

Product datasheet for TP720466

AIM (CD5L) (NM 005894) Human Recombinant Protein

Product data:

Product Type:

or AA Sequence:

Predicted MW:

Concentration:

Description:

Species:

Tag:

Purity:

Buffer:

Endotoxin:

Recombinant Proteins Recombinant protein of human CD5 molecule-like (CD5L) Human **HEK293 Expression Host:** Expression cDNA Clone Ser20-Gly347 C-His 37.1 kDa lot specific >95% as determined by SDS-PAGE and Coomassie blue staining Provided lyophilized from a 0.2 µm filtered solution of 20 mM Tris-HCl, 150 mM NaCl < 0.1 EU per µg protein as determined by LAL test **Reconstitution Method:** Always centrifuge tubes before opening. Do not mix by vortex or pipetting. Dissolve the lyophilized protein in ddH2O. It is not recommended to reconstitute a concentration less than 100 µg/ml. Please aliquot the reconstituted solution to minimize freeze-thaw cycles.

Store at -80°C. Storage: Stability: Stable for at least 6 months from date of receipt under proper storage and handling conditions. NP 005885

RefSeq: Locus ID: 922 **UniProt ID:** 043866 Cytogenetics: 1q23.1 Synonyms: AIM; API6; CT-2; hAIM; PRO229; SP-ALPHA; Spalpha

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STREAM AIM (CD5L) (NM_005894) Human Recombinant Protein – TP720466

Secreted protein that acts as a key regulator of lipid synthesis: mainly expressed by Summary: macrophages in lymphoid and inflamed tissues and regulates mechanisms in inflammatory responses, such as infection or atherosclerosis. Able to inhibit lipid droplet size in adipocytes. Following incorporation into mature adipocytes via CD36-mediated endocytosis, associates with cytosolic FASN, inhibiting fatty acid synthase activity and leading to lipolysis, the degradation of triacylglycerols into glycerol and free fatty acids (FFA). CD5L-induced lipolysis occurs with progression of obesity: participates in obesity-associated inflammation following recruitment of inflammatory macrophages into adipose tissues, a cause of insulin resistance and obesity-related metabolic disease. Regulation of intracellular lipids mediated by CD5L has a direct effect on transcription regulation mediated by nuclear receptors ROR-gamma (RORC). Acts as a key regulator of metabolic switch in T-helper Th17 cells. Regulates the expression of pro-inflammatory genes in Th17 cells by altering the lipid content and limiting synthesis of cholesterol ligand of RORC, the master transcription factor of Th17-cell differentiation. CD5L is mainly present in non-pathogenic Th17 cells, where it decreases the content of polyunsaturated fatty acyls (PUFA), affecting two metabolic proteins MSMO1 and CYP51A1, which synthesize ligands of RORC, limiting RORC activity and expression of pro-inflammatory genes. Participates in obesity-associated autoimmunity via its association with IgM, interfering with the binding of IgM to Fcalpha/mu receptor and enhancing the development of long-lived plasma cells that produce high-affinity IgG autoantibodies (By similarity). Also acts as an inhibitor of apoptosis in macrophages: promotes macrophage survival from the apoptotic effects of oxidized lipids in case of atherosclerosis (PubMed:24295828). Involved in early response to microbial infection against various pathogens by acting as a pattern recognition receptor and by promoting autophagy (PubMed:16030018, PubMed:24223991, PubMed:24583716, PubMed:25713983).[UniProtKB/Swiss-Prot Function]

Protein Families:

Druggable Genome, Secreted Protein

Product images:

95-65⁻ 45-30-20-14-

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