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Anti-Human PD1 (Spartalizumab Biosimilar)

Catalog Number:	505801, 505802, 505803
Size:	1 mg, 5 mg, 20 mg
Target Name:	PD1, PD-1, PDCD1, CD279, SLEB2
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	Spartalizumab
Application:	Flow cytometry, animal model study
Reactivity:	Human
Format:	Liquid
Product Description:	Anti-Human PD-1 (Spartalizumab Biosimilar)
Isotype:	Human IgG4
Clonality:	Recombinant
Immunogen:	Human PD1
Species specificity:	Human
Purity:	>95% by reducing SDS-PAGE
Grade:	In vivo
Storage Conditions:	4ºC
Maximal Shelf Life:	12 months
Synonyms:	CD279
Antibody Type:	Recombinant
Reactivity:	Human

BACKGROUND INFORMATION

Spartalizumab is a fully human monoclonal antibody of the immunoglobulin G4 kappa (IgG4κ) subclass, specifically engineered to bind to and inhibit the programmed death-1 (PD-1) receptor, an immune checkpoint expressed on activated T lymphocytes. Structurally, Spartalizumab is a glycoprotein with a molecular weight of approximately 147 kilodaltons (kDa). It consists of two identical heavy chains and two identical light chains linked by interchain disulfide bonds, forming the typical Y-shaped architecture found in IgG antibodies. Each heavy chain comprises a variable (VH) domain and three constant domains (CH1-CH3), while each light chain contains one variable (VL) and one constant (CL) domain. It is expressed in mammalian cell systems, commonly Chinese Hamster Ovary (CHO) cells, to ensure proper glycosylation, folding, and structural integrity.

The variable domains contain complementarity-determining regions (CDRs) that form the antigen-binding sites responsible for high-affinity and specific interaction with PD-1’s extracellular domain. The CDRs engage PD-1 through a combination of hydrogen

bonding, electrostatic interactions, and hydrophobic contacts, blocking its binding to endogenous ligands programmed death-ligand 1 (PD-L1) and programmed death-ligand 2 (PD-L2). This blockade disrupts the inhibitory signaling cascade mediated by PD-1 engagement, which normally suppresses T-cell activation. In immunological model systems, Spartalizumab prevents the attenuation of T-cell receptor (TCR) signaling, contributing to sustained cytokine production, proliferation, and effector activity.

The Fc (fragment crystallizable) region of Spartalizumab is derived from the IgG4 subclass, which is selected for its naturally low capability to elicit immune effector functions such as antibody-dependent cellular cytotoxicity (ADCC) and complement-dependent cytotoxicity (CDC). To enhance structural stability, the hinge region contains an S228P mutation that prevents half-antibody exchange. The Fc domain also interacts with neonatal Fc receptors (FcRn), enabling antibody recycling and prolonging plasma half-life.

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