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InnoCyto Inc.

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

APC Human PD-L2 (CD273) Protein (C-Fc)

Catalog Number:	813603, 813604
Size:	25 ug, 100 ug
Target Name:	PDL2, , Butyrophilin B7-DC, CD273, PDCD1 ligand 2
Regulatory Status:	RUO

PRODUCT DETAILS

Application:	Flow Cytometry
Format:	Liquid, APC
Expression Host:	CHO
Species:	Human
Sources:	Recombinant Human PD-L2 (Leu20-Pro219) with C-terminus Fc-tag is expressed in CHO cell and conjugated to APC.
Accession Number:	Q9BQ51
Molecular Weight:	The protein has a predicted molecular weight of 48.8 kDa. Under DTT-reducing conditions, it migrates at approximately 65 kDa on SDS-PAGE prior to conjugation.
Affinity Tag:	C-Fc
Formulation:	1xPBS buffer, pH7.4, 0.09% NaN3 with a carrier protein
Endotoxin level:	Not tested
Protein Concentration:	25μg size is bottled at 0.2 mg/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

CD273, also known as programmed death-ligand 2 (PD-L2) or B7-DC, is a transmembrane protein that functions as an immune checkpoint molecule regulating T cell activation and immune responses. CD273 is a member of the B7 family of costimulatory molecules and is primarily expressed on antigen-presenting cells such as dendritic cells, macrophages, and B cells, with expression upregulated during inflammation and immune activation. The protein binds to programmed cell death protein 1 (PD-1) on T cells, delivering inhibitory signals that suppress T cell proliferation, cytokine production, and effector functions. This interaction plays a crucial role in maintaining immune homeostasis, preventing autoimmunity, and resolving inflammatory responses. PD-L2 plays a pivotal role in negative regulation of the adaptive immune response, and unlike its related molecule PD-L1 (CD274), CD273 expression is more restricted and primarily induced under specific inflammatory conditions.

Structurally, CD273 is a type I transmembrane glycoprotein of approximately 25-30 kDa belonging to the immunoglobulin superfamily. The extracellular region contains two immunoglobulin-like domains: an N-terminal IgV-like domain that mediates PD-1

binding and a membrane-proximal IgC-like domain that provides structural support. The protein also contains a single transmembrane domain and a short cytoplasmic tail. Structural studies have revealed the features that distinguish PD-L2 from PD-L1 in their interactions with PD-1. The IgV domain of CD273 interacts with PD-1 through a binding interface similar to that of PD-L1, though CD273 binds PD-1 with approximately 2-6 fold higher affinity than PD-L1. This higher binding affinity allows CD273 to effectively engage PD-1 and deliver coinhibitory signals that modulate T cell responses.

The primary ligand for CD273 is PD-1 (CD279), an inhibitory receptor expressed on activated T cells, B cells, and myeloid cells. PD-1 has two naturally occurring ligands: PD-L1 and PD-L2 (CD273), both identified over a decade ago. The CD273-PD-1 interaction delivers negative regulatory signals that attenuate immune responses. Additionally, CD273 has been reported to bind to repulsive guidance molecule b (RGMb), though the functional significance of this interaction remains under investigation. The binding of CD273 to PD-1 results in recruitment of phosphatases that inhibit T cell receptor signaling pathways, reducing T cell activation and promoting immune tolerance.

In disease contexts, CD273 plays a significant role in cancer immune evasion and is associated with patient prognosis. As an immune checkpoint molecule, PD-L2 was reported to be associated with patient outcomes and plays a pivotal role in cancer cell immune escape. Many tumors upregulate CD273 expression to suppress antitumor T cell responses, contributing to immune evasion. Elevated CD273 expression has been observed in various cancers, including Hodgkin lymphoma, mediastinal B cell lymphoma, and certain solid tumors, where it correlates with immune suppression in the tumor microenvironment. Therapeutically, the evolving landscape of PD-L2 is bringing new light to checkpoint immunotherapy. While most immune checkpoint inhibitors have focused on the PD-1/PD-L1 axis, the PD-1/PD-L2 (CD273) pathway is also being investigated as a therapeutic target. Some anti-PD-1 antibodies such as nivolumab and pembrolizumab block both PD-L1 and PD-L2 binding to PD-1, contributing to their therapeutic efficacy. Additionally, CD273 is being explored as a biomarker for predicting response to immunotherapy, and selective CD273-targeting strategies are under development to modulate immune responses in cancer and autoimmune diseases while potentially minimizing toxicity compared to broader PD-1/PD-L1 blockade.

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