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InnoCyto Inc.

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Anti-Human PD-L1 (Atezolizumab Biosimilar)

Catalog Number:	500601, 500602, 500603, 500604, 500605
Size:	1 mg, 5 mg, 20 mg, 5 mg, 20 mg
Target Name:	PD-L1, PDL1 CD274, B7-H1
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	Atezolizuma
Application:	Flow cytometry, animal model study
Reactivity:	Human
Format:	Liquid
Product Description:	Atezolizumab Biosimilar, Endotoxin 0.05 EU/mg
Isotype:	Human IgG1
Clonality:	Recombinant
Immunogen:	Human PD-L1
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:	CD274, B7-H1
RRID:	AB_3739282
Antibody Type:	Recombinant
Reactivity:	Human
GeneID:	29126

BACKGROUND INFORMATION

Atezolizumab is a fully humanized monoclonal antibody designed to specifically target and bind to the programmed death-ligand 1 (PD-L1) molecule. Structurally, it belongs to the immunoglobulin G1 (IgG1) subclass and has a molecular weight of approximately 145 kilodaltons (kDa). The molecule consists of two identical heavy chains and two identical light chains, each containing variable (V) and constant (C) domains, joined by disulfide bonds to form the classical Y-shaped antibody structure.

The variable regions of Atezolizumab's Fab fragments contain highly specific complementarity-determining regions (CDRs) that recognize and bind to human PD-L1. This binding interface involves both hydrogen bonding and hydrophobic interactions that confer high affinity and selectivity. By engaging PD-L1, Atezolizumab prevents it from interacting with its receptors, programmed death-1 (PD-1) and B7.1 (CD80), on T cells and other immune cells. The inhibition of these ligand-receptor interactions disrupts key inhibitory signaling cascades that normally attenuate T-cell activation, thereby restoring or sustaining the functional activity of immune effector cells in experimental systems.

Importantly, the Fc region of Atezolizumab has been selectively engineered to eliminate antibody-dependent cellular cytotoxicity (ADCC) and complement-dependent cytotoxicity (CDC). This is achieved through specific amino acid substitutions in the Fc domain that reduce binding to Fc gamma receptors (FcγRs) and complement component C1q. These modifications allow Atezolizumab to block PD-L1 function without depleting cells that express the molecule. Biophysically, the molecule demonstrates high solubility and stability, with a long serum half-life maintained through neonatal Fc receptor (FcRn) recycling. In sum, Atezolizumab exemplifies a rationally engineered IgG1 framework optimized for precise ligand blockade and controlled immune signaling modulation.

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