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In Vivo Star Anti-Mouse CD370 (CLEC9A) Antibody

Catalog Number:	511501, 511502, 511503
Size:	1 mg, 5 mg, 25 mg
Target Name:	CD370, C-type lectin family member 9A
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	10B4
Application:	ELISA, WB, Flow cytometry, IHC, ICC, animal model study
Reactivity:	Mouse
Format:	Liquid
Product Description:	In vivo Grade Recombinant Anti-mouse CD370 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_3739389
GeneID:	232414

BACKGROUND INFORMATION

CD370, more commonly known as CLEC9A or DNGR-1 (dendritic cell NK lectin group receptor 1), is a C-type lectin–like receptor selectively expressed on a specialized subset of dendritic cells, particularly conventional type 1 dendritic cells (cDC1). These cells are highly efficient at cross-presenting antigens to CD8+ T cells, placing CD370 at the center of immune responses against viruses, intracellular pathogens, and tumors. Rather than broadly activating immunity, CD370 functions as a sensor of cellular damage and necrosis.

Structurally, CD370 is a type II transmembrane protein belonging to the C-type lectin receptor family. It contains a short N-terminal cytoplasmic tail, a single transmembrane region, and a C-terminal extracellular C-type lectin–like domain. Unlike classical lectins, CD370 does not bind carbohydrates in a calcium-dependent manner. Its cytoplasmic tail contains a conserved hemITAM (hemi–immunoreceptor tyrosine-based activation motif), which enables signaling through spleen tyrosine kinase (Syk) upon ligand

engagement.

The primary ligand for CD370 is filamentous actin (F-actin), which becomes exposed when cells undergo necrosis or mechanical damage. By recognizing exposed F-actin, CD370 allows dendritic cells to selectively detect dead or damaged cells while ignoring apoptotic cells, which maintain membrane integrity. Engagement of CD370 does not strongly activate dendritic cells on its own but instead promotes the routing of captured antigens into cross-presentation pathways, enhancing presentation on MHC class I molecules and subsequent activation of cytotoxic CD8+ T cells.

CD370 plays an important role in disease contexts where cell death and antigen cross-presentation are prominent. In viral infections and cancer, CD370-expressing dendritic cells are crucial for initiating effective CD8+ T cell responses against infected or malignant cells. In autoimmune disease, dysregulated sensing of self-derived antigens from damaged tissue could contribute to pathological T cell activation, although CD370 is generally thought to promote tolerance by limiting inflammatory signaling. In cancer, CD370-positive dendritic cells are often functionally impaired within the tumor microenvironment, reducing anti-tumor immunity.

Therapeutically, CD370 is an attractive target for vaccine and immunotherapy strategies. Antigen delivery systems that target CD370 can efficiently direct antigens to cross-presenting dendritic cells, enhancing CD8+ T cell responses without excessive inflammation. This approach is being explored in cancer vaccines and antiviral immunotherapies. Because of its restricted expression pattern and specialized function, CD370 represents a promising avenue for selectively manipulating cytotoxic immune responses while minimizing off-target immune activation.

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