

Product datasheet for **TL512882V**

Foxp1 Mouse shRNA Lentiviral Particle (Locus ID 108655)

Product data:

Product Type:	shRNA Lentiviral Particles
Product Name:	Foxp1 Mouse shRNA Lentiviral Particle (Locus ID 108655)
Locus ID:	108655
Synonyms:	3110052D19Rik; 4932443N09Rik; AI461938; AW494214
Vector:	pGFP-C-shLenti (TR30023)
Format:	Lentiviral particles
Components:	Foxp1 - Mouse shRNA lentiviral particles (4 unique 29mer target-specific shRNA, 1 scramble control), 0.5 ml each, >10 ⁷ TU/ml.
RefSeq:	BC064764 , NM_001197321 , NM_001197322 , NM_001347345 , NM_053202 , NM_053202.1 , NM_053202.2 , NM_001197322.1 , NM_001197321.1 , BC065386
UniProt ID:	P58462



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Summary:	<p>Transcriptional repressor. Can act with CTBP1 to synergistically repress transcription but CTPBP1 is not essential (PubMed:11358962, PubMed:14701752). Plays an important role in the specification and differentiation of lung epithelium. Acts cooperatively with FOXP4 to regulate lung secretory epithelial cell fate and regeneration by restricting the goblet cell lineage program; the function may involve regulation of AGR2 (PubMed:11358962, PubMed:22675208). Essential transcriptional regulator of B-cell development (PubMed:16819554). Involved in regulation of cardiac muscle cell proliferation (PubMed:20713518). Involved in the columnar organization of spinal motor neurons. Promotes the formation of the lateral motor neuron column (LMC) and the preganglionic motor column (PGC) and is required for respective appropriate motor axon projections. The segment-appropriate generation of spinal chord motor columns requires cooperation with other Hox proteins (PubMed:18667151, PubMed:18662545). Can regulate PITX3 promoter activity; may promote midbrain identity in embryonic stem cell-derived dopamine neurons by regulating PITX3 (PubMed:20175877). Negatively regulates the differentiation of T follicular helper cells T(FH)s (PubMed:24859450). Involved in maintenance of hair follicle stem cell quiescence; the function probably involves regulation of FGF18 (PubMed:23946441). Represses transcription of various pro-apoptotic genes and cooperates with NF-kappa B-signaling in promoting B-cell expansion by inhibition of caspase-dependent apoptosis. Binds to CSF1R promoter elements and is involved in regulation of monocyte differentiation and macrophage functions; repression of CSF1R in monocytes seems to involve NCOR2 as corepressor. Involved in endothelial cell proliferation, tube formation and migration indicative for a role in angiogenesis; the role in neovascularization seems to implicate suppression of SEMA5B. Can negatively regulate androgen receptor signaling (By similarity). [UniProtKB/Swiss-Prot Function]</p>
shRNA Design:	<p>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.</p>
Performance Guaranteed:	<p>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.</p> <p>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).</p>