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InnoCyto Inc.

15375 Barranca Pkwy, Suite I-103
Irvine, CA 92618
Email: info@innocyto.com
Phone: 1-949-418-7543

# Biotin Human CTLA4 (CD152) Protein (C-His-Avi)

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| **Catalog Number:** | 807803, 807804 |
| **Size:** | 25 ug, 100 ug |
| **Target Name:** | CTLA4, CD152 |
| **Regulatory Status:** | RUO |

## PRODUCT DETAILS

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| **Application:** | ELISA, BLI |
| **Format:** | Liquid, Biotinylated |
| **Expression Host:** | CHO |
| **Species:** | Human |
| **Sources:** | Recombinant Human CTLA4 Protein (Ala37-Phe162) with C-terminus His-Avi-tag is expressed in CHO cell. This protein was site-specifically labeled with Biotin by BirA ligase. |
| **Accession Number:** | P16410 |
| **Molecular Weight:** | The protein has a predicted molecular weight of 17.1 kDa. Under DTT-reducing conditions, it migrates at approximately 20 kDa on SDS-PAGE. |
| **Affinity Tag:** | C-His-Avi |
| **Purity:** | >95% based on SDS-PAGE under reducing condition |
| **Formulation:** | 1xPBS buffer, pH7.4, 0.22 μm filtered |
| **Endotoxin level:** | Not tested |
| **Protein Concentration:** | 25μg size is bottled at 0.2mg/mL concentration. 100 μg size is supplied at a lot-specific concentration. |
| **Storage and Handling:** | Briefly centrifuge the vial upon receipt. An unopened vial can be stored at 4°C for up to 2 weeks, or at -20°C or below for up to six months. The protein may be further diluted to 0.1 mg/mL using 0.22 μm-filtered PBS buffer (pH 7.4). For long-term storage, the diluted stock solution should be aliquoted and stored at ≤ –70°C to minimize freeze-thaw cycles. If additional dilution is required, carrier proteins such as FBS or BSA should be added to maintain protein stability. |
| **Recommended Usage:** | For detection, use a secondary reagent with this product. |

## 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.

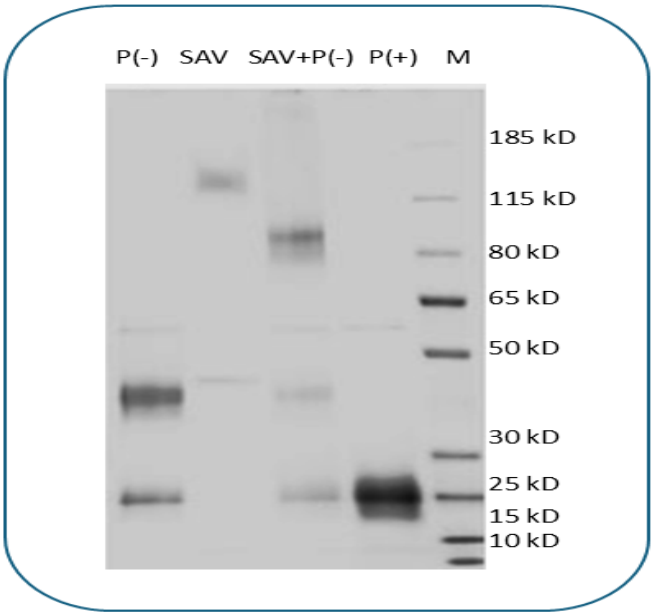
Structurally, CTLA-4 is a type I transmembrane glycoprotein and a member of the immunoglobulin superfamily. Its extracellular region consists of a single IgV-like domain responsible for ligand binding, followed by a transmembrane region and a short cytoplasmic tail. The cytoplasmic domain lacks intrinsic 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.

## PRODUCT DATA




Human CTLA4 Protein (C-His-Avi) was biotinylated in vitro using BirA ligase. SDS-PAGE analysis under reducing (P+) and non-reducing (P−) conditions shows the protein has a purity greater than 95%. A gel shift assay using co-incubation with streptavidin indicates that the biotinylation efficiency of the CTLA4 protein exceeds 90%.

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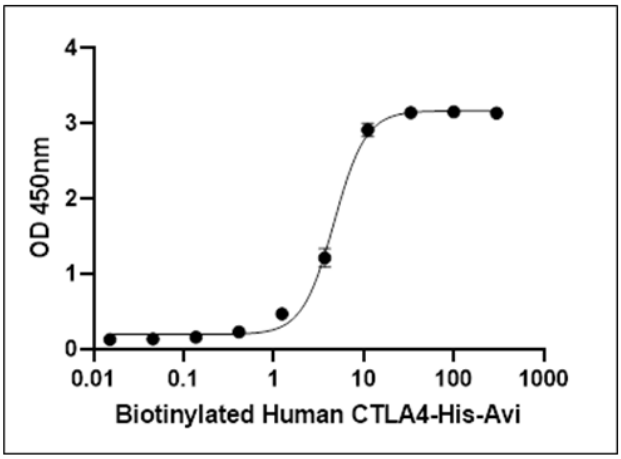


Human B7-1 with Fc tag is coated at 2ug/mL (200ng/well). Biotinylated human CTLA4 (C-His-Avi) can bind B7-1 in dose-dependent manner with the ED50 of 3-10 ng/mL

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