METTL7B (S-14): sc-138422



The Power to Question

BACKGROUND

METTL7B (methyltransferase-like protein 7B) is a 244 amino acid protein belonging to the methyltransferase superfamily. METTL7B is believed to have methyltransferase activity, wherein METTL7B catalyzes the transfer of a methyl group from one compound to another. The gene that encodes METTL7B maps to chromosome 12 which makes up about 4.5% of the human genome. A number of skeletal deformities are linked to chromosome 12 including hypochondrogenesis, achondrogenesis and Kniest dysplasia. Chromosome 12 is also home to a homeobox gene cluster which encodes crucial transcription factors for morphogenesis, and the natural killer complex gene cluster encoding C-type lectin proteins which mediate the NK cell response to MHC I interaction. Trisomy 12p leads to facial development defects, seizure disorders and a host of other symptoms varying in severity depending on the extent of mosaicism and is most severe in cases of complete trisomy.

REFERENCES

- Allen, T.L., et al. 1996. Cytogenetic and molecular analysis in trisomy 12p. Am. J. Med. Genet. 63: 250-256.
- Yang, W., et al. 1998. Low basal transcripts of the COL2A1 collagen gene from lymphoblasts show alternative splicing of exon 12 in the Kniest form of spondyloepiphyseal dysplasia. Hum. Mutat. 1: S1-S2.
- 3. Trowsdale, J., et al. 2001. The genomic context of natural killer receptor extended gene families. Immunol. Rev. 181: 20-38.
- 4. Zumkeller, W., et al. 2004. Genotype/phenotype analysis in a patient with pure and complete trisomy 12p. Am. J. Med. Genet. A 129: 261-264.
- Kelley, J., et al. 2005. Comparative genomics of natural killer cell receptor gene clusters. PLoS Genet. 1: e27.
- Kemmer, L.A., et al. 2006. The natural history of trisomy 12p. Am. J. Med. Genet. A 140: 695-703.

CHROMOSOMAL LOCATION

Genetic locus: METTL7B (human) mapping to 12q13.2; Mettl7b (mouse) mapping to 10 D3.

SOURCE

METTL7B (S-14) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping within an internal region of METTL7B of human origin.

PRODUCT

Each vial contains 100 μg lgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-138422 P, (100 μg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

APPLICATIONS

METTL7B (S-14) is recommended for detection of METTL7B of mouse, rat and human origin by Western Blotting (starting dilution 1:100, dilution range 1:50-1:500), immunofluorescence (starting dilution 1:25, dilution range 1:25-1:250) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000); non cross-reactive with METTL7A.

Suitable for use as control antibody for METTL7B siRNA (h): sc-96008, METTL7B siRNA (m): sc-149392, METTL7B shRNA Plasmid (h): sc-96008-SH, METTL7B shRNA Plasmid (m): sc-149392-SH, METTL7B shRNA (h) Lentiviral Particles: sc-96008-V and METTL7B shRNA (m) Lentiviral Particles: sc-149392-V.

Molecular Weight of METTL7B: 28 kDa.

RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use goat anti-rabbit IgG-HRP: sc-2004 (dilution range: 1:2000-1:100,000) or Cruz Marker™ compatible goat anti-rabbit IgG-HRP: sc-2030 (dilution range: 1:2000-1:5000), Cruz Marker™ Molecular Weight Standards: sc-2035, TBS Blotto A Blocking Reagent: sc-2333 and Western Blotting Luminol Reagent: sc-2048. 2) Immunofluorescence: use goat anti-rabbit IgG-FITC: sc-2012 (dilution range: 1:100-1:400) or goat anti-rabbit IgG-TR: sc-2780 (dilution range: 1:100-1:400) with UltraCruz™ Mounting Medium: sc-24941.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.



Try METTL7B (D-2): sc-398626 or METTL7B (E-10): sc-515267, our highly recommended monoclonal alternatives to METTL7B (S-14).

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