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Product datasheet for TL512582

Ceacam1 Mouse shRNA Plasmid (Locus ID 26365)

Product data:

Product Type:	shRNA Plasmids
Product Name:	Ceacam1 Mouse shRNA Plasmid (Locus ID 26365)
Locus ID:	26365
Synonyms:	bb-1; Bgp; Bgp1; C-CAM; Cc1; CD66a; Cea-1; Cea-7; Cea1; Cea7; Hv-2; Hv2; mCEA1; Mhv-1; MHVR; MHVR1
Vector:	pGFP-C-shLenti (TR30023)
E. coli Selection:	Chloramphenicol (34 ug/ml)
Mammalian Cell Selection:	Puromycin
Format:	Lentiviral plasmids
Components:	Ceacam1 - Mouse, 4 unique 29mer shRNA constructs in lentiviral GFP vector(Gene ID = 26365). 5µg purified plasmid DNA per construct 29-mer scrambled shRNA cassette in pGFP-C-shLenti Vector, TR30021, included for free.
RefSeq:	BC016891, NM_001039185, NM_001039186, NM_001039187, NM_011926, NM_001039185.1, NM_001039186.1, NM_011926.1, NM_011926.2, NM_001039187.1, BC114998, BC121189
UniProt ID:	P31809
Summary:	Isoform 1: Cell adhesion protein that mediates homophilic cell adhesion in a calcium-independent manner (By similarity). Plays a role as coinhibitory receptor in immune response, insulin action and functions also as an activator during angiogenesis (PubMed:16680193, PubMed:17081782, PubMed:18544705, PubMed:21029969, PubMed:21081647, PubMed:22496641, PubMed:22962327, PubMed:23696226). Its coinhibitory receptor function is phosphorylation- and PTPN6 -dependent, which in turn, suppress signal transduction of associated receptors by dephosphorylation of their downstream effectors (PubMed:17081782, PubMed:21029969, PubMed:22496641). Plays a role in immune response, of T-cells, natural killer (NK) and neutrophils (PubMed:17081782, PubMed:23696226, PubMed:22496641, PubMed:21029969). Upon TCR/CD3 complex stimulation, inhibits TCR-mediated cytotoxicity by blocking granule exocytosis by mediating homophilic binding to adjacent cells, allowing interaction with and phosphorylation by LCK and interaction with the TCR/CD3 complex which recruits PTPN6 resulting in dephosphorylation of CD247 and ZAP70 (PubMed:22496641). Also inhibits T-cell proliferation

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and cytokine production through inhibition of JNK cascade and plays a crucial role in regulating autoimmunity and anti-tumor immunity by inhibiting T-cell through its interaction with HAVCR2 (PubMed:17081782). Upon natural killer (NK) cells activation, inhibit KLRK1-mediated cytolysis of CEACAM1-bearing tumor cells by trans-homophilic interactions with CEACAM1 on the target cell and lead to cis-interaction between CEACAM1 and KLRK1, allowing PTPN6 recruitment and then VAV1 dephosphorylation (PubMed:23696226). Upon neutrophils activation negatively regulates IL1B production by recruiting PTPN6 to a SYK-TLR4-CEACAM1 complex, that dephosphorylates SYK, reducing the production of reactive oxygen species (ROS) and lysosome disruption, which in turn, reduces the activity of the inflammasome (PubMed:22496641). Downregulates neutrophil production by acting as a coinhibitory receptor for CSF3R by downregulating the CSF3R-STAT3 pathway through recruitment of PTPN6 that dephosphorylates CSF3R (PubMed:21029969). Also regulates insulin action by promoting INS clearance and regulating lipogenesis in liver through regulating insulin signaling (PubMed:18544705). Upon INS stimulation, undergoes phosphorylation by INSR leading to INS clearance by increasing receptor-mediated insulin endocytosis. This inernalization promotes interaction with FASN leading to receptor-mediated insulin degradation and to reduction of FASN activity leading to negative regulation of fatty acid synthesis. INSR-mediated phosphorylation also provokes a down-regulation of cell proliferation through SHC1 interaction resulting in decrease coupling of SHC1 to the MAPK3/ERK1-MAPK1/ERK2 and phosphatidylinositol 3-kinase pathways (By similarity). Functions as activator in angiogenesis by promoting blood vessel remodeling through endothelial cell differentiation and migration and in arteriogenesis by increasing the number of collateral arteries and collateral vessel calibers after ischemia (PubMed:16680193, PubMed:22962327). Also regulates vascular permeability through the VEGFR2 signaling pathway resulting in control of nitric oxide production (PubMed:21081647). Downregulates cell growth in response to EGF through its interaction with SHC1 that mediates interaction with EGFR resulting in decrease coupling of SHC1 to the MAPK3/ERK1-MAPK1/ERK2 pathway (PubMed:15467833). Negatively regulates platelet aggregation by decreasing platelet adhesion on type I collagen through the GPVI-FcRgamma complex (PubMed:19008452). Inhibits cell migration and cell scattering through interaction with FLNA; interfers with the interaction of FLNA with RALA (By similarity). Mediates bile acid transport activity in a phosphorylation dependent manner (By similarity). Negatively regulates osteoclastogenesis (PubMed:25490771).[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).