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InnoCyto Inc.

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Anti-Human TNF alpha (Infliximab Biosimilar)

Catalog Number:	503301, 503302, 503303
Size:	1 mg, 5 mg, 20 mg
Target Name:	TNFα
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	Infliximab
Application:	Neutralization, Intracellular Flow cytometry, animal model study
Reactivity:	Human
Format:	Liquid
Product Description:	Infliximab Biosimilar, TNF alpha Monoclonal Antibody
Isotype:	Human IgG1
Clonality:	Recombinant
Immunogen:	Recombinant human TNF protein
Clone Number:	cA2
Species specificity:	Human
Purity:	>95% by reducing SDS-PAGE
Grade:	In vivo
Min Sample Size:	1 mg
Storage Conditions:	4ºC
Maximal Shelf Life:	12 months
Synonyms:	TNFa

BACKGROUND INFORMATION

Infliximab is a chimeric monoclonal antibody belonging to the immunoglobulin G1 kappa (IgG1κ) subclass, engineered to specifically bind and neutralize tumor necrosis factor-alpha (TNF-α), a proinflammatory cytokine. Structurally, Infliximab is a glycoprotein with a molecular weight of approximately 149 kilodaltons (kDa). It is composed of two identical heavy chains and two identical light chains connected by disulfide bonds, forming the characteristic Y-shaped antibody structure. The molecule combines murine (mouse-derived) variable regions with human constant regions, thereby retaining antigen specificity while exhibiting improved molecular compatibility with human immune system components. It is expressed in mammalian cell systems, such as Chinese Hamster Ovary (CHO) cells, to ensure proper folding, disulfide linkage formation, and glycosylation.

The antigen-binding fragments (Fab) of Infliximab contain complementarity-determining regions (CDRs) within the variable domains of both heavy (VH) and light (VL) chains. These CDRs form a high-affinity binding site that specifically recognizes and binds to

soluble and transmembrane forms of TNF-α. The binding interface is stabilized by hydrogen bonds and hydrophobic interactions, producing nanomolar-level affinity. By occupying key epitopes on TNF-α, Infliximab prevents it from engaging its native receptors (TNFR1 and TNFR2) on target cells, thereby inhibiting downstream signaling cascades such as the NF-κB and MAPK pathways, which regulate cytokine production, apoptosis, and cell activation in experimental systems.

The Fc (fragment crystallizable) region of the human IgG1 portion provides Infliximab with structural stability, extended serum half-life through neonatal Fc receptor (FcRn) recycling, and the potential for engaging Fc gamma receptors (FcγRs). This enables effector mechanisms such as complement activation and antibody-dependent cellular cytotoxicity (ADCC) in vitro. Overall, Infliximab represents a rationally designed chimeric IgG1 antibody that combines specific cytokine neutralization with structural robustness, an essential model for exploring antibody-mediated modulation of immune signaling pathways in biochemical and cellular research.

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