

## In Vivo Star Anti-Mouse CD279 (PD1) Antibody

<b>Catalog Number:</b>	506901, 506902, 506903
<b>Size:</b>	1 mg, 5 mg, 25 mg
<b>Target Name:</b>	mouse PD-1, CD279
<b>Regulatory Status:</b>	RUO

### PRODUCT DETAILS

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<b>Clone:</b>	RMP1-14.1-m2a
<b>Application:</b>	ELISA, WB, Flow cytometry, IHC, ICC, animal model study
<b>Reactivity:</b>	Mouse
<b>Format:</b>	Liquid
<b>Product Description:</b>	In Vivo Grade Recombinant Anti-mouse PD-1 Monoclonal Antibody
<b>Isotype:</b>	Mouse IgG2a Kappa
<b>Antibody Type:</b>	Recombinant
<b>Purity:</b>	>95% by reducing SDS-PAGE
<b>Endotoxin:</b>	< 1 EU per 1 mg of the protein by the LAL method.
<b>Storage Conditions:</b>	4°C
<b>Grade:</b>	In vivo
<b>Recommended Usage:</b>	This product is suitable for in vivo animal use. Optimal amounts need to be determined empirically for each experiment.
<b>Hidden Synonyms:</b>	InVivoMab, InVivoPlus, GoInVivo, In Vivo Gold
<b>RRID:</b>	AB_3739343

### BACKGROUND INFORMATION

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CD279, also known as Programmed Cell Death Protein 1 (PD-1), is a crucial immune checkpoint receptor that regulates T cell activation and prevents autoimmunity. This transmembrane protein plays a pivotal role in maintaining immune homeostasis by delivering inhibitory signals that dampen excessive immune responses.

PD-1 is a type I transmembrane glycoprotein belonging to the immunoglobulin superfamily. It contains an extracellular immunoglobulin variable (IgV)-like domain, a transmembrane region, and an intracellular tail with two tyrosine-based signaling motifs: an immunoreceptor tyrosine-based inhibitory motif (ITIM) and an immunoreceptor tyrosine-based switch motif (ITSM). When engaged, these motifs recruit phosphatases that inhibit T-cell receptor signaling, effectively suppressing T-cell activation, proliferation, and cytokine production.

PD-1 interacts with two primary ligands: PD-L1 (B7-H1/CD274) and PD-L2 (B7-DC/CD273). PD-L1 is widely expressed on various cell types, including tumor cells, antigen-presenting cells, and non-hematopoietic tissues, while PD-L2 expression is more restricted to

antigen-presenting cells. These ligand-receptor interactions serve as critical brakes on immune responses. In cancer, tumor cells exploit the PD-1/PD-L1 pathway to evade immune surveillance. By upregulating PD-L1 expression, tumors effectively "turn off" infiltrating T-cells, preventing effective anti-tumor immunity. This mechanism contributes to tumor progression and immune escape across multiple cancer types.

The discovery of PD-1's role in cancer has revolutionized oncology through immune checkpoint inhibitors. Monoclonal antibodies targeting PD-1 (pembrolizumab, nivolumab) or PD-L1 (atezolizumab, durvalumab) block this inhibitory pathway, reinvigorating anti-tumor T-cell responses. These therapies have demonstrated remarkable success in treating melanoma, non-small cell lung cancer, renal cell carcinoma, and numerous other malignancies, fundamentally transforming cancer treatment paradigms and offering durable responses in previously untreatable cancers.

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