

## Anti-Human PD-1 (Cemiplimab Biosimilar)

<b>Catalog Number:</b>	501301, 501302, 501303
<b>Size:</b>	1 mg, 5 mg, 20 mg
<b>Regulatory Status:</b>	RUO

### PRODUCT DETAILS

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

### BACKGROUND INFORMATION

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Cemiplimab is a fully human immunoglobulin G4 (IgG4) monoclonal antibody engineered to specifically bind the programmed death-1 (PD-1) receptor on T lymphocytes. The molecule has a molecular weight of approximately 146 kilodaltons (kDa) and is produced using recombinant DNA technology in mammalian cell expression systems such as Chinese Hamster Ovary (CHO) cells. It is composed of two identical heavy chains and two identical light chains connected by disulfide bonds, forming the classic Y-shaped structure characteristic of antibodies. Each heavy chain contains one variable (VH) and three constant (CH1-CH3) domains, while each light chain consists of a variable (VL) and a constant (CL) domain.

The antigen-binding specificity of Cemiplimab is determined by its complementarity-determining regions (CDRs) located within the VH and VL domains. These CDRs form a highly complementary surface that enables precise and high-affinity interaction with the PD-1 receptor. By binding to PD-1, Cemiplimab blocks its engagement with the endogenous ligands programmed death-ligand 1 (PD-L1) and programmed death-ligand 2 (PD-L2). This interruption prevents ligand-induced signaling that would ordinarily attenuate T-cell activation, cytokine production, and proliferation. In experimental immunological studies, such blockade restores or enhances T-cell effector activity and modifies intracellular signaling cascades involved in adaptive immune regulation.

The Fc region of Cemiplimab, derived from the IgG4 subclass, includes an engineered serine-to-proline (S228P) substitution in the hinge region, stabilizing the molecule and preventing half-antibody exchange. Unlike IgG1 antibodies, IgG4 has minimal affinity for

Fc gamma receptors (FcγRs) and complement proteins, which minimizes effector functions such as antibody-dependent cellular cytotoxicity (ADCC) and complement-dependent cytotoxicity (CDC). The Fc region also interacts with neonatal Fc receptors (FcRn), extending serum half-life by protecting the antibody from lysosomal degradation. Overall, Cemiplimab exemplifies precision antibody engineering, combining structural stability, selective receptor targeting, and finely tuned immune modulation for mechanistic research applications.

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