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In Vivo Star Anti-Mouse CD279 (PD1) / VEGFR-2 Bispecific Antibody

Catalog Number:	514901, 514902, 514903
Size:	1 mg, 5 mg, 25 mg
Target Name:	PD1, PD-1, PDCD1, CD279, SLEB2, VEGFR2, VEGF-R2, KDR, Tyrosin kinase growth factor receptor, homolo
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	RMP1-14 / DC101
Application:	Functional assay, animal model study
Reactivity:	Mouse
Format:	Liquid
Product Description:	In Vivo Grade Recombinant Anti-mouse PD-1 / VEGFR-2 Bispecific Antibody
Isotype:	Mouse IgG2c LALAPG 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

BACKGROUND INFORMATION

Programmed cell death protein 1 (PD-1) is an immune checkpoint receptor expressed primarily on activated T cells, B cells, and some myeloid cells. It plays a crucial role in regulating immune responses by maintaining peripheral tolerance and preventing autoimmunity. When PD-1 binds to its ligands PD-L1 or PD-L2 on antigen-presenting or tumor cells, it recruits SHP-2 phosphatase, which dephosphorylates key signaling molecules in the T cell receptor (TCR) pathway. This suppresses T cell proliferation, cytokine secretion, and cytotoxic activity. In cancer, chronic PD-1 engagement results in “T cell exhaustion,” allowing tumor cells to evade immune destruction. Therapeutic blockade of PD-1 with monoclonal antibodies such as nivolumab or pembrolizumab reactivates these T cells, enabling potent anti-tumor immune responses.

Vascular Endothelial Growth Factor Receptor 2 (VEGFR2), also known as KDR (kinase insert domain receptor) or Flk-1, is the principal receptor mediating the angiogenic effects of VEGF-A. VEGFR2 is expressed mainly on endothelial cells and functions as a receptor tyrosine kinase. Upon VEGF-A binding, VEGFR2 dimerizes and becomes autophosphorylated, initiating downstream signaling pathways such as MAPK, PI3K-Akt, and PLCγ, which drive endothelial cell proliferation, migration, and new vessel formation. In tumors, overactivation of VEGFR2 results in aberrant angiogenesis, leading to structurally and functionally abnormal

vasculature that not only sustains tumor growth and metastasis but also creates an immunosuppressive microenvironment by limiting immune cell infiltration and promoting hypoxia.

A bispecific antibody targeting PD-1 and VEGFR2 would provide a synergistic approach to enhancing anti-tumor immunity. By simultaneously blocking PD-1, the antibody would reinvigorate exhausted cytotoxic T cells, restoring their ability to recognize and kill tumor cells. Concurrent inhibition of VEGFR2 signaling would normalize the tumor vasculature, reduce hypoxia, and improve the trafficking of activated T cells into the tumor microenvironment. Moreover, suppressing VEGF/VEGFR2-mediated immunosuppression would shift the tumor milieu toward a more pro-inflammatory and immune-permissive state. This dual mechanism could overcome resistance observed with single-agent checkpoint inhibitors or anti-angiogenic therapies alone. Additionally, combining checkpoint modulation and vascular normalization into a single molecule may optimize pharmacokinetics, enhance co-localization within the tumor, and reduce toxicity relative to combination regimens. Thus, a PD-1 × VEGFR2 bispecific antibody represents a promising next-generation immunotherapy platform capable of simultaneously activating immune effector cells and dismantling the physical and molecular barriers that protect tumors from immune attack.

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