SANTA CRUZ BIOTECHNOLOGY, INC.

ATF-2 (1-96): sc-4114



BACKGROUND

Eukaryotic gene transcription is regulated by sequence-specific transcription factors which bind modular *cis*-acting promotor and enhancer elements. The ATF/CREB transcription factor family binds the palindromic cAMP response element (CRE) octanucleotide TGACGTCA. The ATF/CREB family includes CREB-1, CREB-2 (also designated ATF-4), ATF-1, ATF-2 and ATF-3. This family of proteins contain highly divergent N-terminal domains, but share a C-terminal leucine zipper for dimerization and DNA binding. ATF-2 forms homodimers and heterodimers with c-Jun to initiate CRE-dependent transcription. Phosphorylation of ATF-2 at Thr 69 and Thr 71 by stress-activated kinases is necessary for transcriptional activation. Myc also induces phosphorylation of ATF-2 at Thr 69 and Thr 71 to prolong the half-life of ATF-2. ATF-2 also functions as a histone acetyltransferase (HAT) by specifically acetylating Histones H2B and H4 *in vitro*.

REFERENCES

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- Lin, Y. and Green, M.R. 1988. Interaction of a common cellular transcription factor, ATF, with regulatory elements in both Ela-and cyclic AMP-inducible promoters. Proc. Natl. Acad. Sci. USA 85: 3396-3400.
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- Kara, C.J., et al. 1990. A cDNA for a human cyclic AMP response elementbinding protein which is distinct from CREB and expressed preferentially in brain. Mol. Cell. Biol. 10: 1347-1357.

CHROMOSOMAL LOCATION

Genetic locus: ATF2 (human) mapping to 2q31.1; Atf2 (mouse) mapping to 2 C3.

SOURCE

ATF-2 (1-96) is produced in *E. coli* as a 40 kDa tagged fusion protein corresponding to amino acids 1-96 mapping at the amino terminal domain of ATF-2 of human origin.

PRODUCT

ATF-2 (1-96) is purified (>95%) by glutathione affinity chromatography; supplied as 50 µg protein in PBS with 5 mM DTT and 50% glycerol.

APPLICATIONS

ATF-2 (1-96) functions as a substrate for JNK and p38 MAP kinases.

Also suitable as a Western blotting positive control for sc-6233.

Molecular Weight of ATF-2: 70 kDa.

STORAGE

Store at -20° C; stable for one year from the date of shipment.

SELECT PRODUCT CITATIONS

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- 14. Samuel, I., et al. 2005. Bile-pancreatic juice exclusion increases p38^{MAPK} activation and TNF- α production in ligation-induced acute pancreatitis in rats. Pancreatology 5: 20-26.

RESEARCH USE

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