

Product datasheet

Human NSD1 (KMT3B) knockout HeLa cell lysate ab258547

1 Image

Overview

Product name	Human NSD1 (KMT3B) knockout HeLa cell lysate
Product overview	Knockout cell lysate achieved by CRISPR/Cas9.
Parental Cell Line	HeLa
Organism	Human
Mutation description	Knockout achieved by using CRISPR/Cas9, Homozygous: 8 bp deletion in exon 5.
Passage number	<20
Knockout validation	Sanger Sequencing
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. <i>*Usage of SDS sample buffer is not recommended with these lyophilized lysates.</i>

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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Properties

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

Components	1 kit
ab260581 - Human NSD1 knockout HeLa cell lysate	1 x 100µg
ab255929 - Human wild-type HeLa cell lysate	1 x 100µg

Cell type epithelial

Disease Adenocarcinoma

Gender Female

STR Analysis Amelogenin X D5S818: 11, 12 D13S317: 12, 13.3 D7S820: 8, 12 D16S539: 9, 10 vWA: 16, 18 TH01: 7 TPOX: 8, 12 CSF1PO: 9, 10

Target

Function Histone methyltransferase. Preferentially methylates 'Lys-36' of histone H3 and 'Lys-20' of histone H4 (in vitro). Transcriptional intermediary factor capable of both negatively or positively influencing transcription, depending on the cellular context.

Tissue specificity Expressed in the fetal/adult brain, kidney, skeletal muscle, spleen, and the thymus, and faintly in the lung.

Involvement in disease Defects in NSD1 are the cause of Sotos syndrome (SOTOSS) [MIM:117550]; also known as cerebral gigantism. It is a disorder characterized by excessively rapid growth, acromegalic features, and a nonprogressive cerebral disorder with mental retardation. High-arched palate and prominent jaw are noted in several patients. Most cases of Sotos syndrome are sporadic and may represent new dominant mutation.

Defects in NSD1 are the cause of Weaver syndrome (WES) [MIM:277590]. WES is a syndrome of accelerated growth and osseous maturation, unusual craniofacial appearance, hoarse and low-pitched cry, and hypertonia with camptodactyly.

Defects in NSD1 are a cause of Beckwith-Wiedemann syndrome (BWS) [MIM:130650]. BWS is a genetically heterogeneous disorder characterized by anterior abdominal wall defects including exomphalos (omphalocele), pre- and postnatal overgrowth, and macroglossia. Additional less frequent complications include specific developmental defects and a predisposition to embryonal tumors.

Note=A chromosomal aberration involving NSD1 is found in childhood acute myeloid leukemia. Translocation t(5;11)(q35;p15.5) with NUP98.

Note=A chromosomal aberration involving NSD1 is found in an adult form of myelodysplastic syndrome (MDS). Insertion of NUP98 into NSD1 generates a NUP98-NSD1 fusion product.

Sequence similarities Belongs to the histone-lysine methyltransferase family.

Contains 1 AWS domain.

Contains 4 PHD-type zinc fingers.

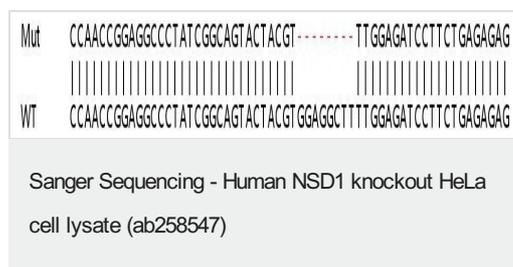
Contains 1 post-SET domain.

Contains 2 PWWP domains.

Contains 1 SET domain.

Cellular localization Nucleus. Chromosome.

Images



Homozygous: 8 bp deletion in exon 5

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