SANTA CRUZ BIOTECHNOLOGY, INC.

JMY (m): 293T Lysate: sc-121158

BACKGROUND

p300 and CBP (CREB-binding proteins) function as coactivators for various transcription factors, including p53. As cofactors, p300 and CBP possesses intrinsic acetyltransferase activity which may allow p300/CBP proteins to regulate transcription through direct acetylation and thereafter, enhance DNA binding activity. JMY is a nuclear cofactor for p300 that cooperatively enhances p53 activation in response to cellular stress. The p53 protein requires p300/CBP coactivator proteins in order to transcriptionally activate target genes. When p53 is activated, p300 component of the coactivator protein complexes associate with JMY and potentiate p53-dependent transcription and apoptosis. p53 acts as a sequence-specific transcription factor and upon stimulation, induces transcription of genes involved in growth arrest, including the waf1/cip1, bax, mdm2, and gadd45 genes. Disruption of p300 and JMY complexes inhibits p53-induced transcription of Bax and blocks apoptosis. Due to alternative splicing, several isoforms of JMY are produced, and these various isoforms have different influencing affects on p53 activation, with some isoforms markedly enhancing p53 responses compared to the other splicing variants.

REFERENCES

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2. Snowden, A.W., et al. 1998. Cell cycle regulation of the transcriptional coactivators p300 and CREB binding protein. Biochem. Pharmacol. 55: 1947-1954.
3. Thomas, A., et al. 1998. Suppression of the p300-dependent mdm2 negative-feedback loop induces the p53 apoptotic function. Genes Dev. 12: 1975-1985.
4. Liu, L., et al. 1999. p53 sites acetylated *in vitro* by PCAF and p300 are acetylated *in vivo* in response to DNA damage. Mol. Cell. Biol. 19: 1202-1209.
5. Shikama, N., et al. 1999. A novel cofactor for p300 that regulates the p53 response. Mol. Cell. 4: 365-376.
6. Yuan, Z.M., et al. 1999. Role for p300 in stabilization of p53 in the response to DNA damage. J. Biol. Chem. 274: 1883-1886.
7. Li, J., et al. 2000. p300 requires its histone acetyltransferase activity and SRC-1 interaction domain to facilitate thyroid hormone receptor activation in chromatin. Mol. Cell. Biol. 20: 2031-2042.
8. Coutts, AS. et al. 2006. The p53 response during DNA damage: impact of transcriptional cofactors. Biochem. Soc. Symp. 73:181-189.
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CHROMOSOMAL LOCATION

Genetic locus: Jmy (mouse) mapping to 13 C3.

PRODUCT

JMY (m): 293T Lysate represents a lysate of mouse JMY transfected 293T cells and is provided as 100 µg protein in 200 µl SDS-PAGE buffer.

APPLICATIONS

JMY (m): 293T Lysate is suitable as a Western Blotting positive control for mouse reactive JMY antibodies. Recommended use: 10-20 µl per lane.

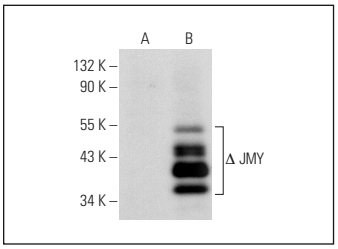
Control 293T Lysate: sc-117752 is available as a Western Blotting negative control lysate derived from non-transfected 293T cells.

JMY (G-11): sc-166030 is recommended as a positive control antibody for Western Blot analysis of enhanced mouse JMY expression in JMY transfected 293T cells (starting dilution 1:100, dilution range 1:100-1:1,000).

RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Western Blotting: use m-IgGκ BP-HRP: sc-516102 or m-IgGκ BP-HRP (Cruz Marker): sc-516102-CM (dilution range: 1:1000-1:10000), Cruz Marker™ Molecular Weight Standards: sc-2035, UltraCruz® Blocking Reagent: sc-516214 and Western Blotting Luminol Reagent: sc-2048.

DATA



JMY (G-11): sc-166030. Western blot analysis of JMY expression in non-transfected: sc-117752 (**A**) and truncated mouse JMY transfected: sc-121158 (**B**) 293T whole cell lysates.

STORAGE

Store at -20° C. Repeated freezing and thawing should be minimized. Sample vial should be boiled once prior to use. Non-hazardous. No MSDS required.

RESEARCH USE

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

PROTOCOLS

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