

**KHDRBS2 Antibody**  
**Purified Mouse Monoclonal Antibody**  
**Catalog # AO1934a**

**Specification**

**KHDRBS2 Antibody - Product Information**

Application	<b>E, WB, FC, IHC</b>
Primary Accession	<a href="#">Q5VWX1</a>
Reactivity	<b>Human, Mouse</b>
Host	<b>Mouse</b>
Clonality	<b>Monoclonal</b>
Isotype	<b>IgG1</b>
Calculated MW	<b>39kDa KDa</b>

**Description**

RNA-binding protein that plays a role in the regulation of alternative splicing and influences mRNA splice site selection and exon inclusion. Its phosphorylation by FYN inhibits its ability to regulate splice site selection. Induces an increased concentration-dependent incorporation of exon in CD44 pre-mRNA by direct binding to purine-rich exonic enhancer. May function as an adapter protein for Src kinases during mitosis. Binds both poly(A) and poly(U) homopolymers. Phosphorylation by PTK6 inhibits its RNA-binding ability (By similarity)

**Immunogen**

Purified recombinant fragment of human KHDRBS2 (AA: 160-349) expressed in E. Coli.

**Formulation**

Purified antibody in PBS with 0.05% sodium azide.

**KHDRBS2 Antibody - Additional Information**

**Gene ID** 202559

**Other Names**

KH domain-containing, RNA-binding, signal transduction-associated protein 2, Sam68-like mammalian protein 1, SLM-1, hSLM-1, KHDRBS2, SLM1

**Dilution**

E~~1/10000  
WB~~1/500 - 1/2000  
FC~~1/200 - 1/400  
IHC~~1/200 - 1/1000

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

**Precautions**

KHDRBS2 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

## KHDRBS2 Antibody - Protein Information

**Name** KHDRBS2

**Synonyms** SLM1

### Function

RNA-binding protein that plays a role in the regulation of alternative splicing and influences mRNA splice site selection and exon inclusion. Binds both poly(A) and poly(U) homopolymers. Phosphorylation by PTK6 inhibits its RNA-binding ability (By similarity). Induces an increased concentration-dependent incorporation of exon in CD44 pre- mRNA by direct binding to purine-rich exonic enhancer. Can regulate alternative splicing of NRXN1 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. Regulates cell-type specific alternative splicing of NRXN1 at AS4 and acts synergistically with SAM68 in exon skipping. In contrast acts antagonistically with SAM68 in NRXN3 exon skipping at AS4. Its phosphorylation by FYN inhibits its ability to regulate splice site selection. May function as an adapter protein for Src kinases during mitosis.

### Cellular Location

Nucleus {ECO:0000250|UniProtKB:Q9WU01}.

### Tissue Location

Highly expressed in brain, lung, kidney and small intestine. Weakly expressed in placenta, liver, spleen, thymus, ovary and colon.

## KHDRBS2 Antibody - Protocols

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

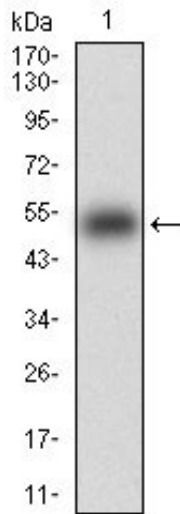
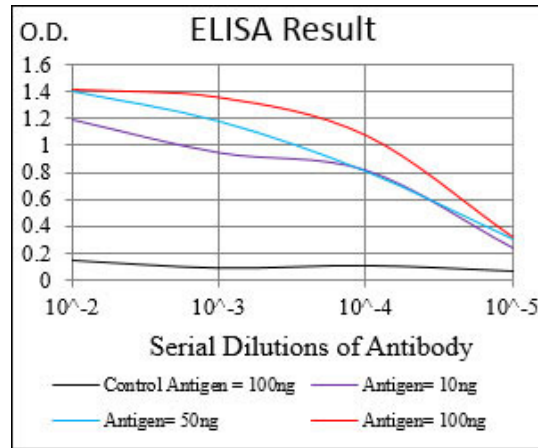


Figure 1: Western blot analysis using KHDRBS2 mAb against human KHDRBS2 (AA: 160-349) recombinant protein. (Expected MW is 46.3 kDa)

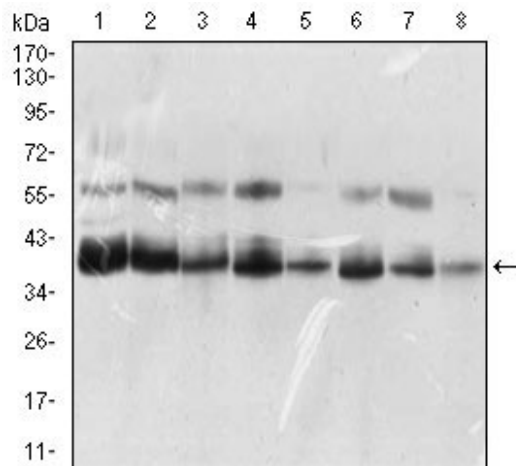


Figure 2: Western blot analysis using KHDRBS2 mouse mAb against K562 (1), HEK293 (2), NTERA-2 (3), HeLa (4), HepG2 (5), Jurkat (6), A431 (7), NIH/3T3 (8) cell lysate.

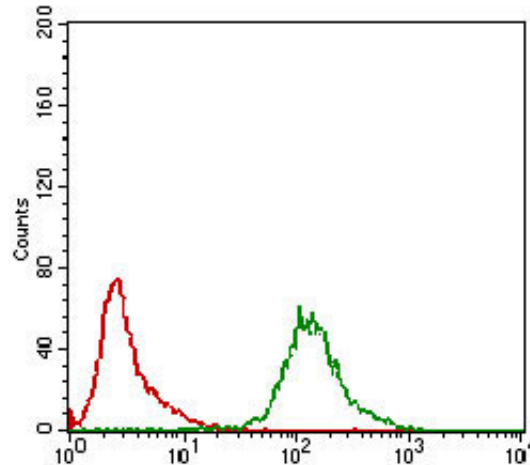


Figure 3: Flow cytometric analysis of K562 cells using KHDRBS2 mouse mAb (green) and negative control (red).

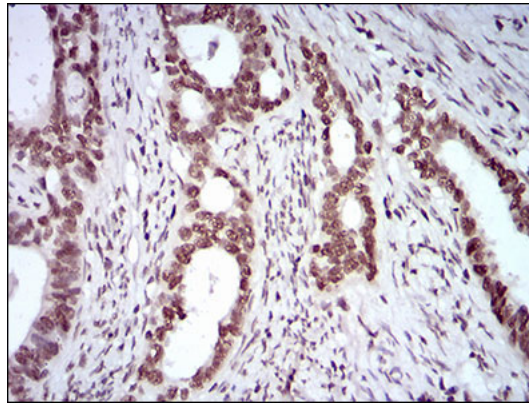


Figure 4: Immunohistochemical analysis of paraffin-embedded cervical cancer tissues using KHDRBS2 mouse mAb with DAB staining.

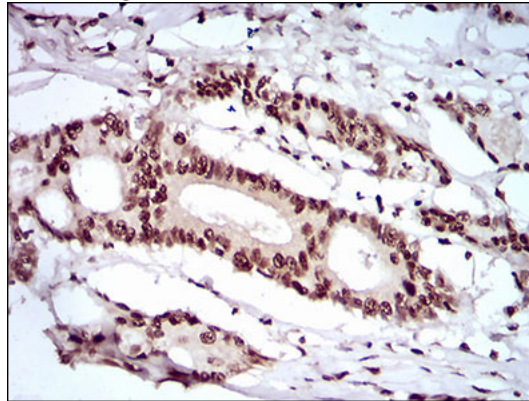


Figure 5: Immunohistochemical analysis of paraffin-embedded colon cancer tissues using KHDRBS2 mouse mAb with DAB staining.

#### **KHDRBS2 Antibody - References**

1. Mol Biol Cell. 2003 Jan;14(1):274-87. 2.