

ROUG

PhosphoPlus[®] SAPK/JNK (Thr183/Tyr185) Antibody Duet



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For Research Use Only. Not for Use in Diagnostic Procedures.

UniProt ID: Entrez-Gene Id: #P45983 5599

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Phospho-SAPK/JNK (Thr183/Tyr185) (81E11) Rabbit mAb	4668	100 µl	46, 54 kDa	Rabbit IgG
SAPK/JNK Antibody	9252	200 µl	46, 54 kDa	Rabbit

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

Description

PhosphoPlus[®] Duets from Cell Signaling Technology (CST) provide a means to assess protein activation status. Each Duet contains an activation-state and total protein antibody to your target of interest. These antibodies have been selected from CST's product offering based upon superior performance in specified applications.

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, $100 \mu g/ml$ BSA, 50% glycerol and less than 0.02% sodium azide. Store at -20°C. Do not aliquot the antibody.

Background

The stress-activated protein kinase/Jun-amino-terminal kinase SAPK/JNK is potently and preferentially activated by a variety of environmental stresses, including UV and gamma radiation, ceramides, inflammatory cytokines, and in some instances, growth factors and GPCR agonists (1-6). As with the other MAPKs, the core signaling unit is composed of a MAPKKK, typically MEKK1-MEKK4, or by one of the mixed lineage kinases (MLKs), which phosphorylate and activate MKK47. Upon activation, MKKs phosphorylate and activate the SAPK/JNK kinase (2). Stress signals are delivered to this cascade by small GTPases of the Rho family (Rac, Rho, cdc42) (3). Both Rac1 and cdc42 mediate the stimulation of MEKKs and MLKs (3). Alternatively, MKK4/7 can be activated in a GTPase-independent mechanism via stimulation of a germinal center kinase (GCK) family member (4). There are three SAPK/JNK genes each of which undergoes alternative splicing, resulting in numerous isoforms (3). SAPK/JNK, when active as a dimer, can translocate to the nucleus and regulate transcription through its effects on c-Jun, ATF-2, and other transcription factors (3,5).

Background References

- 1. Davis, R.J. (1999) Biochem Soc Symp 64, 1-12.
- 2. Ichijo, H. (1999) Oncogene 18, 6087-93.
- 3. Kyriakis, J.M. and Avruch, J. (2001) *Physiol Rev* 81, 807-69.
- 4. Kyriakis, J.M. (1999) J Biol Chem 274, 5259-62.
- 5. Leppä, S. and Bohmann, D. (1999) Oncogene 18, 6158-62.
- 6. Whitmarsh, A.J. and Davis, R.J. (1998) Trends Biochem Sci 23, 481-5.

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