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# AIM Antibody
**Catalog # ASC10419**

## Specification

### AIM Antibody - Product Information

| | |
|---|---|
| Application | **WB, IHC** |
| Primary Accession | O43866 |
| Other Accession | AAD01446, 4102235 |
| Reactivity | **Human, Mouse** |
| Host | **Rabbit** |
| Clonality | **Polyclonal** |
| Isotype | **IgG** |
| Application Notes | **AIM antibody can be used for the detection of AIM by Western blot at 0.5 - 2 µg/mL. Antibody can also be used for immunohistochemistry starting at 10 µg/mL.** |

### AIM Antibody - Additional Information

Gene ID **922**

**Other Names**
AIM Antibody: AIM, API6, PRO229, Spalpha, SP-ALPHA, UNQ203/PRO229, CD5 antigen-like, CT-2, CD5 molecule-like

**Target/Specificity**
CD5L;

**Reconstitution & Storage**
AIM antibody can be stored at 4℃ for three months and -20℃, stable for up to one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. Antibodies should not be exposed to prolonged high temperatures.

**Precautions**
AIM Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

### AIM Antibody - 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:<a href="http://www.uniprot.org/citations/24295828" target="_blank">24295828</a>). Involved in early response to microbial infection against various pathogens by acting as a pattern recognition receptor and by promoting autophagy (PubMed:<a href="http://www.uniprot.org/citations/16030018" target="_blank">16030018</a>, PubMed:<a href="http://www.uniprot.org/citations/24223991" target="_blank">24223991</a>, PubMed:<a href="http://www.uniprot.org/citations/24583716" target="_blank">24583716</a>, PubMed:<a href="http://www.uniprot.org/citations/25713983" target="_blank">25713983</a>).

**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

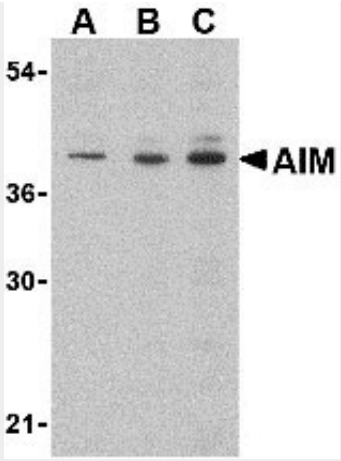
## AIM Antibody - Protocols

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

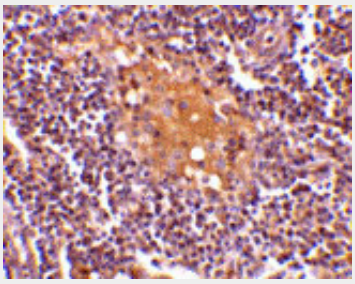
- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

## AIM Antibody - Images

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



Immunohistochemistry of AIM in human lymph node tissue with AIM antibody at 10 µg/mL.

**AIM Antibody - Background**

AIM Antibody: Apoptosis inhibitor of macrophages (AIM) is a member of the scavenger receptor cysteine-rich domain superfamily (SRCR-SF) initially identified as an inducible cell surface ligand of CD5. It was shown that AIM functions in the thymus as the inducer of resistance to apoptosis within CD4+/CD8+ thymocytes and as the supporter of the viability of these cells before thymic selection. AIM was also shown to support macrophage survival and enhance their phagocytic function. More recent experiments using recombinant AIM significantly inhibited apoptosis of NKT and T cells obtained from C. parvum-stimulated livers in vitro, suggesting that AIM functions to induce resistance to apoptosis in these cells and supports host defense against inflammation during infection.

**AIM Antibody - References**

Miyazaki T, Hirokami Y, Matsuhashi N, et al. Increased susceptibility of thymocytes to apoptosis in mice lacking AIM, a novel murine macrophage-derived soluble factor belonging to the scavenger receptor cysteine-rich domain superfamily. J. Exp. Med. 1999; 189:413-22.
Haruta I, Kato Y, Hashimoto E, et al. Association of AIM, a novel apoptosis inhibitory factor, with hepatitis via supporting macrophage survival and enhancing phagocytic function of macrophages. J. Biol. Chem.2001; 276:22910-
Haruta I, Kato Y, Hashimoto E, et al. Association of AIM, a novel apoptosis inhibitory factor, with hepatitis via supporting macrophage survival and enhancing phagocytic function of macrophages. J. Biol. Chem.2001; 276:22910-
Kuwata K, Watanabe H, Jiang S-Y, et al. AIM inhibits apoptosis of T cells and NKT cells in Corynebacterium-induced granuloma formation in mice. Am. J. Path. 2003; 162:837-47.