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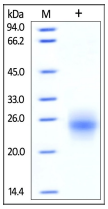
CTLA 4 Recombinant Protein

Cat. No.: 96-220



CTLA 4 Recombinant Protein

Specifications

SPECIES:	Human
SOURCE SPECIES:	HEK293 cells
SEQUENCE:	Ala 37 - Phe 162
FUSION TAG:	C-Twin Strep Tag
TESTED APPLICATIONS:	WB
APPLICATIONS:	This recombinant protein can be used for WB. For research use only.
PREDICTED MOLECULAR WEIGHT:	16.5 kDa

Properties

PURITY:	>95% as determined by SDS-PAGE.
PHYSICAL STATE:	Lyophilized
BUFFER:	PBS, pH7.4
STORAGE CONDITIONS:	Lyophilized Protein should be stored at -20˚C or lower for long term storage. Upon reconstitution, working aliquots should be stored at -20˚C or -70˚C. Avoid repeated freeze-thaw cycles.

Additional Info

OFFICIAL SYMBOL:	CTLA4
ALTERNATE NAMES:	CTLA4, CD152, CELIAC3, GRD4, GSE, ICOS, IDDM12
ACCESSION NO.:	NP_005205.2
GENE ID:	1493

Background and References

BACKGROUND:	CTLA-4 (Cytotoxic T-Lymphocyte Antigen 4) is also known as CD152 (Cluster of differentiation 152), is a protein receptor that downregulates the immune system. CTLA4 is a member of the immunoglobulin superfamily, which is expressed on the surface of Helper T cells and transmits an inhibitory signal to T cells. The protein contains an extracellular V domain, a transmembrane domain, and a cytoplasmic tail. Alternate splice variants, encoding different isoforms. CTLA4 is similar to the T-cell co-stimulatory protein, CD28, and both molecules bind to CD80 and CD86, also called B7-1 and B7-2 respectively, on antigen-presenting cells. CTLA4 transmits an inhibitory signal to T cells, whereas CD28 transmits a stimulatory signal. Intracellular CTLA4 is also found in regulatory T cells and may be important to their function. T cell activation through the T cell receptor and CD28 leads to increased expression of CTLA-4, an inhibitory receptor for B7 molecules. Fusion proteins of CTLA4 and antibodies (CTLA4-Ig) have been used in clinical trials for rheumatoid arthritis.
REFERENCES:	1) Waterhouse P, et al., 1995, Science 270 (5238): 985–8.
	2) Magistrelli G, et al., 1999. Eur. J. Immunol. 29 (11): 3596–602.
	3) Rudd, CE. et al., 2009, Immunol. Rev. 229 (1): 12-26.