# abcam

# Product datasheet

# Human SMARCB1 (SNF5) knockout HEK-293T cell lysate ab257688

# 4 Images

Overview

Product name Human SMARCB1 (SNF5) knockout HEK-293T cell lysate

**Product overview** 

Knockout cell lysate achieved by CRISPR/Cas9.

Parental Cell Line HEK293T
Organism Human

Mutation description Knockout achieved by using CRISPR/Cas9, 16 bp deletion in exon2 and 1 bp insertion in exon2.

Passage number <20

**Knockout validation** Sanger Sequencing, Western Blot (WB)

**Reconstitution notes**To use as WB control, resuspend the lyophilizate in 50 μL of LDS\* Sample Buffer to have a final

concentration of 2 mg/ml. For reducing conditions, we recommend a final concentration of 0.1 M

DTT.

 $^{\star}$ Usage of SDS sample buffer is not recommended with these lyophilized lysates.

Notes

Lysate preparation: Our lysates are made using RIPA buffer to which we add a protease

inhibitor cocktail and phosphatase inhibitor cocktail (ratio: 300:100:10). *This means that the protein of interest is denatured.* If you require a native form of the protein please use the live cell version - found **here**. Please refer to our lysis protocol for further details on how our lysates are

prepared.

User storage instructions: Lyophilizate may be stored at 4°C. After reconstitution, store at -

20°C for short-term storage or -80°C for long-term storage.

Access thousands of knockout cell lysates, generated from commonly used cancer cell lines.

See here for more information on knockout cell lysates.

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Tested applications Suitable for: WB

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#### **Properties**

# Storage instructions

Store at -80°C. Please refer to protocols.

Components	1 kit
ab262188 - Human SMARCB1 knockout HEK293T cell lysate	1 x 100µg
ab255553 - Human wild-type HEK293T cell lysate	1 x 100μg

#### Cell type

epithelial

**STR Analysis** 

Amelogenin X D5S818: 8, 9 D13S317: 12, 14 D7S820: 11 D16S539: 9, 13 vWA: 16, 19 TH01:

7, 9.3 TPOX: 11 CSF1PO: 11, 12

#### **Target**

#### **Function**

Core component of the BAF (hSWI/SNF) complex. This ATP-dependent chromatin-remodeling complex plays important roles in cell proliferation and differentiation, in cellular antiviral activities and inhibition of tumor formation. The BAF complex is able to create a stable, altered form of chromatin that constrains fewer negative supercoils than normal. This change in supercoiling would be due to the conversion of up to one-half of the nucleosomes on polynucleosomal arrays into asymmetric structures, termed altosomes, each composed of 2 histones octamers. Stimulates in vitro the remodeling activity of SMARCA4/BRG1/BAF190A. Involved in activation of CSF1 promoter. Belongs to the neural progenitors-specific chromatin remodeling complex (npBAF complex) and the neuron-specific chromatin remodeling complex (nBAF complex). During neural development a switch from a stem/progenitor to a post-mitotic chromatin remodeling mechanism occurs as neurons exit the cell cycle and become committed to their adult state. The transition from proliferating neural stem/progenitor cells to post-mitotic neurons requires a switch in subunit composition of the npBAF and nBAF complexes. As neural progenitors exit mitosis and differentiate into neurons, npBAF complexes which contain ACTL6A/BAF53A and PHF10/BAF45A, are exchanged for homologous alternative ACTL6B/BAF53B and DPF1/BAF45B or DPF3/BAF45C subunits in neuron-specific complexes (nBAF). The npBAF complex is essential for the self-renewal/proliferative capacity of the multipotent neural stem cells. The nBAF complex along with CREST plays a role regulating the activity of genes essential for dendrite growth (By similarity). Plays a key role in cell-cycle control and causes cell cycle arrest in G0/G1. Also involved in vitamin D-coupled transcription regulation via its association with the WINAC complex, a chromatin-remodeling complex recruited by vitamin D receptor (VDR), which is required for the ligand-bound VDR-mediated transrepression of the CYP27B1 gene.

### Involvement in disease

Defects in SMARCB1 are a cause of rhabdoid tumor (RDT) [MIM:609322]; also known as malignant rhabdoid tumor (MRT). RDT are a highly malignant group of neoplasms that usually occur in early childhood. SMARCB1/INI1 is also frequently inactivated in epithelioid sarcomas. Defects in SMARCB1 are a cause of schwannomatosis (SCHWA) [MIM:162091]; also called congenital cutaneous neurilemmomatosis. Schwannomas are benign tumors of the peripheral nerve sheath that usually occur singly in otherwise normal individuals. Multiple schwannomas in the same individual suggest an underlying tumor-predisposition syndrome. The most common such syndrome is NF2. The hallmark of NF2 is the development of bilateral vestibular-nerve schwannomas; but two-thirds or more of all NF2-affected individuals develop schwannomas in

other locations, and dermal schwannomas may precede vestibular tumors in NF2-affected children. There have been several reports of individuals with multiple schwannomas who do not show evidence of vestibular schwannoma. Clinical report suggests that schwannomatosis is a clinical entity distinct from other forms of neurofibromatosis.

Sequence similarities

Belongs to the SNF5 family.

Post-translational

Phosphorylated upon DNA damage, probably by ATM or ATR.

modifications

Cellular localization Nucleus.

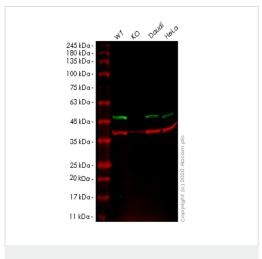
#### **Applications**

The Abpromise guarantee Our Abpromise guarantee covers the use of ab257688 in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
WB		Use at an assay dependent concentration. Predicted molecular weight: 44 kDa.

#### **Images**



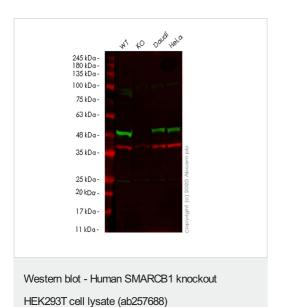
Western blot - Human SMARCB1 knockout HEK293T cell lysate (ab257688) Lane 1: Wild-type HEK293T cell lysate (20 ug)

Lane 2:SMARCB1 knockout HEK293T cell lysate (20 ug)

Lane 3: Daudi cell lysate (20 ug)

Lane 4: HeLa cell lysate (20 ug)

<u>ab126734</u> was shown to specifically react with SNF5/SMARCB1 in wild-type HEK293T cells. Loss of signal was observed when knockout cell line <u>ab267269</u> (knockout cell lysate ab257688) was used. Wild-type and SNF5/SMARCB1 knockout samples were subjected to SDS-PAGE. <u>ab126734</u> and Anti-GAPDH antibody [6C5] - Loading Control (<u>ab8245</u>) were incubated overnight at 4°C at 1 in 1000 dilution and 1 in 20000 dilution respectively. Blots were developed with Goat anti-Rabbit lgG H&L (IRDye<sup>®</sup> 800CW) preadsorbed (<u>ab216773</u>) and Goat anti-Mouse lgG H&L (IRDye<sup>®</sup> 680RD) preadsorbed (<u>ab216776</u>) secondary antibodies at 1 in 20000 dilution for 1 hour at room temperature before imaging.



Lane 1: Wild-type HEK293T cell lysate (20 ug)

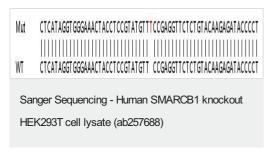
Lane 2:SMARCB1 knockout HEK293T cell lysate (20 ug)

Lane 3: Daudi cell lysate (20 ug)

Lane 4: HeLa cell lysate (20 ug)

ab192864 was shown to specifically react with SNF5/SMARCB1 in wild-type HEK293T cells. Loss of signal was observed when knockout cell line ab267269 (knockout cell lysate ab257688) was used. Wild-type and SNF5/SMARCB1 knockout samples were subjected to SDS-PAGE. ab192864 and Anti-GAPDH antibody [6C5] - Loading Control (ab8245) were incubated overnight at 4°C at 1 in 1000 dilution and 1 in 20000 dilution respectively. Blots were developed with Goat anti-Rabbit lgG H&L (IRDye® 800CW) preadsorbed (ab216773) and Goat anti-Mouse lgG H&L (IRDye® 680RD) preadsorbed (ab216776) secondary antibodies at 1 in 20000 dilution for 1 hour at room temperature before imaging.

Allele-1: 16 bp deletion in exon2



Allele-2: 1 bp insertion in exon2

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