10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

PAK2 Antibody (N-term) Blocking Peptide
Synthetic peptide
Catalog # BP7927a

Specification

PAK2 Antibody (N-term) Blocking Peptide - Product Information

Primary Accession Q13177

PAK2 Antibody (N-term) Blocking Peptide - Additional Information

Gene ID 5062

Other Names
Serine/threonine-protein kinase PAK 2, Gamma-PAK, PAK65, S6/H4 kinase, p21-activated kinase 2, PAK-2, p58, PAK-2p27, p27, PAK-2p34, p34, C-t-PAK2, PAK2

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP7927a was selected from the N-term region of human PAK2 . A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

PAK2 Antibody (N-term) Blocking Peptide - Protein Information

Name PAK2

Function
Serine/threonine protein kinase that plays a role in a variety of different signaling pathways including cytoskeleton regulation, cell motility, cell cycle progression, apoptosis or proliferation (PubMed:7744004, PubMed:19273597, PubMed:19923322, PubMed:9171063, PubMed:12853446, PubMed:16617111, PubMed:33693784). Acts as a downstream effector of the small GTPases CDC42 and RAC1 (PubMed:7744004). Activation by

the binding of active CDC42 and RAC1 results in a conformational change and a subsequent autophosphorylation on several serine and/or threonine residues (PubMed:7744004). Full- length PAK2 stimulates cell survival and cell growth (PubMed:7744004). Phosphorylates MAPK4 and MAPK6 and activates the downstream target MAPKAPK5, a regulator of F-actin polymerization and cell migration (PubMed:21317288). Phosphorylates JUN and plays an important role in EGF-induced cell proliferation (PubMed:21177766). Phosphorylates many other substrates including histone H4 to promote assembly of H3.3 and H4 into nucleosomes, BAD, ribosomal protein S6, or MBP (PubMed:21724829). Phosphorylates CASP7, thereby preventing its activity (PubMed:21555521, PubMed:27889207). Additionally, associates with ARHGEF7 and GIT1 to perform kinase-independent functions such as spindle orientation control during mitosis (PubMed:19273597, PubMed:19923322). On the other hand, apoptotic stimuli such as DNA damage lead to caspase-mediated cleavage of PAK2, generating PAK-2p34, an active p34 fragment that translocates to the nucleus and promotes cellular apoptosis involving the JNK signaling pathway (PubMed:9171063, PubMed:12853446, PubMed:16617111). Caspase-activated PAK2 phosphorylates MKNK1 and reduces cellular translation (PubMed:15234964).

Cellular Location
[Serine/threonine-protein kinase PAK 2]: Cytoplasm Nucleus Note=MYO18A mediates the cellular distribution of the PAK2-ARHGEF7-GIT1 complex to the inner surface of the cell membrane

Tissue Location
Ubiquitously expressed. Higher levels seen in skeletal muscle, ovary, thymus and spleen

PAK2 Antibody (N-term) Blocking Peptide - Protocols

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

- Blocking Peptides

PAK2 Antibody (N-term) Blocking Peptide - Images

PAK2 Antibody (N-term) Blocking Peptide - Background

PAK2, a member of the STE20 subfamily of Ser/Thr protein kinases, acts on a variety of targets. It phosphorylates ribosomal protein S6, histone H4 and myelin basic protein. PAK2 interacts tightly with GTP-bound but not GDP-bound CDC42/p21 and RAC1. Expression is ubiquitous, with higher levels seen in skeletal muscle, ovary, thymus and spleen. PAK2 is autophosphorylated when activated by CDC42/p21. The protein structure contains 1 CRIB domain.

PAK2 Antibody (N-term) Blocking Peptide - References

Benner, G.E., et al., J. Biol. Chem. 270(36):21121-21128 (1995).Martin, G.A., et al., EMBO J. 14(9):1970-1978 (1995).Martin, G.A., et al., EMBO J. 14 (17), 4385 (1995).