SANTA CRUZ BIOTECHNOLOGY, INC.

ZEB2 (L-20): sc-18392

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

SMAD regulates gene expression by interacting with different classes of transcription factors including DNA-binding multi-zinc finger proteins. ZEB2 (zinc finger E-box-binding protein 2) is a member of the δ-EF1/Zfh1 family of 2-handed zinc finger/homeodomain proteins. ZEB2 contains a SMAD-binding domain, a homeodomain and two clusters of zinc fingers on the N- and C-termini. ZEB2, also known as SMADIP1, ZFHX1B and SIP1 (SMAD interacting protein 1), may be induced by TGFβ treatment. ZEB2 plays a crucial role in normal embryonic development of neural structures and neural crest. The human ZEB2 gene maps to chromosome 2q22.3. Mutations in the ZEB2 gene cause a form of Hirschsprung disease (HSCR). Patients with ZEB2 mutations show mental retardation, delayed motor development, epilepsy, microcephaly, distinct facial features and/or congenital heart disease, all symptoms of HSCR.

REFERENCES

1. van Grunsven, L., et al. 2001. SIP1 (Smad interacting protein1) and δEF1 (δ-crystallin enhancer binding factor) are stucturally similar transcriptional repressors. J. Bone Joint Surg. Am. 83: 40-47.
2. Tylzanowski, P., et al. 2001. Smad-interacting protein 1 is a repressor of liver/bone/kidney alkaline phosohatase transcription in bone morphogenetic protein-induced osteogenic differentiation of C2C12 cells. J. Biol. Chem. 276: 40001-40007.
3. Cacheux, V., et al. 2001. Loss-of-function mutations in SIP1 SMAD interacting protein1 result in a syndromic Hirschsprung disease. Hum. Mol. Genet. 10: 1503-1510.
4. Yamada, K., et al. 2001. Nonsense and frameshift mutations in ZFHX1B, encoding Smad-interacting protein 1, cause a complex developmental disorder with a great variety of clinical features. Am. J. Hum. Genet. 69: 1178-1185.

CHROMOSOMAL LOCATION

Genetic locus: ZEB2 (human) mapping to 2q22.3; Zeb2 (mouse) mapping to 2 B.

SOURCE

ZEB2 (L-20) is an affinity purified goat polyclonal antibody raised against a peptide mapping within an internal region of ZEB2 of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-18392 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.

RESEARCH USE

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

APPLICATIONS

ZEB2 (L-20) is recommended for detection of ZEB2 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

ZEB2 (L-20) is also recommended for detection of ZEB2 in additional species, including equine, canine and porcine.

Suitable for use as control antibody for ZEB2 siRNA (h): sc-38641, ZEB2 siRNA (m): sc-38642, ZEB2 shRNA Plasmid (h): sc-38641-SH, ZEB2 shRNA Plasmid (m): sc-38642-SH, ZEB2 shRNA (h) Lentiviral Particles: sc-38641-V and ZEB2 shRNA (m) Lentiviral Particles: sc-38642-V.

Molecular Weight of ZEB2: 157 kDa.

Positive Controls: Hep G2 cell lysate: sc-2227.

RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use donkey anti-goat IgG-HRP: sc-2020 (dilution range: 1:2000-1:100,000) or Cruz Marker™ compatible donkey anti-goat IgG-HRP: sc-2033 (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 donkey anti-goat IgG-FITC: sc-2024 (dilution range: 1:100-1:400) or donkey anti-goat IgG-TR: sc-2783 (dilution range: 1:100-1:400) with UltraCruz™ Mounting Medium: sc-24941.

SELECT PRODUCT CITATIONS

1. Imamichi, Y., et al. 2007. Collagen type I-induced Smad-interacting protein 1 expression downregulates E-cadherin in pancreatic cancer. Oncogene 26: 2381-2385.
2. Yoshida, J., et al. 2009. Changes in the expression of E-cadherin repressors, snail, SLUG, SIP1, and twist, in the development and progression of ovarian carcinoma: the important role of snail in ovarian tumorigenesis and progression. Med. Mol. Morphol. 42: 82-91.
3. Miura, N., et al. 2009. Clinicopathological significance of Sip1-associated epithelial mesenchymal transition in non-small cell lung cancer progression. Anticancer Res. 29: 4099-4106.
4. Oztas, E., et al. 2010. Novel monoclonal antibodies detect Smad-interacting protein 1 (SIP1) in the cytoplasm of human cells from multiple tumor tissue arrays. Exp. Mol. Pathol. 89: 182-189.