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# In Vivo Star Anti-Mouse PLVAP/PV-1 Antibody

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| **Catalog Number:** | 512701, 512702, 512703 |
| **Size:** | 1 mg, 5 mg, 25 mg |
| **Target Name:** | PLVAP, Plasmalemma vesicle-associated protein, PV-1, PV1 FELS |
| **Regulatory Status:** | RUO |

## PRODUCT DETAILS

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| **Clone:** | MECA-32 |
| **Application:** | ELISA, WB, Flow cytometry, IHC, ICC, animal model study |
| **Reactivity:** | Mouse |
| **Format:** | Liquid |
| **Product Description:** | In vivo Grade Recombinant Anti-mouse PLVAP/PV-1 Monoclonal Antibody |
| **Isotype:** | Rat 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 |
| **RRID:** | AB_3739401 |
| **GeneID:** | 84094 |

## BACKGROUND INFORMATION

Plasmalemma Vesicle-Associated Protein (PLVAP), also known as PV-1, is an endothelial-specific membrane glycoprotein that plays a critical role in regulating vascular permeability and the structure of endothelial diaphragms. It was first identified as a major component of the stomatal and fenestral diaphragms, thin filtration-like structures found at the openings of caveolae, fenestrae, and transendothelial channels in continuous and fenestrated capillaries. These structures are important for controlling the exchange of plasma components between the blood and surrounding tissues, thereby maintaining vascular homeostasis.

Structurally, PLVAP is a type II transmembrane protein composed of a short N-terminal cytoplasmic domain, a single transmembrane helix, and a large extracellular C-terminal region. The extracellular domain forms homodimers and undergoes extensive glycosylation, which contributes to the formation of the characteristic diaphragm-like meshwork in endothelial cells. PLVAP is highly expressed in fenestrated and sinusoidal endothelia, especially in organs such as the liver, endocrine glands, and

kidney glomeruli, where rapid molecular exchange is required. In contrast, it is absent or expressed at very low levels in the brain, consistent with the tight endothelial barrier properties of the blood–brain barrier.

PLVAP does not have a single defined ligand in the classical sense but functions as part of a structural complex mediating endothelial barrier selectivity and transcellular transport. Its expression is dynamically regulated by vascular endothelial growth factor (VEGF) and other angiogenic or inflammatory cues. Under pathological conditions, PLVAP expression can be upregulated, leading to increased vascular leakiness and tissue edema. Elevated PLVAP is observed in inflammatory disorders, septic shock, and tumor vasculature, where it contributes to the characteristic leakiness of tumor-associated vessels.

In disease contexts, PLVAP acts as both a biomarker and potential therapeutic target. In cancer, its upregulation marks tumor neovasculature, making it attractive for targeted drug delivery or imaging. Studies suggest that blocking or modulating PLVAP function could reduce pathological vascular permeability without completely disrupting normal endothelial function. Additionally, engineered antibodies or antibody-drug conjugates targeting PLVAP may allow selective delivery of therapeutics to tumor-associated endothelium while sparing healthy vasculature. Beyond oncology, understanding PLVAP biology holds promise for developing therapies to regulate vascular leakage in inflammatory and ischemic diseases, offering opportunities to restore endothelial integrity and improve tissue outcomes.

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