

In Vivo Star Anti-Mouse CD370 / CD274 (PD-L1) Bispecific Antibody

Catalog Number:	513801, 513802, 513803
Size:	1 mg, 5 mg, 25 mg
Target Name:	mouse CD370, PD-L1 (CD274)
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	10B4 / 10F.9G2.1
Application:	Functional assay, animal model study
Reactivity:	Mouse
Format:	Liquid
Product Description:	In Vivo Grade Recombinant Anti-mouse CD370 / PD-L1 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:	4°C
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_3739414

BACKGROUND INFORMATION

CD370, also known as CLEC9A (C-type lectin domain family 9 member A), is a receptor primarily expressed on a specialized subset of dendritic cells known as CD141⁺ (BDCA3⁺) dendritic cells in humans. These cells play an essential role in the cross-presentation of antigens, a process by which exogenous antigens are presented on MHC class I molecules to activate cytotoxic CD8⁺ T cells. Structurally, CD370 is a type II transmembrane glycoprotein with a single extracellular C-type lectin-like domain and a short cytoplasmic tail containing an immunoreceptor tyrosine-based activation motif (ITAM-like) that transmits activation signals through the SYK kinase pathway. Functionally, it recognizes exposed actin filaments from necrotic or damaged cells, enabling dendritic cells to uptake and process antigens from dying cells, initiating robust T cell-mediated immune responses.

PD-L1 (Programmed Death-Ligand 1, also known as CD274) is an immune checkpoint molecule expressed on various immune and tumor cells. It interacts with its receptor PD-1 on T cells to inhibit T cell activation, proliferation, and cytokine production, thereby maintaining immune tolerance and preventing autoimmunity. However, many tumors exploit this pathway by overexpressing PD-L1

to suppress anti-tumor immunity and evade immune destruction. Hence, the PD-1/PD-L1 axis has become a central target in cancer immunotherapy, with monoclonal antibodies like pembrolizumab and atezolizumab already benefiting patients across multiple cancer types.

Combining CD370 and PD-L1 in a single bispecific antibody offers a synergistic therapeutic strategy that unites immune activation with checkpoint blockade. A bispecific antibody targeting CD370 could specifically target and deliver immune-stimulating signals or antigen payloads directly to cross-presenting dendritic cells, enhancing their ability to prime CD8⁺ T cells. Simultaneously, engagement of PD-L1 blockade would relieve inhibitory signals in the tumor microenvironment, sustaining this T cell activation. Such a dual-target approach could bridge innate and adaptive immunity, reinvigorating existing T cells while recruiting new ones through enhanced antigen presentation. Additionally, the localized targeting of dendritic cells may reduce systemic immune-related toxicities often seen with global checkpoint inhibition. Overall, a CD370 × PD-L1 bispecific antibody could potentiate anti-tumor immunity by turning immunologically “cold” tumors into “hot” ones, maximizing T cell activation and persistence while minimizing off-target effects, representing a promising direction in next-generation cancer immunotherapies.

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