

Product datasheet for **KN316273LP**

Smad4 Mouse Gene Knockout Kit (CRISPR)

Product data:

Product Type:	Knockout Kits (CRISPR)
Format:	2 gRNA vectors, 1 Luciferase-Puro donor, 1 scramble control
Donor DNA:	Luciferase-Puro
Symbol:	Smad4
Locus ID:	17128
Components:	KN316273G1 , Smad4 gRNA vector 1 in pCas-Guide CRISPR vector (GE100002) KN316273G2 , Smad4 gRNA vector 2 in pCas-Guide CRISPR vector (GE100002) KN316273LPD , donor DNA containing left and right homologous arms and Luciferase-Puro functional cassette. GE100003 , scramble sequence in pCas-Guide vector
Disclaimer:	These products are manufactured and supplied by OriGene under license from ERS. The kit is designed based on the best knowledge of CRISPR technology. The system has been functionally validated for knocking-in the cassette downstream the native promoter. The efficiency of the knock-out varies due to the nature of the biology and the complexity of the experimental process.
RefSeq:	NM_008540 , NM_001364967 , NM_001364968
UniProt ID:	P97471
Synonyms:	AW743858; D18Wsu70e; DPC4; Madh4



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Summary:

Common SMAD (co-SMAD) is the coactivator and mediator of signal transduction by TGF-beta (transforming growth factor). Component of the heterotrimeric SMAD2/SMAD3-SMAD4 complex that forms in the nucleus and is required for the TGF-mediated signaling. Promotes binding of the SMAD2/SMAD4/FAST-1 complex to DNA and provides an activation function required for SMAD1 or SMAD2 to stimulate transcription. Component of the multimeric SMAD3/SMAD4/JUN/FOS complex which forms at the AP1 promoter site; required for synergistic transcriptional activity in response to TGF-beta. May act as a tumor suppressor. Positively regulates PDPK1 kinase activity by stimulating its dissociation from the 14-3-3 protein YWHAQ which acts as a negative regulator (By similarity). Acts synergistically with SMAD1 and YY1 in bone morphogenetic protein (BMP)-mediated cardiac-specific gene expression (PubMed:15329343). Binds to SMAD binding elements (SBEs) (5'-GTCT/AGAC-3') within BMP response element (BMPRE) of cardiac activating regions (PubMed:15329343). In muscle physiology, plays a central role in the balance between atrophy and hypertrophy. When recruited by MSTN, promotes atrophy response via phosphorylated SMAD2/4. MSTN decrease causes SMAD4 release and subsequent recruitment by the BMP pathway to promote hypertrophy via phosphorylated SMAD1/5/8.[UniProtKB/Swiss-Prot Function]

Product images:
