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# APC Human B7-2 (CD86) Protein (C-His)

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| **Catalog Number:** | 817403, 817404 |
| **Size:** | 25 ug, 100 ug |
| **Target Name:** | CD86, B7-2, B70, CD28LG2 |
| **Regulatory Status:** | RUO |

## PRODUCT DETAILS

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| **Application:** | Flow Cytometry |
| **Format:** | Liquid, APC |
| **Expression Host:** | CHO |
| **Species:** | Human |
| **Sources:** | Recombinant Human B7-2 (CD86) (Leu20-Pro247) with C-terminus His-tag is expressed in CHO cell and conjugated to APC. |
| **Accession Number:** | P05408 |
| **Molecular Weight:** | The protein has a predicted molecular weight of 27.5 kDa. Under DTT-reducing conditions, it migrates at approximately 45 kDa on SDS-PAGE prior to conjugation. |
| **Affinity Tag:** | C-His |
| **Formulation:** | 1xPBS buffer, pH7.4, 0.09% NaN3 with a carrier protein |
| **Endotoxin level:** | Not tested |
| **Protein Concentration:** | 25µg size is bottled at 0.1mg/mL concentration. 100 µg size is bottled at lot specific concentration. |
| **Storage and Handling:** | Briefly centrifuge the vial upon receipt. An unopened vial may be stored at 2–8°C for up to six months. |

## 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.

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