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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 CD152 (CTLA-4) Antibody

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| **Catalog Number:** | 510001, 510002, 510003 |
| **Size:** | 1 mg, 5 mg, 25 mg |
| **Target Name:** | CTLA4, CD152, CELIAC3, GSE, IDDM12 |
| **Regulatory Status:** | RUO |

## PRODUCT DETAILS

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| **Clone:** | 9D9-m2aSL |
| **Application:** | ELISA, WB, Flow cytometry, IHC, ICC, animal model study |
| **Reactivity:** | Mouse |
| **Format:** | Liquid |
| **Product Description:** | In vivo Grade Recombinant Anti-mouse CTLA-4 Monoclonal Antibody |
| **Isotype:** | Mouse IgG2a-L234A L235A P329G (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:** | 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_3739374 |
| **GeneID:** | 12477 |

## BACKGROUND INFORMATION

Cytotoxic T-lymphocyte–associated protein 4 (CTLA-4), also known as CD152, is a critical immune checkpoint receptor that functions as a negative regulator of T cell activation. It is primarily expressed on activated CD4+ and CD8+ T cells and is constitutively expressed at high levels on regulatory T cells (Tregs). CTLA-4 plays a central role in maintaining immune homeostasis by limiting excessive T cell responses and promoting peripheral tolerance.

Structurally, CTLA-4 is a type I transmembrane glycoprotein and a member of the immunoglobulin superfamily. Its extracellular region consists of a single IgV-like domain responsible for ligand binding, followed by a transmembrane region and a short cytoplasmic tail. The cytoplasmic domain lacks intrinsic enzymatic activity but contains conserved signaling motifs, including a tyrosine-based motif that mediates interactions with intracellular signaling and trafficking proteins. CTLA-4 predominantly resides in intracellular vesicles and is rapidly transported to the cell surface following T cell activation.

The primary ligands for CTLA-4 are the B7 family co-stimulatory molecules CD80 (B7-1) and CD86 (B7-2), which are expressed on antigen-presenting cells such as dendritic cells, macrophages, and B cells. CTLA-4 binds CD80 and CD86 with significantly higher affinity and avidity than the activating receptor CD28. By outcompeting CD28 for ligand binding and actively removing CD80/CD86 from the surface of antigen-presenting cells through trans-endocytosis, CTLA-4 effectively dampens co-stimulatory signaling and restrains T cell activation.

Dysregulation of CTLA-4 function is associated with a range of diseases. Genetic deficiency or loss-of-function mutations in CTLA-4 can lead to severe lymphoproliferative disorders, autoimmunity, and immune dysregulation due to uncontrolled T cell activation. Conversely, excessive CTLA-4 activity can contribute to impaired immune responses, including reduced anti-tumor immunity. In cancer, tumor-induced upregulation of CTLA-4 signaling contributes to immune evasion by suppressing effective T cell responses.

CTLA-4 is a landmark target in immunotherapy. Therapeutic antibodies that block CTLA-4, such as immune checkpoint inhibitors, enhance T cell activation and proliferation by restoring co-stimulatory signaling, leading to improved anti-tumor immune responses in several cancers. However, CTLA-4 blockade can also disrupt immune tolerance, resulting in immune-related adverse events. Conversely, strategies that enhance CTLA-4 function or signaling are being explored for the treatment of autoimmune and inflammatory diseases. Together, these approaches highlight CTLA-4’s pivotal role at the intersection of immune regulation, disease, and therapy.

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