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Product datasheet for TR314074

AIM (CD5L) Human shRNA Plasmid Kit (Locus ID 922)

Product data:

Product Type:	shRNA Plasmids
Product Name:	AIM (CD5L) Human shRNA Plasmid Kit (Locus ID 922)
Locus ID:	922
Synonyms:	AIM; API6; CT-2; hAIM; PRO229; SP-ALPHA; Spalpha
Vector:	pRS (TR20003)
E. coli Selection:	Ampicillin
Mammalian Cell Selection:	Puromycin
Format:	Retroviral plasmids
Components:	CD5L - Human, 4 unique 29mer shRNA constructs in retroviral untagged vector(Gene ID = 922). 5µg purified plasmid DNA per construct 29-mer scrambled shRNA cassette in pRS Vector, TR30012, included for free.
RefSeq:	NM_005894, NM_001347698, NM_005894.1, NM_005894.2, BC033586, BC033586.1, NM_005894.3
UniProt ID:	O43866

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Summary:

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).[UniProtKB/Swiss-Prot Function]

shRNA Design:

These shRNA constructs were designed against multiple splice variants at this gene locus. To be certain that your variant of interest is targeted, please contact techsupport@origene.com. If you need a special design or shRNA sequence, please utilize our custom shRNA service.

Performance Guaranteed:

OriGene guarantees that the sequences in the shRNA expression cassettes are verified to correspond to the target gene with 100% identity. One of the four constructs at minimum are guaranteed to produce 70% or more gene expression knock-down provided a minimum transfection efficiency of 80% is achieved. Western Blot data is recommended over qPCR to evaluate the silencing effect of the shRNA constructs 72 hrs post transfection. To properly assess knockdown, the gene expression level from the included scramble control vector must be used in comparison with the target-specific shRNA transfected samples.

For non-conforming shRNA, requests for replacement product must be made within ninety (90) days from the date of delivery of the shRNA kit. To arrange for a free replacement with newly designed constructs, please contact Technical Services at techsupport@origene.com. Please provide your data indicating the transfection efficiency and measurement of gene expression knockdown compared to the scrambled shRNA control (Western Blot data preferred).