

**KHDRBS1 Blocking Peptide (Center)**  
**Synthetic peptide**  
**Catalog # BP21904c****Specification**

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**KHDRBS1 Blocking Peptide (Center) - Product Information**Primary Accession [Q07666](#)**KHDRBS1 Blocking Peptide (Center) - Additional Information****Gene ID** 10657**Other Names**

KH domain-containing, RNA-binding, signal transduction-associated protein 1, GAP-associated tyrosine phosphoprotein p62, Src-associated in mitosis 68 kDa protein, Sam68, p21 Ras GTPase-activating protein-associated p62, p68, KHDRBS1 ([http://www.genenames.org/cgi-bin/gene\\_symbol\\_report?hgnc\\_id=18116](http://www.genenames.org/cgi-bin/gene_symbol_report?hgnc_id=18116))  
HGNC:18116

**Target/Specificity**

The synthetic peptide sequence is selected from aa 279-291 of HUMAN KHDRBS1 ([http://www.genenames.org/cgi-bin/gene\\_symbol\\_report?hgnc\\_id=18116](http://www.genenames.org/cgi-bin/gene_symbol_report?hgnc_id=18116))  
HGNC:18116

**Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

**Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

**KHDRBS1 Blocking Peptide (Center) - Protein Information****Name** KHDRBS1 ([HGNC:18116](#))**Function**

Recruited and tyrosine phosphorylated by several receptor systems, for example the T-cell, leptin and insulin receptors. Once phosphorylated, functions as an adapter protein in signal transduction cascades by binding to SH2 and SH3 domain-containing proteins. Role in G2-M progression in the cell cycle. Represses CBP-dependent transcriptional activation apparently by competing with other nuclear factors for binding to CBP. Also acts as a putative regulator of mRNA stability and/or translation rates and mediates mRNA nuclear export. Positively regulates the association of constitutive transport element (CTE)-containing mRNA with large polyribosomes and translation initiation. According to some authors, is not involved in the nucleocytoplasmic export of unspliced (CTE)-containing RNA species according to (PubMed: <a

[22253824](http://www.uniprot.org/citations/22253824)). RNA-binding protein that plays a role in the regulation of alternative splicing and influences mRNA splice site selection and exon inclusion. Binds to RNA containing 5'-[AU]UAA- 3' as a bipartite motif spaced by more than 15 nucleotides. Binds poly(A). Can regulate CD44 alternative splicing in a Ras pathway-dependent manner (By similarity). In cooperation with HNRNPA1 modulates alternative splicing of BCL2L1 by promoting splicing toward isoform Bcl-X(S), and of SMN1 (PubMed:[17371836](http://www.uniprot.org/citations/17371836), PubMed:[20186123](http://www.uniprot.org/citations/20186123)). Can regulate alternative splicing of NRXN1 and NRXN3 in the laminin G-like domain 6 containing the evolutionary conserved neurexin alternative spliced segment 4 (AS4) involved in neurexin selective targeting to postsynaptic partners. In a neuronal activity-dependent manner cooperates synergistically with KHDRBS2/SLIM-1 in regulation of NRXN1 exon skipping at AS4. The cooperation with KHDRBS2/SLIM-1 is antagonistic for regulation of NRXN3 alternative splicing at AS4 (By similarity).

#### **Cellular Location**

Nucleus. Cytoplasm Membrane. Note=Predominantly located in the nucleus but also located partially in the cytoplasm

#### **Tissue Location**

Ubiquitously expressed in all tissue examined. Isoform 1 is expressed at lower levels in brain, skeletal muscle, and liver whereas isoform 3 is intensified in skeletal muscle and in liver

### **KHDRBS1 Blocking Peptide (Center) - Protocols**

Provided below are standard protocols that you may find useful for product applications.

- [Blocking Peptides](#)

### **KHDRBS1 Blocking Peptide (Center) - Images**

### **KHDRBS1 Blocking Peptide (Center) - Background**

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### **KHDRBS1 Blocking Peptide (Center) - References**

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Ota T.,et al.Nat. Genet. 36:40-45(2004).  
Gregory S.G.,et al.Nature 441:315-321(2006).  
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