10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

CD5L

Catalog # PVGS1874

Product Information

Primary Accession	Q9QWK4
Species	Mouse
Sequence	Glu22-Val352
Purity	> 95% as determined by Bis-Tris PAGE > 95% as determined by HPLC
Endotoxin Level	Less than 1EU per μg by the LAL method.
Expression System	HEK293
Theoretical Molecular Weight	37.7 kD
Formulation	Lyophilized from a 0.22 μm filtered solution in PBS，(pH 7.4).
Reconstitution	Centrifuge the tube before opening. Reconstituting to a concentration more than 100 μg/ml is recommended. Dissolve the lyophilized protein in distilled water.
Storage & Stability	Upon receiving, the product remains stable up to 6 months at -20 °C or below. Upon reconstitution, the product should be stable for 3 months at -80 °C. Avoid repeated freeze-thaw cycles.

Additional Information

Gene ID	11801
Other Names	CD5 antigen-like, Apoptosis inhibitor expressed by macrophages, mAIM, Apoptosis inhibitory 6, SP-alpha, Cd5l, Aim {ECO:0000303|PubMed:9892623}, Api6
Target Background	CD5L, a soluble protein belonging to the SRCR superfamily, is expressed mostly by macrophages in lymphoid and inflamed tissues. The expression of this protein is transcriptionally controlled by LXRs, members of the nuclear receptor family that play major roles in lipid homeostasis. Research undertaken over the last decade has uncovered critical roles of CD5L as a PRR of bacterial and fungal components and in the control of key mechanisms in inflammatory responses, with involvement in processes, such as infection, atherosclerosis, and cancer.

Protein Information

Name	Cd5l
Synonyms	Aim {ECO:0000303|PubMed:9892623}, Api6
Function	Secreted protein that acts as a key regulator of lipid synthesis: mainly expressed by macrophages in lymphoid and inflamed tissues and regulates mechanisms in inflammatory responses, such as infection or atherosclerosis (PubMed:26048980). Able to inhibit lipid droplet size in adipocytes (PubMed:20519120, PubMed:22579686). 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) (PubMed:20519120). 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 (PubMed:21730133). Regulation of intracellular lipids mediated by CD5L has a direct effect on transcription regulation mediated by nuclear receptors ROR-gamma (RORC) (PubMed:22579686, PubMed:26607793). Acts as a key regulator of metabolic switch in T-helper Th17 cells (PubMed:26607793, PubMed:26607794). 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 (PubMed:26607793). 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 (PubMed:26607793). 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 (PubMed:23562157). Also acts as an inhibitor of apoptosis in macrophages: promotes macrophage survival from the apoptotic effects of oxidized lipids in case of atherosclerosis (PubMed:16054063, PubMed:9892623). Involved in early response to microbial infection against various pathogens by acting as a pattern recognition receptor and by promoting autophagy (By similarity). Promotes recovery from acute kidney injury (AKI) by accumulating on necrotic cell debris within the kidney proximal tubules following AKI and interacting with HAVCR1 expressed on the surface of injured kidney tubular epithelial cells (PubMed:26726878). This enhances HAVCR1-mediated phagocytosis of intraluminal necrotic debris and contributes to kidney tissue repair (PubMed:26726878). Also enhances necrotic debris uptake by kidney macrophages in a HAVCR1- independent manner (PubMed:26726878).
Cellular Location	Secreted. Cytoplasm Note=Secreted by macrophages and circulates in the blood (PubMed:20519120). Transported into the cytoplasm via CD36-mediated endocytosis (PubMed:20519120). Following acute kidney injury, dissociates from IgM in the blood and is excreted into urine, leading to its accumulation at intraluminal debris in the kidney proximal tubules (PubMed:26726878).
Tissue Location	Specifically expressed in tissue macrophages from where it is secreted (PubMed:9892623). Expressed in thymus, liver, spleen and lymph nodes (PubMed:10651944). Present in Th17 cells; mainly present in non-pathogenic Th17 cells (PubMed:26607793). Accumulates on necrotic cell debris within the kidney proximal tubules following acute kidney injury (PubMed:26726878).