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Anti-mouse CD279 (PD-1) Antibody

Catalog Number:	205001, 205002
Size:	100 μg, 500 μg
Target Name:	CD279, Programmed Death-1, PD 1, PDCD1, PD-1
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	29F.1A12
Application:	Flow Cytometry, IHC-F, ELISA, Blocking
Reactivity:	Mouse
Format:	Purified
Isotype:	Rat IgG2a
Antibody Type:	Monoclonal
Formulation:	Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide
Protein Concentration:	0.5 mg/mL
Storage&Handling:	The antibody solution should be stored between 2°C and 8°C
Isotype Controls:	303501
Antibody Family:	Mouse Antibodies
GeneID:	18566
RRID:	AB_3759410

BACKGROUND INFORMATION

Mouse CD279, more commonly known as programmed cell death protein 1 (PD-1), is an inhibitory immune checkpoint receptor expressed primarily on activated T cells, as well as B cells and some myeloid populations. It plays a critical role in maintaining peripheral tolerance and preventing excessive immune activation by downregulating T cell responses during chronic antigen exposure, such as infection or inflammation. PD-1 is rapidly induced following T cell receptor engagement and acts as a key regulator of immune homeostasis.

Structurally, PD-1 is a type I transmembrane protein belonging to the immunoglobulin superfamily. It contains a single extracellular IgV-like domain responsible for ligand binding, a transmembrane region, and a cytoplasmic tail with immunoreceptor tyrosine-based inhibitory (ITIM) and switch (ITSM) motifs. Upon ligand engagement, these motifs recruit phosphatases such as SHP-2, which attenuate proximal T cell receptor signaling pathways.

The primary ligands of PD-1 are PD-L1 (CD274) and PD-L2 (CD273), which are expressed on antigen-presenting cells and various non-hematopoietic tissues. Interaction of PD-1 with its ligands suppresses T cell proliferation, cytokine production, and survival, promoting an exhausted T cell phenotype during chronic immune stimulation.

PD-1 signaling is implicated in chronic infections, cancer, and autoimmune diseases. In tumors, PD-1-mediated inhibition allows cancer cells to evade immune surveillance by suppressing anti-tumor T cell activity.

Therapeutically, blockade of the PD-1/PD-L1 axis using monoclonal antibodies has revolutionized cancer immunotherapy by restoring T cell function. Conversely, enhancing PD-1 signaling may be beneficial in treating autoimmune diseases and preventing transplant rejection, making it a versatile target in immune modulation.

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