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Anti-Mouse CD274 (PD-L1) Antibody

Catalog Number:	201301, 201302
Size:	50 μg, 500 μg
Target Name:	CD274, PD-L1, B7-H1
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	10F.9G2
Application:	Flow Cytometry
Reactivity:	Mouse
Format:	Purified
Isotype:	Rat IgG2b
Antibody Type:	Monoclonal
Formulation:	Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide
Protein Concentration:	0.5 mg/mL
Storage and Handling:	The antibody solution should be stored between 2°C and 8°C
Recommended Usage:	For flow cytometric staining, it is recommended to use less than 0.1 µg of this reagent per 0.5-1.0 million cells in a 100 µL volume. Optimal reagent performance should be determined by titration for each specific application
Isotype Control:	300301

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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