

DESCRIPTION

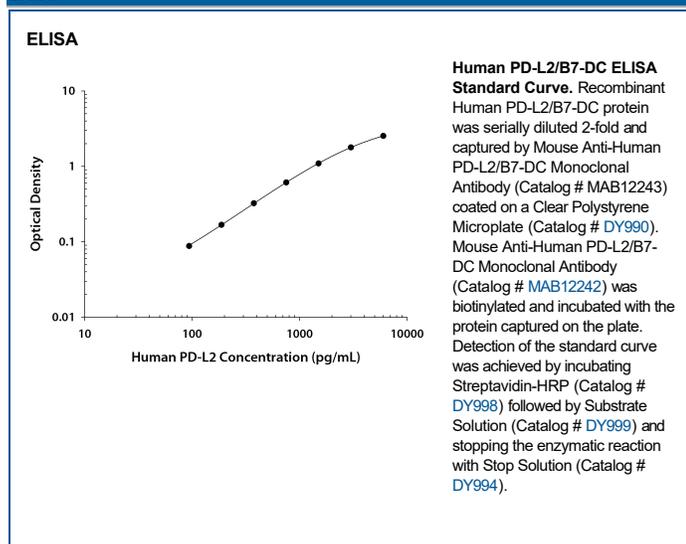
Species Reactivity	Human
Specificity	Detects human PD-L2/B7-DC in direct ELISAs.
Source	Monoclonal Mouse IgG _{2A} Clone # 176610
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human PD-L2/B7-DC Leu20-Pro219 Accession # Q9BQ51
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the [Technical Information](#) section on our website.

ELISA	This antibody functions as an ELISA capture antibody when paired with Mouse Anti-Human PD-L2/B7-DC Monoclonal Antibody (Catalog # MAB12242). <i>This product is intended for assay development on various assay platforms requiring antibody pairs. We recommend the Human PD-L2/B7-DC DuoSet ELISA Kit (Catalog # DY1224) for convenient development of a sandwich ELISA.</i>
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DATA



PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> • 12 months from date of receipt, -20 to -70 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

T cells require a signal induced by the engagement of the T cell receptor and a "co-stimulatory" signal(s) through distinct T cell surface molecules for optimal T cell activation and tolerance. Members of the B7 superfamily of counter-receptors were identified by their ability to interact with co-stimulatory molecules found on the surface of T cells. Members of the B7 superfamily include B7-1 (CD80), B7-2 (CD86), B7-H1 (PD-L1), B7-H2 (B7RP-1), B7-H3, and PD-L2 (B7-DC) (1). B7 proteins are immunoglobulin (Ig) superfamily members with extracellular Ig-V-like and Ig-C-like domains and short cytoplasmic domains. Among the family members, they share from 20-40% amino acid (aa) sequence identity. The cloned human PD-L2 cDNA encodes a 273 aa type I membrane precursor protein with a putative 20 aa signal peptide, a 201 aa extracellular region containing one V-like and one C-like Ig domain, a 24 aa transmembrane region, and a 28 aa cytoplasmic domain. The extracellular domains of mouse and human PD-L2 share approximately 70% aa sequence identity (2). PD-L2 is one of two ligands for programmed death-1 (PD-1), a member of the CD28 family of immuno-receptors. The other identified ligand is PD-L1. Human PD-L1 and PD-L2 share approximately 41% aa sequence identity and have similar functions. PD-L2 is broadly expressed in tissues. Highest expression was detected by Northern blot analysis in heart, placenta, liver, pancreas, spleen, and lymph node. Lower amounts of expression were observed in lung, smooth muscle, and thymus. Expression of PD-L2 on antigen presenting cell has been examined in detail. Resting B cells, monocytes and dendritic cells do not express PD-L2, expression however can be induced by LPS or BCR activation in B cells, INF- γ treatment in monocytes, or LPS plus INF- γ treatment of dendritic cells. PD-L2 expression is also up regulated in a variety of tumor cell lines. On previously activated T cells, PD-L2 interaction with PD-1 inhibits TCR-mediated proliferation and cytokine production, suggesting an inhibitory role in regulating immune responses. In contrast, a co-stimulatory function for the PD-L2 on resting T cells activated with sub-optimal TCR signals has also been reported (3).

References:

1. Coyle, A.J. and J-C. Gutierrez-Ramos (2001) *Nature Immunol.* **2**:203.
2. Latchman Y. *et al.* (2001) *Nature Immun.* **2**:261.
3. Carreno, B.M. and M. Collins (2002) *Annu. Rev. Immunol.* **20**:29.