Anti-PD-L1 [29E.2A3]

Catalogue number: 153514 Sub-type: Primary antibody Images:

Contributor

Inventor: Institute: Clonegene LLC Images:

Tool details

*FOR RESEARCH USE ONLY

Name: Anti-PD-L1 [29E.2A3]

ols.org Alternate name: Programmed cell death 1 ligand 1, PDCD1 ligand 1, Programmed death ligand 1, B7 homolog 1, B7-H1, CD274 21

Class: Monoclonal

Conjugate: Unconjugated

Description: Programmed death-ligand 1 (PD-L1) also known as cluster of differentiation 274 (CD274) or B7 homolog 1 (B7-H1) is a protein that in humans is encoded by the CD274 gene. PD-L1 is a 40kDa type 1 transmembrane protein that has been speculated to play a major role in suppressing the immune system during particular events such as pregnancy, tissue allografts, autoimmune disease and other disease states such as hepatitis. Normally the immune system reacts to foreign antigens where there is some accumulation in the lymph nodes or spleen which triggers a proliferation of antigenspecific CD8+ T cell. The formation of PD-1 receptor / PD-L1 or B7.1 receptor /PD-L1 ligand complex transmits an inhibitory signal which reduces the proliferation of these CD8+ T cells at the lymph nodes and supplementary to that PD-1 is also able to control the accumulation of foreign antigen specific T cells in the lymph nodes through apoptosis which is further mediated by a lower regulation of the gene Bcl-2. PD-L1 binds to its receptor, PD-1, found on activated T cells, B cells, and myeloid cells, to modulate activation or inhibition. Engagement of PD-L1 with its receptor PD-1 on T cells delivers a signal that inhibits TCR-mediated activation of IL-2 production and T cell proliferation. The mechanism involves inhibition of ZAP70 phosphorylation and its association with CD3Ä?Â??. PD-1 signaling attenuates PKC-Ä?Â???Â, activation loop phosphorylation (resulting from TCR signaling), necessary for the activation of transcription factors NF-Ä?Â??B and AP-1, and for production of IL-2. PD-L1 binding to PD-1 also contributes to ligand-induced TCR down-modulation during antigen presentation to naive T cells, by inducing the up-regulation of the E3 ubiquitin ligase CBL-b. Upon IFN-Ä?Â?? stimulation, PD-L1 is expressed on T cells, NK cells, macrophages, myeloid DCs, B cells, epithelial cells, and vascular endothelial cells. The PD-L1 gene promoter region has a response element to IRF-

1, the interferon regulatory factor. Type I interferons can also upregulate PD-L1 on murine hepatocytes, monocytes, DCs, and tumor cells. PD-L1 is notably expressed on macrophages. In the mouse, it has been shown that classically activated macrophages (induced by type I helper T cells or a combination of LPS and interferon-gamma) greatly upregulate PD-L1. Alternatively, macrophages activated by IL-4 (alternative macrophages), slightly upregulate PD-L1, while greatly upregulating PD-L2. It has been shown by STAT1-deficient knock-out mice that STAT1 is mostly responsible for upregulation of PD-L1 on macrophages by LPS or interferon-gamma, but is not at all responsible for its constitutive expression before activation in these mice. It appears that upregulation of PD-L1 may allow cancers to evade the host immune system. An analysis of 196 tumor specimens from patients with renal cell carcinoma found that high tumor expression of PD-L1 inhibitors are in development as immuno-oncology therapies and are showing good results in clinical trials.

Purpose: Parental cell: Organism: Tissue: Model: Gender: Isotype: IgG2b Reactivity: Human Selectivity: Host: Mouse Immunogen: PD-L1 Immunogen UNIPROT ID: Sequence: Growth properties: Production details: Formulation: Recommended controls: Bacterial resistance: Selectable markers: Additional notes:

Target details

Target: PD-L1

Target alternate names:

Target background: Programmed death-ligand 1 (PD-L1) also known as cluster of differentiation 274 (CD274) or B7 homolog 1 (B7-H1) is a protein that in humans is encoded by the CD274 gene. PD-L1 is a 40kDa type 1 transmembrane protein that has been speculated to play a major role in suppressing the immune system during particular events such as pregnancy, tissue allografts, autoimmune disease and other disease states such as hepatitis. Normally the immune system reacts to foreign antigens where there is some accumulation in the lymph nodes or spleen which triggers a proliferation of antigen-specific CD8+ T cell. The formation of PD-1 receptor / PD-L1 or B7.1 receptor /PD-L1 ligand

complex transmits an inhibitory signal which reduces the proliferation of these CD8+ T cells at the lymph nodes and supplementary to that PD-1 is also able to control the accumulation of foreign antigen specific T cells in the lymph nodes through apoptosis which is further mediated by a lower regulation of the gene Bcl-2. PD-L1 binds to its receptor, PD-1, found on activated T cells, B cells, and myeloid cells, to modulate activation or inhibition. Engagement of PD-L1 with its receptor PD-1 on T cells delivers a signal that inhibits TCR-mediated activation of IL-2 production and T cell proliferation. The mechanism involves inhibition of ZAP70 phosphorylation and its association with CD3. PD-1 signaling attenuates PKC- activation loop phosphorylation (resulting from TCR signaling), necessary for the activation of transcription factors NF-B and AP-1, and for production of IL-2. PD-L1 binding to PD-1 also contributes to ligand-induced TCR down-modulation during antigen presentation to naive T cells, by inducing the up-regulation of the E3 ubiquitin ligase CBL-b. Upon IFN-? stimulation, PD-L1 is expressed on T cells, NK cells, macrophages, myeloid DCs, B cells, epithelial cells, and vascular endothelial cells. The PD-L1 gene promoter region has a response element to IRF-1, the interferon regulatory factor. Type I interferons can also upregulate PD-L1 on murine hepatocytes, monocytes, DCs, and tumor cells. PD-L1 is notably expressed on macrophages. In the mouse, it has been shown that classically activated macrophages (induced by type I helper T cells or a combination of LPS and interferon-gamma) greatly upregulate PD-L1. Alternatively, macrophages activated by IL-4 (alternative macrophages), slightly upregulate PD-L1, while greatly upregulating PD-L2. It has been shown by STAT1-deficient knock-out mice that STAT1 is mostly responsible for upregulation of PD-L1 on macrophages by LPS or interferongamma, but is not at all responsible for its constitutive expression before activation in these mice. It appears that upregulation of PD-L1 may allow cancers to evade the host immune system. An analysis of 196 tumor specimens from patients with renal cell carcinoma found that high tumor expression of PD-L1 was associated with increased tumor aggressiveness and a 4.5-fold increased risk of death. Many PD-L1 inhibitors are in development as immuno-oncology therapies and are showing good results in clinical trials.

Molecular weight:

Ic50:

Applications

Application: FACS ; IHC ; Fn **Application notes:**

Handling

Format: Liquid Concentration: Passage number: Growth medium: Temperature: Atmosphere: Volume: Storage medium: Storage buffer: Storage conditions: Shipping conditions: Shipping at 4° C

Related tools

Related tools:

References

References:

