



## JNK1/JNK2 (Phospho-Thr183/Tyr185) Antibody

#11504

**Catalog Number:** 11504-1, 11504-2

**Amount:** 50µg/50µl, 100µg/100µl

**Swiss-Prot No. :** P35568

**Form of Antibody:** Rabbit IgG in phosphate buffered saline (without Mg<sup>2+</sup> and Ca<sup>2+</sup>), pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol.

**Storage/Stability:** Store at -20°C/1 year

**Immunogen:** The antiserum was produced against synthesized phosphopeptide derived from human JNK1/JNK2 around the phosphorylation site of Thr183/Tyr185(M-M-T<sub>P</sub>-P-Y<sub>P</sub>-V-V).

**Purification:** The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific phosphopeptide. The antibody against non-phosphopeptide was removed by chromatography using non-phosphopeptide corresponding to the phosphorylation site.

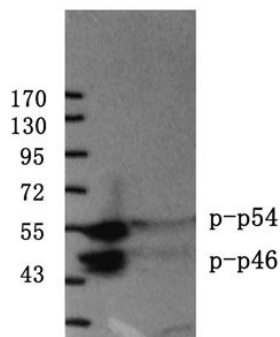
**Specificity/Sensitivity:** JNK1/JNK2 (phospho-Thr183/Tyr185) Antibody detects endogenous levels of JNK1/JNK2 only when phosphorylated at Thr183/Tyr185

**Reactivity:** Human, Mouse, Rat

**Applications:**

Predicted MW: 46 54 kd

WB: 1:500~1:1000 IF: 1:100~1:200

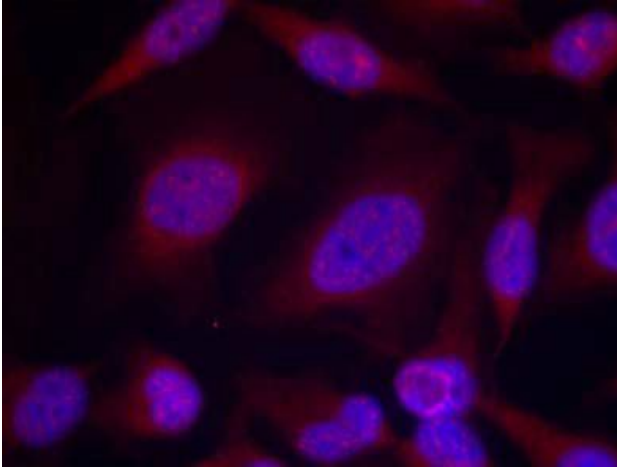


anisomycin + +

p-peptide - +

Western blot analysis of extract from C6 cells (Rat)

using JNK1/JNK2 (phospho-Thr183/Tyr185) Antibody(#11504).



Immunofluorescence staining of methanol-fixed HeLa cells using JNK1/JNK2 (phospho-Thr183/Tyr185)Antibody (#11504, Red).

**Background :**

Responds to activation by environmental stress and pro-inflammatory cytokines by phosphorylating a number of transcription factors, primarily components of AP-1 such as JUN, JDP2 and ATF2 and thus regulates AP-1 transcriptional activity. In T-cells, JNK1 and JNK2 are required for polarized differentiation of T-helper cells into Th1 cells. By similarity. Phosphorylates heat shock factor protein 4 (HSF4). Responds to activation by environmental stress and pro-inflammatory cytokines by phosphorylating a number of transcription factors, primarily components of AP-1 such as c-Jun and ATF2 and thus regulates AP-1 transcriptional activity. In T-cells, JNK1 and JNK2 are required for polarized differentiation of T-helper cells into Th1 cells. JNK2 isoforms display different binding patterns:  $\alpha$ -1 and  $\alpha$ -2 preferentially bind to c-Jun, whereas  $\beta$ -1 and  $\beta$ -2 bind to ATF2. However, there is no correlation between binding and phosphorylation, which is achieved at about the same efficiency by all isoforms. JUNB is not a substrate for JNK2  $\alpha$ -2, and JUND binds only weakly to it. Responds to activation by environmental stress and pro-inflammatory cytokines by phosphorylating a number of transcription factors, primarily components of AP-1 such as c-Jun and ATF2 and thus regulates AP-1 transcriptional activity. Required for stress-induced neuronal apoptosis and the pathogenesis of glutamate excitotoxicity

**References:**

- Davis, R.J. (1999) *Biochem Soc Symp* 64, 1-12.  
Ichijo, H. (1999) *Oncogene* 18, 6087-93.  
Kyriakis, J.M. and Avruch, J. (2001) *Physiol Rev* 81, 807-69.