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# In Vivo Star Anti-Mouse CD274 (PD-L1) / VEGF-A Bispecific Antibody

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| **Catalog Number:** | 514801, 514802, 514803 |
| **Size:** | 1 mg, 5 mg, 25 mg |
| **Target Name:** | PD-L1, PDL1 CD274, B7-H1, Vascular endothelial growth factor, VEGF-A, VEGFA, VEGF |
| **Regulatory Status:** | RUO |

## PRODUCT DETAILS

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| **Clone:** | 10F.9G2 / G6-31 |
| **Application:** | Functional assay, Neutralization, animal model study |
| **Reactivity:** | Mouse |
| **Format:** | Liquid |
| **Product Description:** | In Vivo Grade Recombinant Anti-mouse PD-L1 / VEGF-A 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 Death-Ligand 1 (PD-L1), also known as CD274, is a transmembrane protein expressed on tumor cells, antigen-presenting cells, and various immune cells within the tumor microenvironment. Its primary function is to bind to the inhibitory receptor PD-1 on activated T cells, leading to suppression of T cell activation, cytokine release, and cytotoxic function. This PD-1/PD-L1 interaction is crucial for maintaining peripheral immune tolerance and preventing autoimmunity. However, tumors exploit this pathway by overexpressing PD-L1 to evade immune detection and destruction. Blocking PD-L1 restores T cell activity and has become the foundation of modern cancer immunotherapy, leading to the development of agents such as atezolizumab, durvalumab, and avelumab, which have transformed treatment for several cancers including lung, bladder, and liver cancer.

Vascular Endothelial Growth Factor A (VEGF-A) is a key regulator of angiogenesis, the process of forming new blood vessels. Under normal conditions, VEGF-A supports tissue growth, repair, and oxygen delivery. In cancer, however, VEGF-A is often overexpressed and drives the formation of abnormal and leaky blood vessels, creating a hypoxic and immunosuppressive tumor microenvironment. This dysfunctional vasculature limits immune cell infiltration, supports tumor survival, and contributes to resistance against immunotherapies. Therefore, targeting VEGF-A not only suppresses tumor angiogenesis but can also normalize

tumor vasculature to improve immune access to the tumor site.

A bispecific antibody targeting PD-L1 and VEGF-A combines two powerful therapeutic mechanisms into a single molecule—immune checkpoint blockade and angiogenesis inhibition. Blocking PD-L1 reactivates exhausted T cells, allowing the immune system to attack cancer cells effectively. Simultaneously, inhibition of VEGF-A reduces vascular abnormality and improves oxygenation, alleviating hypoxia-induced immunosuppression and facilitating T cell infiltration into the tumor. This dual targeting strategy synergistically enhances anti-tumor immunity while potentially reducing treatment resistance seen with single-agent therapy. Furthermore, one bispecific antibody may optimize pharmacokinetics and safety by minimizing overlapping toxicities compared to combination regimens.

Clinically, PD-L1 × VEGF-A bispecific antibodies are being explored in several solid tumors, including non-small cell lung cancer and hepatocellular carcinoma. Early studies have shown that these agents can elicit stronger immune responses and more durable tumor regression than monotherapies. By bridging immune activation with vascular normalization, PD-L1/VEGF-A bispecifics represent a promising next-generation approach to overcome immune resistance and improve outcomes in oncology.

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