

**Bi-Phospho-MEK1(S218/222) Antibody**  
**Affinity Purified Rabbit Polyclonal Antibody (Pab)**  
**Catalog # AP3159a****Specification**

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**Bi-Phospho-MEK1(S218/222) Antibody - Product Information**

Application	WB, IHC-P, DB,E
Primary Accession	<a href="#">Q02750</a>
Other Accession	<a href="#">P36506</a> , <a href="#">Q63932</a> , <a href="#">P36507</a> , <a href="#">Q90891</a> , <a href="#">Q05116</a> , <a href="#">Q01986</a> , <a href="#">P29678</a> , <a href="#">P31938</a> , <a href="#">Q63980</a> , <a href="#">Q10664</a> , <a href="#">Q24324</a>
Reactivity	Human
Predicted	Drosophila, C.Elegans, Hamster, Mouse, Rabbit, Rat, Xenopus, Chicken
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG

**Bi-Phospho-MEK1(S218/222) Antibody - Additional Information****Gene ID** 5604**Other Names**

Dual specificity mitogen-activated protein kinase kinase 1, MAP kinase kinase 1, MAPKK 1, MKK1, ERK activator kinase 1, MAPK/ERK kinase 1, MEK 1, MAP2K1, MEK1, PRKMK1

**Target/Specificity**

This MEK1 Antibody is generated from rabbits immunized with a KLH conjugated synthetic phosphopeptide corresponding to amino acid residues surrounding S218/222 of human MEK1.

**Dilution**WB~~1:500  
IHC-P~~1:50~100  
DB~~1:500**Format**

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

**Storage**

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

**Precautions**

Bi-Phospho-MEK1(S218/222) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

**Bi-Phospho-MEK1(S218/222) Antibody - Protein Information**

**Name** MAP2K1 ([HGNC:6840](#))

**Synonyms** MEK1, PRKMK1

**Function** Dual specificity protein kinase which acts as an essential component of the MAP kinase signal transduction pathway. Binding of extracellular ligands such as growth factors, cytokines and hormones to their cell-surface receptors activates RAS and this initiates RAF1 activation. RAF1 then further activates the dual-specificity protein kinases MAP2K1/MEK1 and MAP2K2/MEK2. Both MAP2K1/MEK1 and MAP2K2/MEK2 function specifically in the MAPK/ERK cascade, and catalyze the concomitant phosphorylation of a threonine and a tyrosine residue in a Thr-Glu-Tyr sequence located in the extracellular signal-regulated kinases MAPK3/ERK1 and MAPK1/ERK2, leading to their activation and further transduction of the signal within the MAPK/ERK cascade. Activates BRAF in a KSR1 or KSR2-dependent manner; by binding to KSR1 or KSR2 releases the inhibitory intramolecular interaction between KSR1 or KSR2 protein kinase and N-terminal domains which promotes KSR1 or KSR2-BRAF dimerization and BRAF activation (PubMed:[29433126](#)). Depending on the cellular context, this pathway mediates diverse biological functions such as cell growth, adhesion, survival and differentiation, predominantly through the regulation of transcription, metabolism and cytoskeletal rearrangements. One target of the MAPK/ERK cascade is peroxisome proliferator-activated receptor gamma (PPARG), a nuclear receptor that promotes differentiation and apoptosis. MAP2K1/MEK1 has been shown to export PPARG from the nucleus. The MAPK/ERK cascade is also involved in the regulation of endosomal dynamics, including lysosome processing and endosome cycling through the perinuclear recycling compartment (PNRC), as well as in the fragmentation of the Golgi apparatus during mitosis.

#### **Cellular Location**

Cytoplasm, cytoskeleton, microtubule organizing center, centrosome. Cytoplasm, cytoskeleton, microtubule organizing center, spindle pole body. Cytoplasm. Nucleus Membrane; Peripheral membrane protein. Note=Localizes at centrosomes during prometaphase, midzone during anaphase and midbody during telophase/cytokinesis (PubMed:[14737111](#)). Membrane localization is probably regulated by its interaction with KSR1 (PubMed:[10409742](#))

#### **Tissue Location**

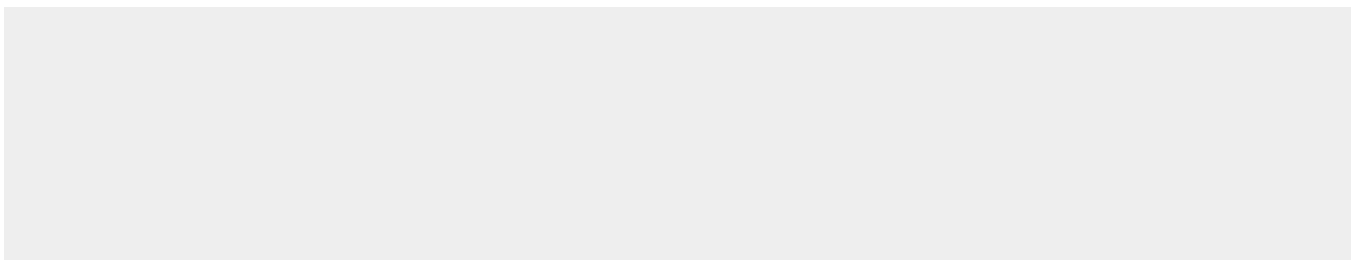
Widely expressed, with extremely low levels in brain.

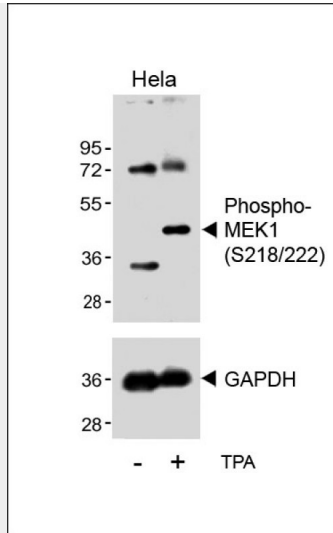
#### **Bi-Phospho-MEK1(S218/222) Antibody - Protocols**

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

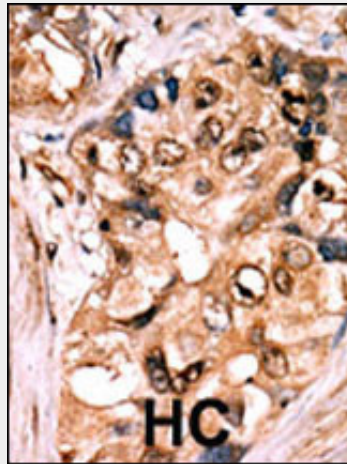
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

#### **Bi-Phospho-MEK1(S218/222) Antibody - Images**

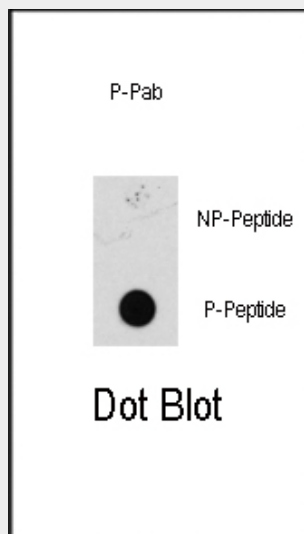




Western blot analysis of lysates from HeLa cell line, untreated or treated with TPA(200nM, 30min), using Bi-Phospho-MEK1(S218/222) Antibody(upper) or GAPDH (lower).



Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by AEC staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.



Dot blot analysis of anti-Phospho-MEK1 Pab (Cat. #AP3159a) on nitrocellulose membrane. 50ng of Phospho-peptide or Non Phospho-peptide per dot were adsorbed. Antibody working concentrations are 0.5ug per ml.

#### **Bi-Phospho-MEK1(S218/222) Antibody - Background**

MEK1 is a dual specificity protein kinase that belongs to the MAP kinase kinase family. This kinase is known to play a critical role in mitogen growth factor signal transduction. It phosphorylates and thus activates MAPK1/ERK2 and MAPK2/ERK3. The activation of this kinase itself is dependent on the Ser/Thr phosphorylation by MAP kinase kinase kinases. The inhibition or degradation of this kinase is found to be involved in the pathogenesis of Yersinia and anthrax.

#### **Bi-Phospho-MEK1(S218/222) Antibody - References**

Ohren, J.F., et al., J. Neural Transm. 11(12):1192-1197 (2004). Naegele, S., et al., J. Biol. Chem. 279(44):46023-46034 (2004). Ussar, S., et al., J. Biol. Chem. 279(42):43861-43869 (2004). Beausoleil, S.A., et al., Proc. Natl. Acad. Sci. U.S.A. 101(33):12130-12135 (2004). Spence, H.J., et al., EMBO Rep. 5(5):484-489 (2004).