www.innocyto.com.cn

InnoCyto Inc.

15375 Barranca Pkwy, Suite I-103
Irvine, CA 92618
Email: info@innocyto.com
Phone: 1-949-418-7543

# In Vivo Star Anti-Mouse VEGF Antibody

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| **Catalog Number:** | 513001, 513002, 513003 |
| **Size:** | 1 mg, 5 mg, 25 mg |
| **Target Name:** | Vascular endothelial growth factor, VEGF-A, VEGFA, VEGF |
| **Regulatory Status:** | RUO |

## PRODUCT DETAILS

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| **Clone:** | G6-23 |
| **Application:** | ELISA, WB, Flow cytometry, IHC, ICC, animal model study |
| **Reactivity:** | Mouse |
| **Format:** | Liquid |
| **Product Description:** | In Vivo Grade Recombinant Anti-mouse VEGF Monoclonal Antibody |
| **Isotype:** | Mouse IgG2a 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

Vascular Endothelial Growth Factor (VEGF) is a potent signaling protein that plays a central role in angiogenesis - the formation of new blood vessels from existing vasculature. It is essential during embryonic development, wound healing, and tissue regeneration, serving as a key regulator of vascular permeability and endothelial cell proliferation. In adults, VEGF facilitates physiological angiogenesis in the ovary, endometrium, and skeletal muscle but also contributes to various pathological processes when dysregulated.

Structurally, VEGF belongs to a larger family of growth factors that includes VEGF-A, VEGF-B, VEGF-C, VEGF-D, and Placental Growth Factor (PlGF). The prototype, VEGF-A, is a homodimeric glycoprotein composed of two approximately 23-kDa subunits linked by disulfide bonds. Alternative splicing of the VEGF-A gene gives rise to multiple isoforms (such as VEGF121, VEGF165, and VEGF189), each differing in heparin-binding affinity and bioavailability. These isoforms allow for fine-tuned regulation of angiogenic signaling within different tissues and physiological contexts.

VEGF family members function by binding to high-affinity tyrosine kinase receptors on endothelial cells: VEGFR-1 (Flt-1), VEGFR-2

(KDR/Flk-1), and VEGFR-3 (Flt-4), in conjunction with co-receptors like neuropilins (NRP-1 and NRP-2). Among these, VEGFR-2 mediates most of the pro-angiogenic effects, triggering intracellular signaling cascades such as the PI3K/Akt and MAPK pathways, which promote endothelial cell survival, proliferation, migration, and vascular permeability.

Dysregulation of VEGF signaling is implicated in numerous diseases. Overexpression of VEGF drives pathological angiogenesis in cancers, diabetic retinopathy, and age-related macular degeneration (AMD). Tumors exploit VEGF signaling to establish a blood supply, providing oxygen and nutrients necessary for growth and metastasis. Conversely, insufficient VEGF expression can lead to impaired wound healing and ischemic diseases due to poor blood vessel development.

Therapeutically, VEGF and its receptors are major targets in modern medicine. Anti-VEGF therapies aim to inhibit excessive angiogenesis and restore vascular balance. Bevacizumab (Avastin), a monoclonal antibody against VEGF-A, was the first anti-angiogenic therapy approved for cancer treatment. Other agents, such as ranibizumab (Lucentis) and aflibercept (Eylea), are widely used in ophthalmology to treat AMD and diabetic macular edema by preventing abnormal vascular leakage and neovascularization in the retina. Additionally, small-molecule inhibitors targeting VEGFR tyrosine kinases, such as sunitinib, sorafenib, and pazopanib, are employed in cancer therapy to block downstream VEGF signaling pathways. Collectively, VEGF-targeted therapies have transformed the treatment landscape of cancer and ocular diseases but continue to be refined to minimize toxicities and resistance mechanisms.

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