

## Technical Data Sheet

### In Vivo Star Anti-Mouse CD152 (CTLA-4) Antibody

**Catalog Number:** 510001, 510002, 510003

**Size:** 1 mg, 5 mg, 25 mg

**Target Name:** mouse CTLA-4, CD152

**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: 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

#### Background Information

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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.