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CD5L Blocking Peptide (N-term)
Synthetic peptide
Catalog # BP19894a

Specification

CD5L Blocking Peptide (N-term) - Product Information

Primary Accession	O43866
Other Accession	NP_005885.1

CD5L Blocking Peptide (N-term) - Additional Information

Gene ID 922

Other Names
CD5 antigen-like, CT-2, IgM-associated peptide, SP-alpha, CD5L, API6

Target/Specificity
The synthetic peptide sequence is selected from aa 79-93 of HUMAN CD5L

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

CD5L Blocking Peptide (N-term) - Protein Information

Name CD5L

Synonyms 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. 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).

Cellular Location
Secreted. Cytoplasm {ECO:0000250|UniProtKB:Q9QWK4} Note=Secreted by macrophages and circulates in the blood (PubMed:24223991, PubMed:24804991). Transported in the cytoplasm via CD36-mediated endocytosis (By similarity) {ECO:0000250|UniProtKB:Q9QWK4, ECO:0000269|PubMed:24223991, ECO:0000269|PubMed:24804991}

Tissue Location
Expressed in spleen, lymph node, thymus, bone marrow, and fetal liver, but not in non-lymphoid tissues

CD5L Blocking Peptide (N-term) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides

CD5L Blocking Peptide (N-term) - Images

CD5L Blocking Peptide (N-term) - Background

CD5L may play a role in the regulation of the immune system. Seems to play a role as an inhibitor of apoptosis.

CD5L Blocking Peptide (N-term) - References

Rose, J.E., et al. Mol. Med. 16 (7-8), 247-253 (2010) :
Davila, S., et al. Genes Immun. 11(3):232-238(2010)
Chapuis, J., et al. Mol. Psychiatry 14(11):1004-1016(2009)
Qu, P., et al. J. Immunol. 182(3):1648-1659(2009)
Kim, W.K., et al. Exp. Mol. Med. 40(6):677-685(2008)