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AIM / CD5L Antibody (N-Terminus)
Rabbit Polyclonal Antibody
Catalog # ALS11533

Specification

AIM / CD5L Antibody (N-Terminus) - Product Information

Application	**WB**
Primary Accession	O43866
Reactivity	**Human, Mouse**
Host	**Rabbit**
Clonality	**Polyclonal**
Calculated MW	**38kDa KDa**

AIM / CD5L Antibody (N-Terminus) - Additional Information

Gene ID 922

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

Target/Specificity
13 amino acid peptide from near the amino terminus of human AIM

Reconstitution & Storage
+4°C or -20°C, Avoid repeated freezing and thawing.

Precautions
AIM / CD5L Antibody (N-Terminus) is for research use only and not for use in diagnostic or therapeutic procedures.

AIM / CD5L Antibody (N-Terminus) - 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}

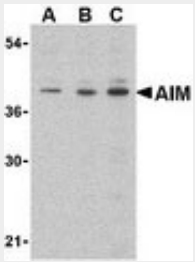
Tissue Location
Expressed in spleen, lymph node, thymus, bone marrow, and fetal liver, but not in non-lymphoid tissues

AIM / CD5L Antibody (N-Terminus) - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

AIM / CD5L Antibody (N-Terminus) - Images



Western blot of AIM in Raji lysate with AIM antibody at (A) 0.5, (B) 1 and (C) 2 g/ml.

AIM / CD5L Antibody (N-Terminus) - Background

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

AIM / CD5L Antibody (N-Terminus) - References

Gebe J.A.,et al.J. Biol. Chem. 272:6151-6158(1997).
Miyazaki T.,et al.Submitted (JUN-1997) to the EMBL/GenBank/DDBJ databases.
Clark H.F.,et al.Genome Res. 13:2265-2270(2003).
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Gregory S.G.,et al.Nature 441:315-321(2006).