

Anti-Human IL-12 / IL-23 (Briakinumab Biosimilar)

Catalog Number:	501001, 501002, 501003
Size:	1 mg, 5 mg, 20 mg
Target Name:	IL-12/IL-23
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	Briakinumab
Application:	Neutralization, Intracellular Flow cytometry, animal model study
Reactivity:	Human
Format:	Liquid
Product Description:	Anti-Human IL-12 / IL-23 (Briakinumab Biosimilar)
Isotype:	Human IgG1
Clonality:	Recombinant
Immunogen:	Human IL-12 / IL-23
Species specificity:	Human
Purity:	>95% by reducing SDS-PAGE
Grade:	In vivo
Min Sample Size:	1 mg
Storage Conditions:	4°C
Maximal Shelf Life:	12 months
Synonyms:	p40
RRID:	AB_3739286

BACKGROUND INFORMATION

Briakinumab is a recombinant, fully human monoclonal antibody that belongs to the immunoglobulin G1 (IgG1) subclass. It is designed to specifically bind to the shared p40 subunit of the cytokines interleukin-12 (IL-12) and interleukin-23 (IL-23), thereby interfering with their interaction with the interleukin-12 receptor β 1 (IL-12R β 1) on the surface of immune cells. Structurally, Briakinumab is a typical IgG1 molecule composed of two identical heavy chains and two identical light chains connected by disulfide bonds, forming a Y-shaped configuration. It has an approximate molecular weight of 148 kilodaltons (kDa) and is produced using mammalian expression systems, such as Chinese Hamster Ovary (CHO) cells, to ensure proper glycosylation and protein folding consistent with human antibodies.

The variable regions of the heavy and light chains in Briakinumab contain complementarity-determining regions (CDRs), which define its antigen-binding specificity. These regions form a paratope that engages with the p40 subunit of IL-12 and IL-23 through

non-covalent interactions, including hydrogen bonding and van der Waals forces. By binding to the p40 subunit, Briakinumab prevents IL-12 and IL-23 from interacting with their receptor complexes on T cells and natural killer (NK) cells. This inhibition disrupts downstream signaling through the Janus kinase-signal transducer and activator of transcription (JAK-STAT) pathway, limiting the production of proinflammatory cytokines such as interferon-gamma (IFN- γ) and interleukin-17 (IL-17) in experimental systems.

The Fc region of Briakinumab provides structural stability, prolongs its serum half-life by interacting with neonatal Fc receptors (FcRn), and maintains effector functions characteristic of IgG1 antibodies. It is glycosylated at the asparagine 297 residue within the CH2 domain, contributing to molecular solubility and conformation.

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