

**Erk2 Antibody**  
**Mouse Monoclonal Antibody (Mab)**  
**Catalog # AW5059****Specification**

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**Erk2 Antibody - Product Information**

Application	WB,E
Primary Accession	<a href="#">P28482</a>
Other Accession	<a href="#">P26696</a> , <a href="#">P63086</a> , <a href="#">P63085</a> , <a href="#">P46196</a>
Reactivity	Human, Mouse, Rat
Predicted	Bovine, Xenopus
Host	Mouse
Clonality	Monoclonal
Calculated MW	H=41;M=41;Rat=41 KDa
Isotype	IgG1,k
Antigen Source	Human

**Erk2 Antibody - Additional Information****Gene ID** 5594**Antigen Region**  
154-183**Other Names**

MAPK1;ERK2; PRKM1; PRKM2; Mitogen-activated protein kinase 1; Mitogen-activated protein kinase 1; ERT1; Mitogen-activated protein kinase 1; Extracellular signal-regulated kinase 2; Mitogen-activated protein kinase 1; MAP kinase isoform p42; Mitogen-activated protein kinase 1; Mitogen-activated protein kinase 2

**Dilution**

WB~~1:1000

**Target/Specificity**

This Erk2 antibody is generated from mice immunized with a KLH conjugated synthetic peptide between 154-183 amino acids from human Erk2.

**Format**

Purified monoclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein G column, followed by dialysis against PBS.

**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**

Erk2 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

**Erk2 Antibody - Protein Information**

**Name** MAPK1 ([HGNC:6871](#))

**Synonyms** ERK2, PRKM1, PRKM2

### Function

Serine/threonine kinase which acts as an essential component of the MAP kinase signal transduction pathway. MAPK1/ERK2 and MAPK3/ERK1 are the 2 MAPKs which play an important role in the MAPK/ERK cascade. They participate also in a signaling cascade initiated by activated KIT and KITLG/SCF. Depending on the cellular context, the MAPK/ERK cascade mediates diverse biological functions such as cell growth, adhesion, survival and differentiation through the regulation of transcription, translation, cytoskeletal rearrangements. The MAPK/ERK cascade also plays a role in initiation and regulation of meiosis, mitosis, and postmitotic functions in differentiated cells by phosphorylating a number of transcription factors. About 160 substrates have already been discovered for ERKs. Many of these substrates are localized in the nucleus, and seem to participate in the regulation of transcription upon stimulation. However, other substrates are found in the cytosol as well as in other cellular organelles, and those are responsible for processes such as translation, mitosis and apoptosis. Moreover, the MAPK/ERK cascade is also involved in the regulation of the 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. The substrates include transcription factors (such as ATF2, BCL6, ELK1, ERF, FOS, HSF4 or SPZ1), cytoskeletal elements (such as CANX, CTTN, GJA1, MAP2, MAPT, PXN, SORBS3 or STMN1), regulators of apoptosis (such as BAD, BTG2, CASP9, DAPK1, IER3, MCL1 or PPARG), regulators of translation (such as EIF4EBP1 and FXR1) and a variety of other signaling-related molecules (like ARHGEF2, DCC, FRS2 or GRB10). Protein kinases (such as RAF1, RPS6KA1/RSK1, RPS6KA3/RSK2, RPS6KA2/RSK3, RPS6KA6/RSK4, SYK, MKNK1/MNK1, MKNK2/MNK2, RPS6KA5/MSK1, RPS6KA4/MSK2, MAPKAPK3 or MAPKAPK5) and phosphatases (such as DUSP1, DUSP4, DUSP6 or DUSP16) are other substrates which enable the propagation the MAPK/ERK signal to additional cytosolic and nuclear targets, thereby extending the specificity of the cascade. Mediates phosphorylation of TPR in response to EGF stimulation. May play a role in the spindle assembly checkpoint. Phosphorylates PML and promotes its interaction with PIN1, leading to PML degradation. Phosphorylates CDK2AP2 (By similarity). Phosphorylates phosphoglycerate kinase PGK1 under hypoxic conditions to promote its targeting to the mitochondrion and suppress the formation of acetyl-coenzyme A from pyruvate (PubMed:<a href="http://www.uniprot.org/citations/26942675" target="\_blank">26942675</a>).

### Cellular Location

Cytoplasm, cytoskeleton, spindle. Nucleus. Cytoplasm, cytoskeleton, microtubule organizing center, centrosome. Cytoplasm. Membrane, caveola {ECO:0000250|UniProtKB:P63086}. Cell junction, focal adhesion {ECO:0000250|UniProtKB:P63085}. Note=Associated with the spindle during prometaphase and metaphase (By similarity). PEA15-binding and phosphorylated DAPK1 promote its cytoplasmic retention. Phosphorylation at Ser- 246 and Ser-248 as well as autophosphorylation at Thr-190 promote nuclear localization.

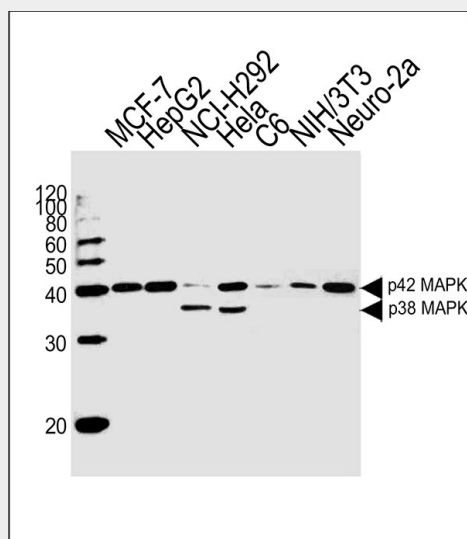
### Erk2 Antibody - Protocols

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](#)

## Erk2 Antibody - Images



Western blot analysis of lysates from MCF-7, HepG2, NCI-H292, Hela, rat C6, NIH/3T3, mouse Neuro-2a cell line (from left to right), using MAPK1 Antibody (Center) (Cat. #AW5059). AW5059 was diluted at 1:1000 at each lane. A goat anti-mouse IgG H&L (HRP) at 1:10000 dilution was used as the secondary antibody. Lysates at 20 µg per lane.

## Erk2 Antibody - Background

Serine/threonine kinase which acts as an essential component of the MAP kinase signal transduction pathway. MAPK1/ERK2 and MAPK3/ERK1 are the 2 MAPKs which play an important role in the MAPK/ERK cascade. They participate also in a signaling cascade initiated by activated KIT and KITLG/SCF. Depending on the cellular context, the MAPK/ERK cascade mediates diverse biological functions such as cell growth, adhesion, survival and differentiation through the regulation of transcription, translation, cytoskeletal rearrangements. The the MAPK/ERK cascade plays also a role in initiation and regulation of meiosis, mitosis, and postmitotic functions in differentiated cells by phosphorylating a number of transcription factors. About 160 substrates have already been discovered for ERKs. Many of these substrates are localized in the nucleus, and seem to participate in the regulation of transcription upon stimulation. However, other substrates are found in the cytosol as well as in other cellular organelles, and those are responsible for processes such as translation, mitosis and apoptosis. Moreover, the MAPK/ERK cascade is also involved in the regulation of the 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. The substrates include transcription factors (such as ATF2, BCL6, ELK1, ERF, FOS, HSF4 or SPZ1), cytoskeletal elements (such as CANX, CTTN, GJA1, MAP2, MAPT, PXN, SORBS3 or STMN1), regulators of apoptosis (such as BAD, BTG2, CASP9, DAPK1, IER3, MCL1 or PPARG), regulators of translation (such as EIF4EBP1) and a variety of other signaling-related molecules (like ARHGEF2, DCC, FRS2 or GRB10). Protein kinases (such as RAF1, RPS6KA1/RSK1, RPS6KA3/RSK2, RPS6KA2/RSK3, RPS6KA6/RSK4, SYK, MKNK1/MNK1, MKNK2/MNK2, RPS6KA5/MSK1, RPS6KA4/MSK2, MAPKAPK3 or MAPKAPK5) and phosphatases (such as DUSP1, DUSP4, DUSP6 or DUSP16) are other substrates which enable the propagation the MAPK/ERK signal to additional cytosolic and nuclear targets, thereby extending the specificity of the cascade.

Acts as a transcriptional repressor. Binds to a [GC]AAA[GC] consensus sequence. Repress the expression of interferon gamma-induced genes. Seems to bind to the promoter of CCL5, DMP1, IFIH1, IFITM1, IRF7, IRF9, LAMP3, OAS1, OAS2, OAS3 and STAT1. Transcriptional activity is independent of kinase activity.

**Erk2 Antibody - References**

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Gonzalez F.A., et al. FEBS Lett. 304:170-178(1992).  
Dunham I., et al. Nature 402:489-495(1999).  
Gevaert K., et al. Nat. Biotechnol. 21:566-569(2003).  
Sgouras D.N., et al. EMBO J. 14:4781-4793(1995).