

ERK 8 Polyclonal Antibody
Catalog # AP69813**Specification**

ERK 8 Polyclonal Antibody - Product Information

Application	WB, IHC-P, IF
Primary Accession	Q8TD08
Reactivity	Human, Mouse
Host	Rabbit
Clonality	Polyclonal

ERK 8 Polyclonal Antibody - Additional Information**Gene ID** 225689**Other Names**

MAPK15; ERK7; ERK8; Mitogen-activated protein kinase 15; MAP kinase 15; MAPK 15; Extracellular signal-regulated kinase 7; ERK-7; Extracellular signal-regulated kinase 8; ERK-8

Dilution

WB~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. Immunofluorescence: 1/200 - 1/1000. ELISA: 1/10000. Not yet tested in other applications.

IHC-P~~N/A

IF~~1:50~200

Format

Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.

Storage Conditions

-20°C

ERK 8 Polyclonal Antibody - Protein Information**Name** MAPK15 ([HGNC:24667](#))**Function**

Atypical MAPK protein that regulates several process such as autophagy, ciliogenesis, protein trafficking/secretion and genome integrity, in a kinase activity-dependent manner (PubMed:20733054, PubMed:21847093, PubMed:22948227, PubMed:24618899, PubMed:29021280). Controls both, basal and starvation-induced autophagy through its interaction with GABARAP, MAP1LC3B and GABARAPL1 leading to autophagosome formation, SQSTM1 degradation and reduced MAP1LC3B inhibitory phosphorylation (PubMed:22948227). Regulates primary cilium formation and the localization of ciliary proteins involved in cilium structure,

transport, and signaling (PubMed:29021280). Prevents the relocation of the sugar-adding enzymes from the Golgi to the endoplasmic reticulum, thereby restricting the production of sugar- coated proteins (PubMed:24618899). Upon amino-acid starvation, mediates transitional endoplasmic reticulum site disassembly and inhibition of secretion (PubMed:21847093). Binds to chromatin leading to MAPK15 activation and interaction with PCNA, that which protects genomic integrity by inhibiting MDM2-mediated degradation of PCNA (PubMed:20733054). Regulates DA transporter (DAT) activity and protein expression via activation of RhoA (PubMed:28842414). In response to H(2)O(2) treatment phosphorylates ELAVL1, thus preventing it from binding to the PDCD4 3'UTR and rendering the PDCD4 mRNA accessible to miR-21 and leading to its degradation and loss of protein expression (PubMed:26595526). Also functions in a kinase activity-independent manner as a negative regulator of growth (By similarity). Phosphorylates in vitro FOS and MBP (PubMed:11875070, PubMed:16484222, PubMed:19166846, PubMed:20638370). During oocyte maturation, plays a key role in the microtubule organization and meiotic cell cycle progression in oocytes, fertilized eggs, and early embryos (By similarity). Interacts with ESRRA promoting its re-localization from the nucleus to the cytoplasm and then prevents its transcriptional activity (PubMed:21190936).

Cellular Location

Cytoplasm, cytoskeleton, cilium basal body. Cell junction, tight junction. Cytoplasm, cytoskeleton, microtubule organizing center, centrosome, centriole Cytoplasmic vesicle, autophagosome. Golgi apparatus. Nucleus. Cytoplasm. Cytoplasm, cytoskeleton, spindle {ECO:0000250|UniProtKB:Q80Y86}. Note=Co-localizes to the cytoplasm only in presence of ESRRA (PubMed:21190936) Translocates to the nucleus upon activation (PubMed:20638370). At prometaphase I, metaphase I (MI), anaphase I, telophase I, and metaphase II (MII) stages, is stably detected at the spindle (By similarity). {ECO:0000250|UniProtKB:Q80Y86, ECO:0000269|PubMed:20638370, ECO:0000269|PubMed:21190936}

Tissue Location

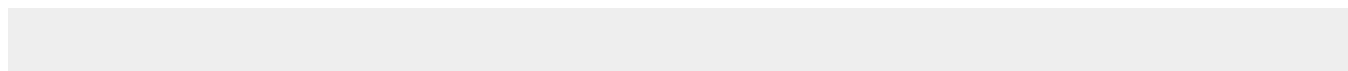
Widely expressed with a maximal expression in lung and kidney.

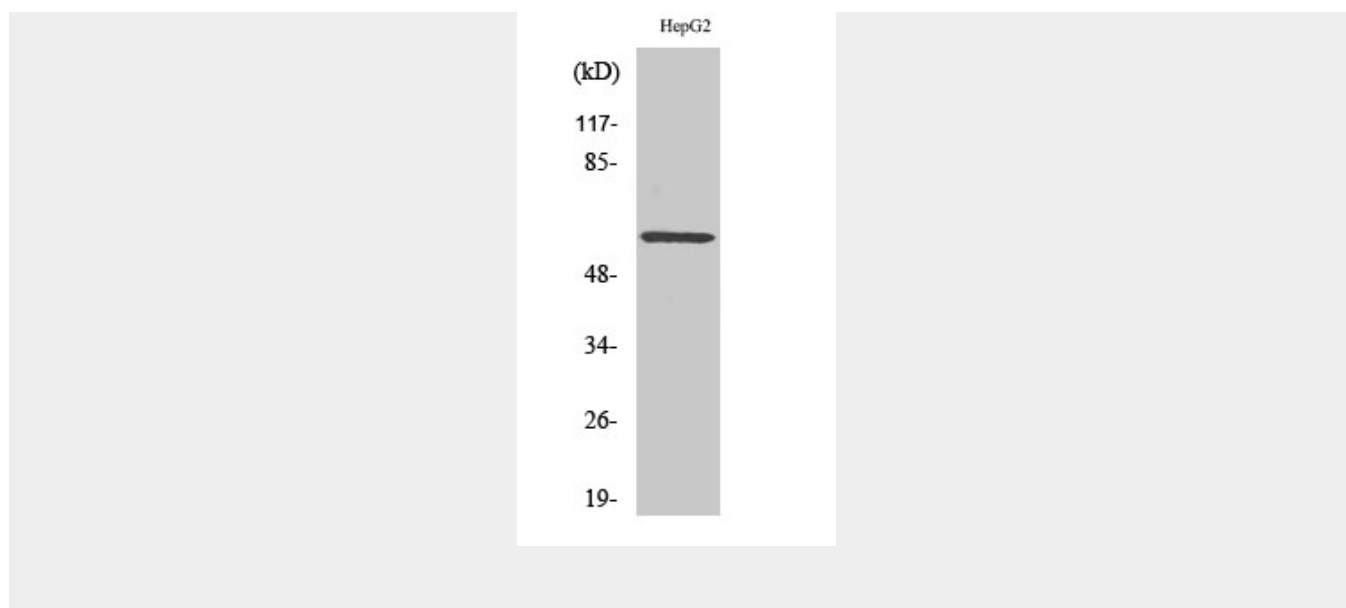
ERK 8 Polyclonal Antibody - Protocols

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](#)

ERK 8 Polyclonal Antibody - Images





ERK 8 Polyclonal Antibody - Background

Atypical MAPK protein that regulates several process such as autophagy, ciliogenesis, protein trafficking/secretion and genome integrity, in a kinase activity-dependent manner (PubMed:22948227, PubMed:24618899, PubMed:29021280, PubMed:21847093, PubMed:20733054). Controls both, basal and starvation-induced autophagy through its interaction with GABARAP, MAP1LC3B and GABARAPL1 leading to autophagosome formation, SQSTM1 degradation and reduced MAP1LC3B inhibitory phosphorylation (PubMed:22948227). Regulates primary cilium formation and the localization of ciliary proteins involved in cilium structure, transport, and signaling (PubMed:29021280). Prevents the relocation of the sugar-adding enzymes from the Golgi to the endoplasmic reticulum, thereby restricting the production of sugar-coated proteins (PubMed:24618899). Upon amino-acid starvation, mediates transitional endoplasmic reticulum site disassembly and inhibition of secretion (PubMed:21847093). Binds to chromatin leading to MAPK15 activation and interaction with PCNA, that which protects genomic integrity by inhibiting MDM2- mediated degradation of PCNA (PubMed:20733054). Regulates DA transporter (DAT) activity and protein expression via activation of RhoA (PubMed:28842414). In response to H₂O₂ treatment phosphorylates ELAVL1, thus preventing it from binding to the PDCD4 3'UTR and rendering the PDCD4 mRNA accessible to miR-21 and leading to its degradation and loss of protein expression (PubMed:26595526). Also functions in a kinase activity-independent manner as a negative regulator of growth (By similarity). Phosphorylates in vitro FOS and MBP (PubMed:11875070, PubMed:16484222, PubMed:20638370, PubMed:19166846). During oocyte maturation, plays a key role in the microtubule organization and meiotic cell cycle progression in oocytes, fertilized eggs, and early embryos (By similarity). Interacts with ESRRB promoting its re-localization from the nucleus to the cytoplasm and then prevents its transcriptional activity (PubMed:21190936).