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In Vivo Star Anti-Mouse CD274 (PD-L1) Antibody

Catalog Number:	507801, 507802, 507803
Size:	1 mg, 5 mg, 25 mg
Target Name:	PD-L1, PDL1 CD274, B7-H1
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	10F.9G2
Application:	ELISA, WB, Flow cytometry, IHC, ICC, animal model study
Reactivity:	Mouse
Format:	Liquid
Product Description:	In Vivo Grade Recombinant Anti-mouse PD-L1 Monoclonal Antibody
Isotype:	Rat IgG2b Kappa
Antibody Type:	Recombinant
Purity:	>95% by reducing SDS-PAGE
Endotoxin:	< 1 EU per 1 mg of the protein by the LAL method.
Storage Conditions:	4ºC
Grade:	In vivo
Recommended Usage:	This product is suitable for in vivo animal use. Optimal amounts need to be determined empirically for each experiment.
Hidden Synonyms:	InVivoMab, InVivoPlus, GoInVivo, In Vivo Gold, 10F.9G2.1

BACKGROUND INFORMATION

Programmed death-ligand 1 (PD-L1), also known as CD274 or B7-H1, is a transmembrane protein that plays a pivotal role in immune regulation by modulating T cell activity. PD-L1 is expressed on a wide range of cells, including antigen-presenting cells, epithelial cells, and many tumor cells. Its primary function is to bind to its receptor, programmed cell death protein 1 (PD-1), located on activated T cells. This interaction delivers an inhibitory signal that reduces T cell proliferation, cytokine production, and cytotoxicity, thereby maintaining immune homeostasis and preventing autoimmunity. However, in pathological contexts such as cancer, PD-L1 expression allows tumor cells to evade immune attack, creating an immunosuppressive microenvironment.

Structurally, PD-L1 is a type I transmembrane glycoprotein belonging to the B7 family of immune checkpoint molecules. The extracellular domain comprises two immunoglobulin-like regions—an IgV-like domain responsible for PD-1 binding and an IgC-like domain that stabilizes the molecule. The protein also contains a single transmembrane helix and a short cytoplasmic tail that lacks classical signaling motifs but may interact with intracellular partners influencing its stability and localization. The PD-L1–PD-1 complex adopts a well-characterized interface where the IgV domains of both molecules interact in a way that blocks T cell

receptor-mediated activation signaling.

The main ligands of PD-L1 are PD-1 and CD80 (B7-1). While PD-1 engagement results in T cell inhibition, interaction with CD80 may yield bidirectional signaling effects depending on the cellular context. PD-L1 can be induced by inflammatory cytokines such as interferon-gamma (IFN-γ), linking innate immune responses to immune checkpoint modulation.

PD-L1 plays a major role in numerous diseases. Overexpression of PD-L1 is a hallmark of many cancers, including lung, melanoma, renal, and breast cancers, where it contributes to immune escape. Therapeutically, blocking the PD-1/PD-L1 axis with immune checkpoint inhibitors has revolutionized cancer treatment. Drugs such as pembrolizumab, nivolumab, and atezolizumab disrupt this inhibitory pathway, restoring antitumor T cell function. Moreover, PD-L1 is being explored as both a predictive biomarker for immunotherapy response and a target for novel therapies, including bispecific antibodies and CAR-T cells aimed at enhancing immune-mediated tumor clearance.

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