

**PRODUCT INFORMATION**

<b>Clone ID</b>	DMC441
<b>Target</b>	CD5L
<b>Synonyms</b>	AIM; API6; CT-2; hAIM; PRO229; SP-ALPHA; Spalpha
<b>Host Species</b>	Rabbit
<b>Description</b>	Anti-CD5L antibody(DMC441); IgG1 Chimeric mAb
<b>Delivery</b>	In Stock
<b>Uniprot ID</b>	O43866
<b>IgG type</b>	Rabbit/Human Fc chimeric IgG1
<b>Clonality</b>	Monoclonal
<b>Reactivity</b>	Human
<b>Applications</b>	Flow Cyt
<b>Recommended Dilutions</b>	Flow Cyt 1:100
<b>Purification</b>	Purified from cell culture supernatant by affinity chromatography
<b>Formulation &amp; Reconstitution</b>	Lyophilized from sterile PBS, pH 7.4. Normally 5 % - 8% trehalose is added as protectants before lyophilization. Please see Certificate of Analysis for specific instructions of reconstitution.
<b>Storage &amp; Shipping</b>	Store at -20°C to -80°C for 12 months in lyophilized form. After reconstitution, if not intended for use within a month, aliquot and store at -80°C (Avoid repeated freezing and thawing). Lyophilized proteins are shipped at ambient temperature.

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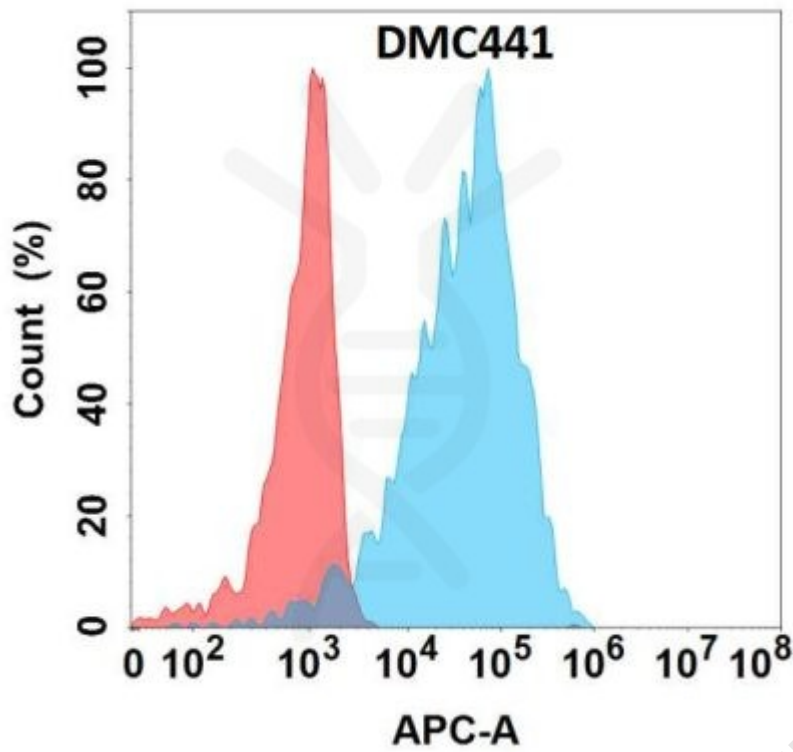
**Background**

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 Fc $\alpha$ : $\mu$  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).

**Usage**

Research use only





**Figure 1.** Flow cytometry analysis with Anti-CD5L (DMC441) on Expi293 cells transfected with human CD5L (Blue histogram) or Expi293 transfected with irrelevant protein (Red histogram).

