

Anti-CTLA-4 Antibody [PS01-34] - BSA and Azide free

HA750581



Product Type:	Recombinant Rabbit monoclonal IgG, primary antibodies
Species reactivity:	Human, Mouse
Applications:	IHC-P
Molecular Wt:	Predicted band size: 25 kDa
Clone number:	PS01-34

Description: CTLA-4 or CTLA4 (cytotoxic T-lymphocyte-associated protein 4), also known as CD152 (cluster of differentiation 152), is a protein receptor that functions as an immune checkpoint and downregulates immune responses. CTLA-4 is constitutively expressed in regulatory T cells but only upregulated in conventional T cells after activation – a phenomenon which is particularly notable in cancers. It acts as an "off" switch when bound to CD80 or CD86 on the surface of antigen-presenting cells. Variants in this gene have been associated with Type 1 diabetes, Graves' disease, Hashimoto's thyroiditis, celiac disease, systemic lupus erythematosus, thyroid-associated orbitopathy, primary biliary cirrhosis and other autoimmune diseases. Polymorphisms of the CTLA-4 gene are associated with autoimmune diseases such as rheumatoid arthritis, autoimmune thyroid disease and multiple sclerosis, though this association is often weak. In systemic lupus erythematosus (SLE), the splice variant sCTLA-4 is found to be aberrantly produced and found in the serum of patients with active SLE. Germline haploinsufficiency of CTLA-4 leads to CTLA-4 deficiency or CHAI disease (CTLA4 haploinsufficiency with autoimmune infiltration), a rare genetic disorder of the immune system. Symptomatic patients with CTLA-4 mutations are characterized by an immune dysregulation syndrome including extensive T cell infiltration in a number of organs, including the gut, lungs, bone marrow, central nervous system. Once a diagnosis is made, the treatment is based on an individual's clinical condition and may include standard management for autoimmunity and immunoglobulin deficiencies. The comparatively higher binding affinity of CTLA-4 than CD28 has made it a potential therapy for autoimmune diseases.

Immunogen:	Synthetic peptide within Human CTLA4 aa 150 to the C-terminus.
Positive control:	Human tonsils tissue, human lymph nodes tissue.
Subcellular location:	Cell membrane.
Database links:	SwissProt: P16410 Human P09793 Mouse
Recommended Dilutions:	
IHC-P	1:200-1:1,000
Storage Buffer:	1*PBS (pH7.4).
Storage Instruction:	Store at +4°C after thawing. Aliquot store at -20°C. Avoid repeated freeze / thaw cycles.
Purity:	Protein A affinity purified.

Hangzhou Huaan Biotechnology Co., Ltd.

Orders:0086-571-88062880

Technical:0086-571-89986345

Service mail:support@huabio.cn

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Images

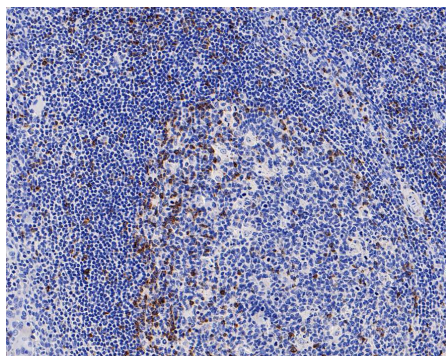


Fig1: Immunohistochemical analysis of paraffin-embedded human tonsils tissue with Rabbit anti-CTLA-4 antibody (HA750581) at 1/1,000 dilution.

The section was pre-treated using heat mediated antigen retrieval with Tris-EDTA buffer (pH 9.0) for 20 minutes. The tissues were blocked in 1% BSA for 20 minutes at room temperature, washed with ddH₂O and PBS, and then probed with the primary antibody (HA750581) at 1/1,000 dilution for 1 hour at room temperature. The detection was performed using an HRP conjugated compact polymer system. DAB was used as the chromogen. Tissues were counterstained with hematoxylin and mounted with DPX.

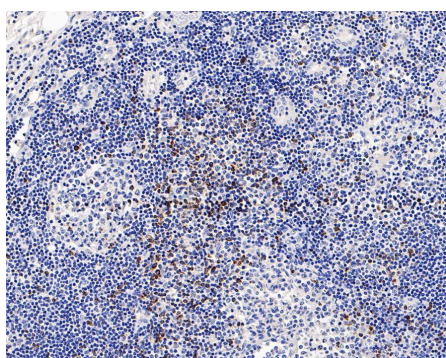


Fig2: Immunohistochemical analysis of paraffin-embedded human lymph nodes tissue with Rabbit anti-CTLA-4 antibody (HA750581) at 1/1,000 dilution.

The section was pre-treated using heat mediated antigen retrieval with Tris-EDTA buffer (pH 9.0) for 20 minutes. The tissues were blocked in 1% BSA for 20 minutes at room temperature, washed with ddH₂O and PBS, and then probed with the primary antibody (HA750581) at 1/1,000 dilution for 1 hour at room temperature. The detection was performed using an HRP conjugated compact polymer system. DAB was used as the chromogen. Tissues were counterstained with hematoxylin and mounted with DPX.

Note: All products are "FOR RESEARCH USE ONLY AND ARE NOT INTENDED FOR DIAGNOSTIC OR THERAPEUTIC USE".

Background References

1. Westra HJ, Martínez-Bonet M, Onengut-Gumuscu S, Lee A, Luo Y, Teslovich N, et al. (October 2018). "Fine-mapping and functional studies highlight potential causal variants for rheumatoid arthritis and type 1 diabetes". *Nature Genetics*. 50 (10): 1366–1374.
2. Schubert D, Bode C, Kenefeck R, Hou TZ, Wing JB, Kennedy A, et al. (December 2014). "Autosomal dominant immune dysregulation syndrome in humans with CTLA4 mutations". *Nature Medicine*. 20 (12): 1410–1416.

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