

**MEKK4 (MAP2K4) Antibody (C-term) Blocking peptide**  
**Synthetic peptide**  
**Catalog # BP7916a****Specification**

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**MEKK4 (MAP2K4) Antibody (C-term) Blocking peptide - Product Information**Primary Accession [P45985](#)**MEKK4 (MAP2K4) Antibody (C-term) Blocking peptide - Additional Information****Gene ID** 6416**Other Names**

Dual specificity mitogen-activated protein kinase kinase 4, MAP kinase kinase 4, MAPKK 4, JNK-activating kinase 1, MAPK/ERK kinase 4, MEK 4, SAPK/ERK kinase 1, SEK1, Stress-activated protein kinase kinase 1, SAPK kinase 1, SAPKK-1, SAPKK1, c-Jun N-terminal kinase kinase 1, JNKK, MAP2K4, JNKK1, MEK4, MKK4, PRKMK4, SEK1, SERK1, SKK1

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP7916a](/product/products/AP7916a) was selected from the C-term region of human MKK4. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

**Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

**Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

**MEKK4 (MAP2K4) Antibody (C-term) Blocking peptide - Protein Information****Name** MAP2K4**Synonyms** JNKK1, MEK4, MKK4, PRKMK4, SEK1, SERK1,**Function**

Dual specificity protein kinase which acts as an essential component of the MAP kinase signal transduction pathway. Essential component of the stress-activated protein kinase/c-Jun N-terminal kinase (SAP/JNK) signaling pathway. With MAP2K7/MKK7, is the one of the only known kinase to directly activate the stress-activated protein kinase/c-Jun N-terminal kinases MAPK8/JNK1, MAPK9/JNK2 and MAPK10/JNK3. MAP2K4/MKK4 and MAP2K7/MKK7 both activate the JNKs by phosphorylation, but they differ in their preference for the phosphorylation site in the Thr-Pro-Tyr motif. MAP2K4 shows preference for phosphorylation of the Tyr residue and MAP2K7/MKK7 for the

Thr residue. The phosphorylation of the Thr residue by MAP2K7/MKK7 seems to be the prerequisite for JNK activation at least in response to pro-inflammatory cytokines, while other stimuli activate both MAP2K4/MKK4 and MAP2K7/MKK7 which synergistically phosphorylate JNKs. MAP2K4 is required for maintaining peripheral lymphoid homeostasis. The MKK/JNK signaling pathway is also involved in mitochondrial death signaling pathway, including the release cytochrome c, leading to apoptosis. Whereas MAP2K7/MKK7 exclusively activates JNKs, MAP2K4/MKK4 additionally activates the p38 MAPKs MAPK11, MAPK12, MAPK13 and MAPK14.

**Cellular Location**

Cytoplasm. Nucleus.

**Tissue Location**

Abundant expression is seen in the skeletal muscle. It is also widely expressed in other tissues

**MEKK4 (MAP2K4) Antibody (C-term) Blocking peptide - Protocols**

Provided below are standard protocols that you may find useful for product applications.

- [Blocking Peptides](#)

**MEKK4 (MAP2K4) Antibody (C-term) Blocking peptide - Images****MEKK4 (MAP2K4) Antibody (C-term) Blocking peptide - Background**

MKK4 is a dual specificity protein kinase that belongs to the Ser/Thr protein kinase family. This kinase is a direct activator of MAP kinases in response to various environmental stresses or mitogenic stimuli. It has been shown to activate MAPK8/JNK1, MAPK9/JNK2, and MAPK14/p38, but not MAPK1/ERK2 or MAPK3/ERK3. This kinase is phosphorylated, and thus activated by MAP3K1/MEKK. The knockout studies in mice suggested the roles of this kinase in mediating survival signal in T cell development, as well as in the organogenesis of liver.

**MEKK4 (MAP2K4) Antibody (C-term) Blocking peptide - References**

Sundarrajan, M., et al., Arthritis Rheum. 48(9):2450-2460 (2003). Ho, D.T., et al., J. Biol. Chem. 278(35):32662-32672 (2003). Javelaud, D., et al., J. Biol. Chem. 278(27):24624-24628 (2003). Witowsky, J.A., et al., J. Biol. Chem. 278(3):1403-1406 (2003). Lee, H.Y., et al., J. Biol. Chem. 278(26):23630-23638 (2003).