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In Vivo Star Anti-Human CD47 Antibody

Catalog Number:	518401, 518402, 518403
Size:	1 mg, 5 mg, 25 mg
Target Name:	CD47, IAP, neurophilin, gp42, Integrin-associated protein
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	B6H12
Application:	Direct ELISA, functional assay, Flow Cytometry
Reactivity:	Human
Format:	Liquid
Product Description:	In vivo Grade Recombinant Anti-Human CD47 Monoclonal Antibody
Isotype:	Mouse IgG1 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 in in vitro functional assays or in vivo on human cells used in animal models. Optimal amounts need to be determined empirically for each experiment.
Hidden Synonyms:	InVivoMab, InVivoPlus, GoInVivo, In Vivo Gold
RRID:	AB_3739459

BACKGROUND INFORMATION

CD47, also known as integrin-associated protein (IAP), is a widely expressed transmembrane receptor that plays a critical role in regulating immune cell communication, self-recognition, and cellular homeostasis. Functionally, it acts as a key "don’t eat me" signal that protects cells from being phagocytosed by macrophages and dendritic cells. CD47 achieves this by interacting with its primary ligand, Signal Regulatory Protein Alpha (SIRPα), present on the surface of phagocytic cells. This interaction delivers an inhibitory signal that suppresses the engulfment of the cell expressing CD47, thereby distinguishing self from non-self tissues, a mechanism vital for maintaining immune tolerance under normal physiological conditions.

Structurally, CD47 is a member of the immunoglobulin superfamily. It consists of a single N-terminal extracellular immunoglobulin-like domain, five membrane-spanning alpha helices, and a short cytoplasmic tail. The extracellular domain mediates ligand binding, including interactions not only with SIRPα but also with thrombospondin-1 (TSP-1) and integrins. Through its association with integrins, CD47 contributes to key cellular processes such as adhesion, migration, and apoptosis. Additionally,

the TSP-1-CD47 interaction influences vascular tone and angiogenesis by modulating nitric oxide signaling.

CD47 overexpression is a hallmark of many cancers, where tumor cells exploit the CD47–SIRPα axis to evade immune clearance. By presenting high levels of CD47 on their surface, malignant cells effectively mask themselves from macrophages, preventing phagocytosis and contributing to tumor progression and metastasis. Elevated CD47 expression has been observed in hematologic malignancies like acute myeloid leukemia (AML) as well as in solid tumors such as breast, ovarian, and colon cancers. Clinically, high CD47 levels are often correlated with poor prognosis and resistance to immune-mediated therapies.

This understanding of CD47's role in tumor immune evasion has led to the development of targeted therapeutics. CD47-blocking antibodies and decoy receptor fusion proteins (such as TTI-621 and magrolimab) are currently undergoing clinical trials aimed at disrupting the CD47–SIRPα interaction. By doing so, these therapies "unmask" cancer cells, allowing macrophages and other immune cells to recognize and eliminate them. Importantly, combination therapies pairing CD47 inhibitors with antibody-based treatments (such as anti-CD20 in lymphoma or anti-HER2 in breast cancer) have demonstrated synergistic effects, enhancing tumor clearance. As research continues, CD47 blockade stands out as one of the most promising strategies to overcome immune resistance and improve the efficacy of next-generation cancer immunotherapies.