

## Product datasheet for **KN201828BN**

### SMAD4 Human Gene Knockout Kit (CRISPR)

#### Product data:

Product Type:	Knockout Kits (CRISPR)
Format:	2 gRNA vectors, 1 mBFP-Neo donor, 1 scramble control
Donor DNA:	mBFP-Neo
Symbol:	SMAD4
Locus ID:	4089
Components:	<b>KN201828G1</b> , SMAD4 gRNA vector 1 in pCas-Guide CRISPR vector (GE100002) <b>KN201828G2</b> , SMAD4 gRNA vector 2 in pCas-Guide CRISPR vector (GE100002) <b>KN201828BND</b> , donor DNA containing left and right homologous arms and mBFP-Neo functional cassette. <b>GE100003</b> , 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:	<a href="#">NM_005359</a>
UniProt ID:	<a href="#">Q13485</a>
Synonyms:	DPC4; JIP; MADH4; MYHRS



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**Summary:**

This gene encodes a member of the Smad family of signal transduction proteins. Smad proteins are phosphorylated and activated by transmembrane serine-threonine receptor kinases in response to transforming growth factor (TGF)-beta signaling. The product of this gene forms homomeric complexes and heteromeric complexes with other activated Smad proteins, which then accumulate in the nucleus and regulate the transcription of target genes. This protein binds to DNA and recognizes an 8-bp palindromic sequence (GTCTAGAC) called the Smad-binding element (SBE). The protein acts as a tumor suppressor and inhibits epithelial cell proliferation. It may also have an inhibitory effect on tumors by reducing angiogenesis and increasing blood vessel hyperpermeability. The encoded protein is a crucial component of the bone morphogenetic protein signaling pathway. The Smad proteins are subject to complex regulation by post-translational modifications. Mutations or deletions in this gene have been shown to result in pancreatic cancer, juvenile polyposis syndrome, and hereditary hemorrhagic telangiectasia syndrome. [provided by RefSeq, Aug 2017]

**Product images:**
