



**DELTA
BIOLABS**

DB027: JNK1 (C17)

Background:

The mitogen-activated protein kinases (MAPK) consist of several subgroups, including the ERK, JNK, and p38 kinases. The members of this MAPK family are regulated by many different extracellular cues ranging from cytokines, growth factors, and neuropeptides (1). These stimuli activate cell surface receptors to stresses such as cold, heat, osmolarity changes and irradiation. The pathways regulated by the MAPKs control a broad array of cellular responses ranging from survival, cell proliferation, and apoptosis (1,2). The MAPKs family is also characterized by their requirement for dual phosphorylation at a conserved threonine and tyrosine residue for enzymatic activation and both must be phosphorylated for full enzymatic activation (3). The closely related ERK1 (44 kDa) and ERK2 (42 kDa) kinases are characterized by their requirement for dual phosphorylation at a conserved T-E-Y motif (4,5). While JNK1 is activated by dual phosphorylation at a T-P-Y motif and p38 is also activated by dual phosphorylation at a T-G-Y motif (6,7).

Origin:

JNK1 (C17) is provided as an affinity purified rabbit polyclonal antibody, raised against a peptide mapping to the carboxy terminus human JNK1.

Product Details:

Each vial contains 200 µg/ml of affinity purified rabbit IgG, JNK1 (C17) *DB027*, in 1 ml PBS containing 0.1 % sodium azide and 0.2% gelatin.

Competition Studies:

A blocking peptide is also available, *DB027P*, for use in competition studies. Each vial contains 100 µg of peptide in 0.5 ml PBS with 0.1% sodium azide and 100 µg BSA.

Specificity:

JNK1 (C17) *DB027* reacts with JNK1 p46 and JNK3 of mouse, rat and human origin by western blotting, immunoprecipitation, and immunohistochemistry.

Storage:

Store this product at 4° C, do not freeze. The product is stable for one year from the date of shipment.

References:

1. Fanger GR. 1999. Regulation of the MAPK family members: role of the subcellular localization and architectural organization. *Histol Histopathol* 14(3):887-894.
2. Kyriakis JM. 1999. Making the connection: coupling of stress-activated ERK/MAPK (extracellular-signal-regulated kinase/mitogen-activated protein kinase) core signaling modules to extracellular stimuli and biological responses. *Biochem Soc Symp* 64:29-48.
3. Prowse CN, Lew J. 2001. Mechanism of activation of ERK2 by dual phosphorylation. *J Biol Chem* 276(1):99-103.
4. Kyriakis JM, Banerjee P, Nikolakaki E, Dai T, Rubie EA, Ahmad MF, Avruch J, Woodgett JR. 1994. The stress-activated protein kinase subfamily of c-Jun kinases. *Nature* 369(6476):156-160.
5. Cha H, Shapiro P. 2001. Tyrosine-phosphorylated extracellular signal- -regulated kinase associates with Golgi complex during G2/M phase of the cell cycle: evidence for regulation of Golgi structure. *J Cell Biol* 153(7):1355-1367.
6. Chan ED, Winston BW, Jarpe MB, Wynes MW, Riches DW. 1997. Preferential activation of the p46 isoform of JNK/SAPK in mouse macrophages by TNF alpha. *Proc Natl Acad Sci USA* 94(24):13169-13174.
7. Nishida E, Gotoh Y. 1993. The MAP kinase cascade is essential for diverse signal transduction pathways. *Trends Biochem Sci* 18:128-131.