

MEK-7 (phospho Ser271) Polyclonal Antibody
Catalog # AP67722**Specification****MEK-7 (phospho Ser271) Polyclonal Antibody - Product Information**

Application	WB, IHC-P
Primary Accession	O14733
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal

MEK-7 (phospho Ser271) Polyclonal Antibody - Additional Information**Gene ID** 5609**Other Names**

MAP2K7; JNKK2; MEK7; MKK7; PRKM7; SKK4; Dual specificity mitogen-activated protein kinase kinase 7; MAP kinase kinase 7; MAPKK 7; JNK-activating kinase 2; MAPK/ERK kinase 7; MEK 7; Stress-activated protein kinase kinase 4; SAPK kinase 4; S

Dilution

WB~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. ELISA: 1/20000. Not yet tested in other applications.
IHC-P~~N/A

Format

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

Storage Conditions

-20°C

MEK-7 (phospho Ser271) Polyclonal Antibody - Protein Information**Name** MAP2K7**Synonyms** JNKK2, MEK7, MKK7, PRKM7, SKK4**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 MAP2K4/MKK4, 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/MKK4 shows preference for phosphorylation of the Tyr residue and MAP2K7/MKK7 for the Thr residue. The monophosphorylation of JNKs on the Thr residue is sufficient to increase JNK activity indicating that MAP2K7/MKK7 is important to trigger JNK activity, while the additional phosphorylation of the Tyr residue by MAP2K4/MKK4 ensures optimal JNK activation. Has a specific

role in JNK signal transduction pathway activated by pro-inflammatory cytokines. The MKK/JNK signaling pathway is also involved in mitochondrial death signaling pathway, including the release cytochrome c, leading to apoptosis. Part of a non-canonical MAPK signaling pathway, composed of the upstream MAP3K12 kinase and downstream MAP kinases MAPK1/ERK2 and MAPK3/ERK1, that enhances the AP-1-mediated transcription of APP in response to APOE (PubMed:28111074).

Cellular Location

Nucleus. Cytoplasm.

Tissue Location

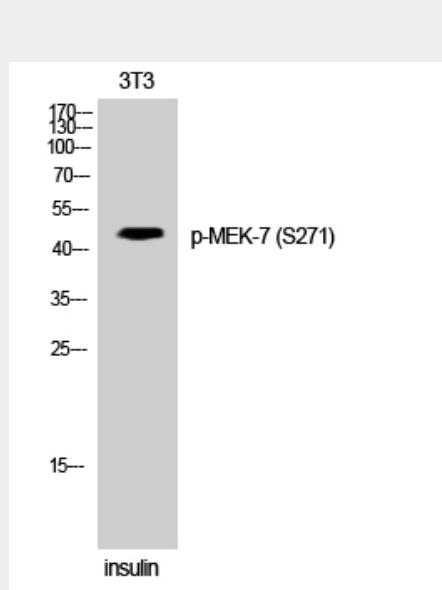
Ubiquitous; with highest level of expression in skeletal muscle. Isoform 3 is found at low levels in placenta, fetal liver, and skeletal muscle.

MEK-7 (phospho Ser271) 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](#)

MEK-7 (phospho Ser271) Polyclonal Antibody - Images



Western Blot analysis of 3T3 cells using Phospho-MEK-7 (S271) Polyclonal Antibody

MEK-7 (phospho Ser271) Polyclonal Antibody - Background

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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/MKK4 shows preference for phosphorylation of the Tyr residue and MAP2K7/MKK7 for the Thr residue. The monophosphorylation of JNKs on the Thr residue is sufficient to increase JNK activity indicating that MAP2K7/MKK7 is important to trigger JNK activity, while the additional phosphorylation of the Tyr residue by MAP2K4/MKK4 ensures optimal JNK activation. Has a specific role in JNK signal transduction pathway activated by proinflammatory cytokines. The MKK/JNK signaling pathway is also involved in mitochondrial death signaling pathway, including the release cytochrome c, leading to apoptosis. Part of a non-canonical MAPK signaling pathway, composed of the upstream MAP3K12 kinase and downstream MAP kinases MAPK1/ERK2 and MAPK3/ERK1, that enhances the AP-1-mediated transcription of APP in response to APOE (PubMed:28111074).