

CRF (H-104): sc-10718

BACKGROUND

Individuals suffering from Alzheimer's disease (AD) exhibit dramatic reductions in the content of corticotropin-releasing factor (CRF), increased expression of CRF receptors (CRFRs) and abnormalities in neuronal morphology in affected brain areas. In addition, AD patients show decreased concentrations of CRF in their cerebrospinal fluid, which may contribute to their cognitive impairment. A high affinity CRF binding protein, designated CRF-BP, has been discovered in post-mortem brain samples from AD patients. CRF-BP serves to bind and inactivate CRF, reducing the pool of "free CRF" available to bind CRFRs. Two CRFRs, designated CRF-RI and CRF-RII, have been described and exhibit distinct brain localizations. There are two forms of CRF-RII, referred to as CRF-RII α and CRF-RII β , that result from alternative mRNA splicings. An additional member of the CRF family, urocortin, shares 63% sequence identity with urotensin and 45% sequence identity with CRF. Urocortin specifically binds to and activates CRF-RI and CRF-RII, but binds to CRF-RII more efficiently than CRF, suggesting that it may be the true, high affinity ligand for the CRF receptor type II.

CHROMOSOMAL LOCATION

Genetic locus: CRH (human) mapping to 8q13.1; Crh (mouse) mapping to 3 A2.

SOURCE

CRF (H-104) is a rabbit polyclonal antibody raised against amino acids 53-156 of CRF of human origin.

PRODUCT

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

APPLICATIONS

CRF (H-104) is recommended for detection of CRF of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g 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).

Suitable for use as control antibody for CRF siRNA (h): sc-39395, CRF siRNA (m): sc-39396, CRF shRNA Plasmid (h): sc-39395-SH, CRF shRNA Plasmid (m): sc-39396-SH, CRF shRNA (h) Lentiviral Particles: sc-39395-V and CRF shRNA (m) Lentiviral Particles: sc-39396-V.

Molecular Weight of CRF: 25 kDa.

Positive Controls: PC-12 cell lysate: sc-2250, PC-12 + NGF cell lysate: sc-3808 or mouse brain extract sc-2253.

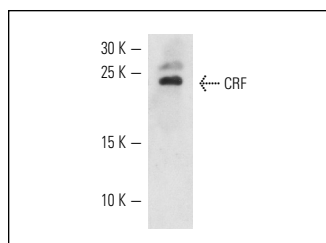
STORAGE

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.

DATA



CRF (H-104): sc-10718. Western blot analysis of CRF expression in PC-12 whole cell lysate.

SELECT PRODUCT CITATIONS

1. Amaral, M.E., et al. 2006. Tumor necrosis factor- α activates signal transduction in hypothalamus and modulates the expression of pro-inflammatory proteins and orexigenic/anorexigenic neurotransmitters. *J. Neurochem.* 98: 203-212.
2. Klimaviciute, A., et al. 2006. Corticotropin-releasing hormone, its binding protein and receptors in human cervical tissue at preterm and term labor in comparison to non-pregnant state. *Reprod. Biol. Endocrinol.* 4: 29.
3. De Souza, C.T., et al. 2008. Distinct subsets of hypothalamic genes are modulated by two different thermogenesis-inducing stimuli. *Obesity* 16: 1239-1247.
4. Pan, Y., et al. 2010. Icarin attenuates chronic mild stress-induced dysregulation of the LHPA stress circuit in rats. *Psychoneuroendocrinology* 35: 272-283.
5. Xu, C., et al. 2011. CRH acts on CRH-R1 and -R2 to differentially modulate the expression of large-conductance calcium-activated potassium channels in human pregnant myometrium. *Endocrinology* 152: 4406-4417.
6. Zhou, Q.G., et al. 2011. Hippocampal neuronal nitric oxide synthase mediates the stress-related depressive behaviors of glucocorticoids by downregulating glucocorticoid receptor. *J. Neurosci.* 31: 7579-7590.
7. Pan, Y., et al. 2013. Impaired hypothalamic Insulin signaling in CUMS rats: restored by icariin and fluoxetine through inhibiting CRF system. *Psychoneuroendocrinology* 38: 122-134.
8. Jimenez, V.A., et al. 2015. An ultrastructural analysis of the effects of ethanol self-administration on the hypothalamic paraventricular nucleus in rhesus macaques. *Front. Cell. Neurosci.* 9: 260.

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