SANTA CRUZ BIOTECHNOLOGY, INC.

CTLA-4 (C-19): sc-1628

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

T cell proliferation and lymphokine production are triggered by occupation of the TCR by antigen, followed by a costimulatory signal that is delivered by a ligand expressed on antigen presenting cells. The B7-related cell surface proteins CD80 (B7-1) and CD86 (B7-2) are expressed on antigen presenting cells, bind the homologous T cell receptors CD28 and CTLA-4 (cytotoxic T lymphocyte-associated protein-4) and trigger costimulatory signals for optimal T cell activation. CTLA-4 shares 31% overall amino acid identity with CD28 and it has been proposed that CD28 and CTLA-4 are functionally redundant. SLAM is a novel receptor on T cells that, when engaged, potentiates T cell expansion in a CD28-independent manner. B7, also designated BB1, is another ligand or counterreceptor for CD28 and CTLA-4 that is expressed on the antigen-presenting cell.

CHROMOSOMAL LOCATION

Genetic locus: CTLA4 (human) mapping to 2q33.2; Ctla4 (mouse) mapping to 1 C2.

SOURCE

CTLA-4 (C-19) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the C-terminus of CTLA-4 of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-1628 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

CTLA-4 (C-19) is recommended for detection of CTLA-4 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

CTLA-4 (C-19) is also recommended for detection of CTLA-4 in additional species, including equine, canine, bovine and porcine.

Suitable for use as control antibody for CTLA-4 siRNA (h): sc-42766, CTLA-4 siRNA (m): sc-42767, CTLA-4 shRNA Plasmid (h): sc-42766-SH, CTLA-4 shRNA Plasmid (m): sc-42767-SH, CTLA-4 shRNA (h) Lentiviral Particles: sc-42766-V and CTLA-4 shRNA (m) Lentiviral Particles: sc-42767-V.

Molecular Weight of CTLA-4 cytosolic and membrane form: 34/30 kDa.

Molecular Weight of glycosulated CTLA-4: 41-43 kDa.

Positive Controls: CTLA-4 (m) 293T Lysate: sc-119504 or Jurkat whole cell lysate: sc-2204.

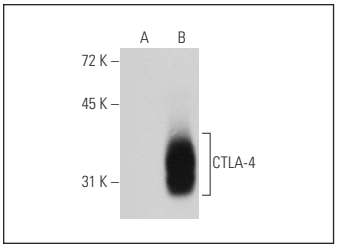
STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

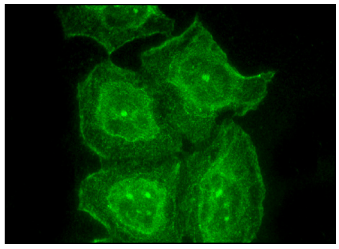
RESEARCH USE

For research use only, not for use in diagnostic procedures.

DATA



CTLA-4 (C-19): sc-1628. Western blot analysis of CTLA-4 expression in non-transfected: sc-117752 (**A**) and mouse CTLA-4 transfected: sc-119504 (**B**) 293T whole cell lysates.



CTLA-4 (C-19): sc-1628. Immunofluorescence staining of methanol-fixed HeLa cells showing membrane localization.

SELECT PRODUCT CITATIONS

1. Metz, D.P., et al. 1998. Differential role of CTLA-4 in regulation of resting memory versus naive CD4 T cell activation. J. Immunol. 161: 5855-5861.
2. Masteller, E.L., et al. 2000. Structural analysis of CTLA-4 function *in vivo*. J. Immunol. 164: 5319-5327.
3. Egen, J.G., et al. 2002. Cytotoxic T lymphocyte antigen-4 accumulation in the immunological synapse is regulated by TCR signal strength. Immunity 16: 23-35.
4. Vijayakrishnan, L., et al. 2004. An autoimmune disease-associated CTLA-4 splice variant lacking the B7 binding domain signals negatively in T cells. Immunity 20: 563-575.
5. Strauss, L., et al. 2005. Dual role of VEGF family members in the pathogenesis of head and neck cancer (HNSCC): possible link between angiogenesis and immune tolerance. Med. Sci. Monit. 11: BR280-BR292.
6. Araki, M., et al. 2009. Genetic evidence that the differential expression of the ligand-independent isoform of CTLA-4 is the molecular basis of the Idd5.1 type 1 diabetes region in nonobese diabetic mice. J. Immunol. 183: 5146-5157.
7. Allen, C.E., et al. 2010. Cell-specific gene expression in Langerhans cell histiocytosis lesions reveals a distinct profile compared with epidermal Langerhans cells. J. Immunol. 184: 4557-4567.
8. Liu, K.K., et al. 2014. Ginsenoside compound K suppresses the abnormal activation of T lymphocytes in mice with collagen-induced arthritis. Acta Pharmacol. Sin. 35: 599-612.