells to inflamed tissues. This reduces the accumulation of inflammatory cells and prevents the exacerbation of tissue injury. Additionally, infliximab may promote the restoration of tissue integrity by reducing the overproduction of pro-inflammatory cytokines and growth factors that contribute to fibrosis and scarring. Infliximab has demonstrated efficacy in the treatment of a wide range of chronic inflammatory diseases, particularly those where TNF-α plays a central role in the pathogenesis. Infliximab has been shown to significantly reduce disease activity, prevent joint damage, and improve physical function in patients with RA. It is often used in combination with methotrexate, a conventional Disease-Modifying Antirheumatic Drug (DMARD), to enhance therapeutic efficacy.

Infliximab is widely used in the management of inflammatory bowel diseases, especially in patients with moderate to severe disease who do not respond to conventional therapies. It reduces inflammation in the gastrointestinal tract, promoting healing of ulcers and preventing disease flares. Infliximab has been effective in treating moderate-to-severe plaque psoriasis by reducing the inflammatory response and promoting skin cell turnover. Infliximab is used to reduce inflammation in the spine and joints of patients with ankylosing spondylitis, helping alleviate pain and improve mobility [4].

Despite its success in treating these diseases, infliximab is not suitable for all patients. Potential side effects include an increased risk of infections, infusion reactions, and the development of certain types of malignancies. Additionally, some patients may develop antibodies to infliximab, leading to reduced efficacy or hypersensitivity reactions. While infliximab has revolutionized the treatment of inflammatory diseases, there are challenges associated with its use. Its high cost limits accessibility for some patients, and it requires administration via intravenous infusion, which may be inconvenient for long-term treatment. Furthermore, as with other biologics, infliximab can increase the risk of serious infections, including tuberculosis, and may require careful screening and monitoring [5].

Conclusion

Infliximab has proven to be a highly effective therapeutic agent for

managing a variety of chronic inflammatory diseases, particularly those in which TNF-α plays a central role in disease pathogenesis. By specifically targeting and neutralizing TNF-α, infliximab reduces inflammation, prevents tissue damage, and improves clinical outcomes in diseases like rheumatoid arthritis, Crohn's disease, and psoriasis. Its ability to modulate immune responses at a molecular level has made it a critical tool in modern inflammatory disease management.

However, despite its therapeutic benefits, infliximab is not without risks, including potential side effects and complications related to long-term use. Ongoing research aims to refine the use of infliximab by exploring combination therapies, identifying biomarkers for patient response, and developing newer, more targeted biologic agents. The continued evolution of biologic therapies will enhance our ability to manage chronic inflammatory diseases more effectively, improving both the clinical outcomes and quality of life for affected patients.

Acknowledgment

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Conflict of Interest

None.

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