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Phospho-MAP4K1(S171) Antibody

Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP3451a

Product Information

Application	WB, E
Primary Accession	Q92918
Other Accession	P70218
Reactivity	Human
Predicted	Mouse, Rat, Bovine, Canine, Chicken
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB12751
Calculated MW	91296

Additional Information

Gene ID	11184
Other Names	Mitogen-activated protein kinase kinase kinase kinase 1, Hematopoietic progenitor kinase, MAPK/ERK kinase kinase kinase 1, MEK kinase kinase 1, MEKKK 1, MAP4K1, HPK1
Target/Specificity	This MAP4K1 Antibody is generated from rabbits immunized with a KLH conjugated synthetic phosphopeptide corresponding to amino acid residues surrounding S171 of human MAP4K1.
Dilution	WB~~1:1000 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.05% (V/V) Proclin 300. 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	Phospho-MAP4K1(S171) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	MAP4K1 (HGNC:6863)
Synonyms	HPK1

Function	Serine/threonine-protein kinase, which plays a role in the response to environmental stress (PubMed:24362026). Appears to act upstream of the JUN N-terminal pathway (PubMed:8824585). Activator of the Hippo signaling pathway which plays a pivotal role in organ size control and tumor suppression by restricting proliferation and promoting apoptosis. MAP4Ks act in parallel to and are partially redundant with STK3/MST2 and STK4/MST2 in the phosphorylation and activation of LATS1/2, and establish MAP4Ks as components of the expanded Hippo pathway (PubMed:26437443). May play a role in hematopoietic lineage decisions and growth regulation (PubMed:24362026, PubMed:8824585). Together with CLNK, it enhances CD3-triggered activation of T-cells and subsequent IL2 production (By similarity).
Tissue Location	Expressed primarily in hematopoietic organs, including bone marrow, spleen and thymus. Also expressed at very low levels in lung, kidney, mammary glands and small intestine

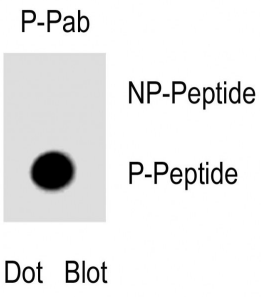
Background

The c-Jun amino-terminal kinases (JNKs)/stress-activated protein kinases (SAPKs) play a crucial role in stress responses in mammalian cells. The mechanism underlying this pathway in the hematopoietic system is unclear, but it is a key in understanding the molecular basis of blood cell differentiation. We have cloned a novel protein kinase, termed hematopoietic progenitor kinase 1 (HPK1), that is expressed predominantly in hematopoietic cells, including early progenitor cells. HPK1 is related distantly to the p21(Cdc42/Rac1)-activated kinase (PAK) and yeast STE20 implicated in the mitogen-activated protein kinase (MAPK) cascade. Expression of HPK1 activates JNK1 specifically, and it elevates strongly AP-1-mediated transcriptional activity in vivo. HPK1 binds and phosphorylates MEKK1 directly, whereas JNK1 activation by HPK1 is inhibited by a dominant-negative MEKK1 or MKK4/SEK mutant. Interestingly, unlike PAK65, HPK1 does not contain the small GTPase Rac1/Cdc42-binding domain and does not bind to either Rac1 or Cdc42, suggesting that HPK1. activation is Rac1/Cdc42-independent. These results indicate that HPK1 is a novel functional activator of the JNK/SAPK signaling pathway.

References

Hu M.C.-T., Genes Dev. 10:2251-2264(1996).
Beausoleil S.A., Proc. Natl. Acad. Sci. U.S.A. 101:12130-12135(2004).
Wissing J., Mol. Cell. Proteomics 6:537-547(2007).

Images



Dot blot analysis of Phospho-MAP4K1(S171) specific Pab (Cat. AP3451a) on nitrocellulose membrane. 50ng of Phospho-peptide or Non Phospho-peptide per dot were adsorbed. Antobodies working concentration was 0. 5ug per ml.

Citations

- Molecular mechanisms controlling translation in a hibernator.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.