SANTA CRUZ BIOTECHNOLOGY, INC.

CtIP (H-300): sc-22838

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The Power to Question

BACKGROUND

CtBP1 is a cellular phosphoprotein that associates with various proteins and functions as a co-repressor of transcription. CtBP1 and the related protein CtBP2 are characterized as C-terminal binding protein of adenovirus E1A, and they preferentially associate with the E1A via a five amino acid motif, PLDLS, to repress E1A-induced oncogenesis and cellular transformation. CtBP1 is expressed from embryo to adult, but CtBP2 is mainly expressed during embryogenesis. During skeletal and T cell development, CtBP1 and CtBP2 associate with the PLDLSL domain of dEF1, a cellular zinc finger-homeodomain protein, and thereby enhance dEF1-induced transcriptional silencing. In addition, CtBP complexes with CtIP, a protein that recognizes distinctly different protein motifs from CtBP. CtIP binds to the BRCT repeats within the breast cancer gene BRCA1 and enables CtBP to influence BRCA1 activity. CtIP/CtBP binding to BRCA1 inhibits the transactivation of the p21 promoter, and it is critical for regulating p21 transcription in response to DNA damage.

REFERENCES

1. Sollerbrant, K., et al. 1996. The CtBP binding domain in the adenovirus E1A protein controls CR1-dependent transactivation. Nucleic Acids Res. 24: 2578-2584.
2. Sekido, R., et al. 1997. Two mechanisms in the action of repressor deltaEF1: binding site competition with an activator and active repression. Genes Cells 2: 771-783.
3. Schaeper, U., et al. 1998. Interaction between a cellular protein that binds to the C-terminal region of adenovirus E1A (CtBP) and a novel cellular protein is disrupted by E1A through a conserved PLDLS motif. J. Biol. Chem. 273: 8549-8552.
4. Turner, J., et al. 1998. Cloning and characterization of mCtBP2, a co-repressor that associates with basic Krüppel-like factor and other mammalian transcriptional regulators. EMBO J. 17: 5129-5140.

CHROMOSOMAL LOCATION

Genetic locus: RBBP8 (human) mapping to 18q11.2; Rbbp8 (mouse) mapping to 18 A1.

SOURCE

CtIP (H-300) is a rabbit polyclonal antibody raised against amino acids 598-897 of CtIP 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.

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

CtIP (H-300) is recommended for detection of CtIP of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], 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).

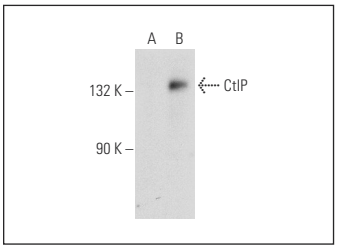
CtIP (H-300) is also recommended for detection of CtIP in additional species, including canine and porcine.

Suitable for use as control antibody for CtIP siRNA (h): sc-37765, CtIP siRNA (m): sc-37766, CtIP shRNA Plasmid (h): sc-37765-SH, CtIP shRNA Plasmid (m): sc-37766-SH, CtIP shRNA (h) Lentiviral Particles: sc-37765-V and CtIP shRNA (m) Lentiviral Particles: sc-37766-V.

Molecular Weight of CtIP: 125 kDa.

Positive Controls: CtIP (m): 293T Lysate: sc-119500, T24 cell lysate: sc-2292 or Jurkat nuclear extract: sc-2132.

DATA



CtIP (H-300): sc-22838. Western blot analysis of CtIP expression in non-transfected: sc-117752 (**A**) and mouse CtIP transfected: sc-119500 (**B**) 293T whole cell lysates.

SELECT PRODUCT CITATIONS

1. Wu, M., et al. 2007. CtIP silencing as a novel mechanism of tamoxifen resistance in breast cancer. Mol. Cancer Res. 5: 1285-1295.
2. Sfeir, A. and de Lange, T. 2012. Removal of shelterin reveals the telomere end-protection problem. Science 336: 593-597.
3. Zimmermann, M., et al. 2013. 53BP1 regulates DSB repair using Rif1 to control 5' end resection. Science 339: 700-704.
4. Lottersberger, F., et al. 2013. Role of 53BP1 oligomerization in regulating double-strand break repair. Proc. Natl. Acad. Sci. USA 110: 2146-2151.