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# ProductInformation

**ANTI-MITOGEN ACTIVATED PROTEIN KINASE KINASE (MEK, MAPKK)**
Developed in Rabbit
Delipidized, Whole Antibody

Product Number **M 5795**

## Product Description

Anti-Mitogen Activated Protein Kinase Kinase (MEK, MAPKK) is developed in rabbit using a synthetic peptide (Lys-Lys-Leu-Glu-Glu-Leu-Glu-Leu-Asp-Glu-Gln-Gln-Arg-Lys-Arg) derived from the N-terminal region (amino acids 34-48) of human MAP-kinase kinase 1 (MEK-1) coupled to KLH as immunogen. This sequence is highly homologous between MEK-1 and MEK-2 isoforms and is identical in human, rabbit, rat and mouse MEK-1.

Anti-MEK reacts in immunoblotting (SDS-PAGE) with MEK-1a (45 kDa protein) and MEK-2 (46 kDa protein) using mouse NIH 3T3 fibroblast cells lysate and rat brain extract, usually forming one band at 46kDa. Inhibition of the MEK band(s) is specifically achieved with MEK-1 peptide (34-48), but not with MAP kinase peptide (317-339), corresponding to subdomain XI of ERK-1.

MAP kinase kinase (MAPKK, mitogen-activated protein kinase kinase, also termed MEK), consists of a family of Thr/Tyr dual specificty protein kinases, which play a crucial role in various signal transduction pathways leading signals of growth factor, receptors as well as G protein-coupled receptors to their intracellular targets.[1-4] MAP kinase kinase regulates several cellular processes including proliferation, differentiation, cellular morphology and oncogenesis.[4,5] Molecular cloning has established that MAP kinase kinase (MAPKK, MEK) consists of three different isoforms with a high degree of homology between them, MEK-1a (45 kDa), MEK-1b (41 kDa), and MEK-2 (46 kDa).[2] Activation of MEK-1 and MEK-2 in mitogen-stimulated cells is directly mediated by MAP kinase kinase kinases (MAPKKKs), such as Raf-1 kinase, which phosphorylates two serine residues in the regulatory sites of MEK.[4,5] Following activation, MEK phosphorylates MAP kinase (ERK-1 and ERK-2) in the MAP kinase cascade. MEK isoforms are widely expressed, in the central nervous system, thymus, spleen, heart, lung, kidney, and are expressed in high levels in PC12 cells and in fibroblasts.[1,3] Antibodies that react specifically with MEK may be used to study the specific activation requirements, differential tissue expression and intracellular localization of MEK in normal and neoplastic tissue.

Anti-MAP Kinase Kinase may be used to detect MEK-1a and MEK-2 isoforms in immunoblotting using cell culture and brain tissue extracts. The antibody may be used to detect the phosphorylated form of MEK.

## Reagents

The antiserum has been treated to remove lipoproteins, and is provided as whole antiserum containing 0.1% sodium azide as a preservative.

## Precautions and Disclaimer

Due to the sodium azide content a material safety sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution. Consult the MSDS for information regarding hazards and safe handling practices.

## Storage/Stability

For continuous use, store at 2-8 °C. For extended storage freeze in working aliquots. Repeated freezing and thawing is not recommended. Storage in "frost-free" freezers is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use.

## Product Profile

1. A titer of 1:20,000 was determined by indirect immunoblotting using rat brain extract. Specific staining of MAP kinase kinase (MEK-1a, a 45 kDa band, and MEK-2, a 46 kDa band), usually forming one band at 46 kDa is observed.
2. A titer of 1:20,000 was determined by indirect immunoblotting using mouse NIH 3T3 fibroblasts lysate. Specific staining of MEK (MEK-1a, a 45kDa band, and MEK-2, a 46 kDa band) , usually forming one band at 46 kDa, was observed.

In order to obtain best results, it is recommended that each user determine the optimal working dilution for individual applications by titration assay.

**References**
1. Ahn, N. G., et al., J. Biol. Chem., **266**, 4220 (1991).
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3. Ahn, N. G., et al., Curr. Opin. Cell. Biol., **4**, 992 (1992).
4. Seger, R., and Krebs, E., FASEB J., **9**, **726** (1995).
5. Cowley, S., et al., Cell, **77**, 841 (1994).

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