

Anti-Human GP IIb/IIIa (Abciximab Biosimilar)

Catalog Number:	500101, 500102, 500103
Size:	1 mg, 5 mg, 20 mg
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	Abciximab
Application:	Flow cytometry, animal model study
Format:	Liquid
Product Description:	Abciximab Biosimilar, Glycoprotein IIb/IIIa Receptor Monoclonal Antibody
Isotype:	Hman IgG1 Fab
Clonality:	Recombinant
Immunogen:	Human Glycoprotein IIb/IIIa Receptor
Species specificity:	Human
Purity:	>95% by reducing SDS-PAGE
Grade:	In vivo
Storage Conditions:	4°C
Maximal Shelf Life:	12 months
Synonyms:	GP1Ib, GP1IIa
RRID:	AB_3739278

BACKGROUND INFORMATION

Abciximab is a chimeric monoclonal antibody fragment developed to selectively bind and inhibit specific integrin receptors on platelet surfaces. Structurally, it is the Fab (antigen-binding) fragment of the monoclonal antibody 7E3, designed using recombinant DNA technology. This antibody originates from a murine (mouse) immunoglobulin engineered to contain both murine variable regions and human constant regions, resulting in a chimeric composition that combines high specificity with structural compatibility to human proteins. The molecule has a molecular mass of approximately 47.6 kilodaltons (kDa) and maintains the typical immunoglobulin Fab architecture, comprising one complete light chain and the variable and first constant domains of the heavy chain, connected via disulfide bonds.

Functionally, Abciximab exerts its activity through high-affinity binding to the glycoprotein (GP) IIb/IIIa receptor complex, also known as integrin α IIb β 3, on platelets. This receptor plays a crucial role in mediating platelet aggregation by serving as the final common pathway for fibrinogen, von Willebrand factor, and other adhesive ligand binding. By occupying the ligand-binding sites of this integrin complex, Abciximab sterically hinders the interaction between platelets and adhesive molecules, effectively inhibiting crosslinking and aggregation processes. In addition to the α IIb β 3 receptor, Abciximab also displays binding affinity toward related integrins such as α v β 3 and Mac-1 (CD11b/CD18), contributing to its ability to modulate a wider range of cell adhesion phenomena

in research contexts.

At the molecular level, Abciximab's binding is non-competitive and characterized by slow dissociation kinetics, allowing the molecule to remain attached to its receptor targets for extended periods. The interaction occurs mainly through complementarity-determining regions (CDRs) located in the antibody's variable domains, which recognize specific conformational epitopes on the integrin surface. Through these mechanisms, Abciximab provides a precise biochemical model for investigating integrin-mediated signaling, receptor-ligand dynamics, and the molecular regulation of platelet adhesion and aggregation.

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