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Anti-Human PD-L1 (Envafolimab Biosimilar)

Catalog Number:	502501, 502502, 502503
Size:	1 mg, 5 mg, 20 mg
Target Name:	PD-L1, PDL1 CD274, B7-H1
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	Envafolimab
Application:	Flow cytometry, animal model study
Reactivity:	Human
Format:	Liquid
Product Description:	Anti-Human PD-L1 (Envafolimab Biosimilar)
Isotype:	Human IgG1
Clonality:	Recombinant
Immunogen:	Human PD-L1
Species specificity:	Human
Purity:	>95% by reducing SDS-PAGE
Grade:	In vivo
Storage Conditions:	4ºC
Maximal Shelf Life:	12 months
Synonyms:	CD274, B7-H1
Antibody Type:	Recombinant
Reactivity:	Human
GeneID:	29126

BACKGROUND INFORMATION

Envafolimab, also known as KN035, is a novel recombinant single-domain antibody–Fc fusion protein engineered to specifically target programmed death-ligand 1 (PD-L1). Structurally, Envafolimab is distinct from conventional monoclonal antibodies because it consists of only the heavy-chain variable domain (VH) derived from camelid antibody fragments, fused directly to the Fc (fragment crystallizable) region of human immunoglobulin G1 (IgG1). This configuration results in a smaller, single-chain architecture with a molecular weight of approximately 80 kilodaltons (kDa), roughly half that of a full-length IgG molecule. The molecule is produced in mammalian expression systems to maintain proper folding, disulfide bond formation, and glycosylation within the Fc portion.

The single-domain variable region of Envafolimab provides specificity for PD-L1 through high-affinity interactions dominated by

hydrogen bonding and hydrophobic contacts. Upon binding, Envafolimab effectively blocks PD-L1 from engaging its receptors, programmed death-1 (PD-1) and B7.1 (CD80), on activated T lymphocytes and antigen-presenting cells. This blockade disrupts inhibitory signaling pathways that would otherwise reduce T-cell proliferation and cytokine secretion. Functionally, this mechanism restores or enhances immune cell activation in experimental systems, making Envafolimab a valuable tool for the study of immune checkpoint regulation and ligand–receptor signaling.

The Fc region of Envafolimab contributes structural stability, mediates dimerization, and extends circulatory half-life through interactions with neonatal Fc receptors (FcRn), which protect the molecule from lysosomal degradation. Because the Fc segment is derived from IgG1, it retains potential for limited Fc receptor binding, though it is not primarily designed for effector function. The compact and stable single-domain architecture grants Envafolimab notable advantages in solubility, tissue penetration, and molecular stability. Overall, Envafolimab exemplifies advanced protein engineering that fuses minimal antigen-binding domains with antibody Fc functionality to create a small, high-affinity immune checkpoint inhibitor optimized for mechanistic and biochemical research applications.

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