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Human B7-2 (CD86) Protein (C-Fc-Avi)

Catalog Number:	817501, 817502
Size:	25 ug, 100 ug
Target Name:	CD86, B7-2, B70, CD28LG2
Regulatory Status:	RUO

PRODUCT DETAILS

Application:	ELISA, BLI
Format:	Liquid, Purified
Expression Host:	CHO
Species:	Human
Sources:	Recombinant Human B7-2 (CD86) (Leu20-Pro247) with C-terminus Fc-Avi-tag is expressed in CHO cell.
Accession Number:	P05408
Molecular Weight:	The protein has a predicted molecular weight of 54.2 kDa. Under DTT-reducing conditions, it migrates at approximately 75 kDa on SDS-PAGE.
Affinity Tag:	C-Fc-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.

BACKGROUND INFORMATION

CD86, also known as B7-2, is a transmembrane glycoprotein that serves as a crucial costimulatory molecule in the adaptive immune system. CD86 is primarily expressed on antigen-presenting cells (APCs) including dendritic cells, macrophages, B cells, and monocytes, where it plays a vital role in T cell activation and immune response regulation. The protein provides essential costimulatory signals for T cell activation by binding to receptors on T cells, working in conjunction with the primary signal delivered through T cell receptor (TCR) recognition of peptide-MHC complexes. CD86 is a member of the B7 family of costimulatory molecules and functions alongside its related molecule CD80 (B7-1), though CD86 is expressed more rapidly and constitutively on APCs,

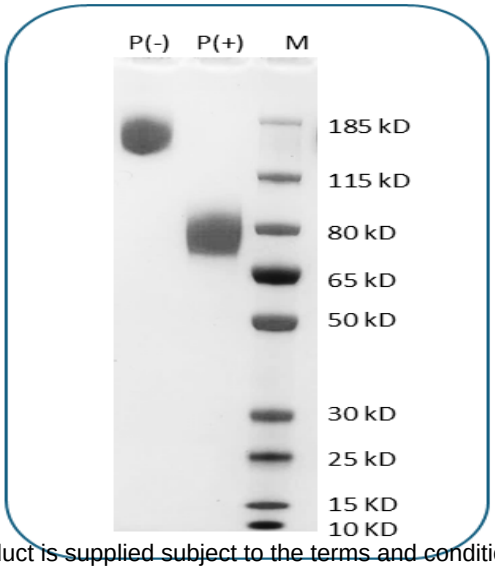
making it particularly important in the early phases of immune responses.

Structurally, CD86 is a type I transmembrane protein of approximately 70-80 kDa belonging to the immunoglobulin superfamily. The extracellular region contains two immunoglobulin-like domains: an N-terminal IgV-like domain that mediates receptor binding and a membrane-proximal IgC-like domain that provides structural support. The protein features a single transmembrane domain and a cytoplasmic tail that may participate in intracellular signaling and regulation of surface expression. The IgV domain contains the binding sites for its cognate receptors, and the overall structure allows CD86 to exist as monomers or dimers on the cell surface. The extracellular domains adopt the characteristic immunoglobulin fold that enables specific recognition by T cell receptors.

CD86 has two primary ligands with opposing immunological functions: CD28 and CTLA-4 (CD152), both expressed on T cells. Binding to CD28, which is constitutively expressed on naive and activated T cells, delivers positive costimulatory signals that promote T cell proliferation, survival, cytokine production (particularly IL-2), and differentiation into effector cells. This CD86-CD28 interaction is critical for initiating productive immune responses. Conversely, binding to CTLA-4, an inhibitory receptor that is upregulated on activated T cells and constitutively expressed on regulatory T cells, delivers negative regulatory signals that suppress T cell activation and maintain immune homeostasis. CTLA-4 binds CD86 with significantly higher affinity than CD28, allowing it to effectively compete for binding and deliver inhibitory signals that prevent excessive immune activation.

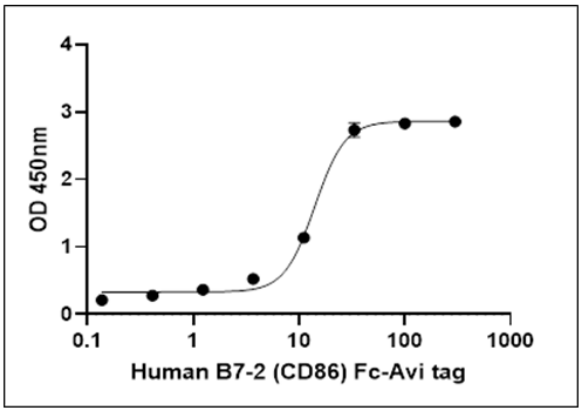
In disease contexts, dysregulation of CD86 expression or function is implicated in various pathological conditions. Aberrant CD86 expression contributes to autoimmune diseases such as rheumatoid arthritis, systemic lupus erythematosus, and multiple sclerosis by promoting inappropriate T cell activation against self-antigens. In transplantation, CD86-mediated costimulation drives allograft rejection. Conversely, tumors may downregulate CD86 on APCs or exploit the CD86-CTLA-4 axis to suppress antitumor immunity. Therapeutically, the CD86-CD28-CTLA-4 pathway has become a major target for immune modulation. CTLA-4 blocking antibodies such as ipilimumab enhance CD28 costimulation by preventing CTLA-4 from binding to CD86/CD80, thereby boosting antitumor T cell responses and achieving clinical success in melanoma and other cancers. Additionally, CTLA-4-Ig fusion proteins like abatacept and belatacept bind to CD86/CD80 to block CD28 costimulation, providing effective immunosuppression for autoimmune diseases and transplant rejection. Selective CD86 targeting strategies, including blocking antibodies and small molecule inhibitors, are being developed to fine-tune immune responses in cancer immunotherapy, autoimmunity, and transplantation, establishing CD86 as a pivotal therapeutic target in modern immunology.

PRODUCT DATA



Human B7-2 (CD86) (C-Fc-Avi) on SDS-PAGE under reducing condition (P+) and non-reducing condition (P-). The gel was stained for 1 hour with BlinkBlue (catalog 700102). The purity of this protein appears to be greater than 95% based on reducing conditions.

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Human CTLA4 (C-His) is coated at 10ug/mL (1ug/well). Human B7-2 (CD86) (C-Fc-Avi) can bind human CTLA-4 in the dose dependent manner with ED50 in the range of 10-30 ng/mL.

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