

PEK/PERK Polyclonal Antibody
Catalog # AP74308**Specification****PEK/PERK Polyclonal Antibody - Product Information**

Application	WB
Primary Accession	Q9NZJ5
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal

PEK/PERK Polyclonal Antibody - Additional Information**Gene ID** 9451**Other Names**

Eukaryotic translation initiation factor 2-alpha kinase 3 (EC 2.7.11.1) (PRKR-like endoplasmic reticulum kinase) (Pancreatic eIF2-alpha kinase) (HsPEK)

Dilution

WB~~WB 1:500-2000, ELISA 1:10000-20000

Format

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

Storage Conditions

-20°C

PEK/PERK Polyclonal Antibody - Protein Information**Name** EIF2AK3 {ECO:0000303|PubMed:10932183, ECO:0000312|HGNC:HGNC:3255}**Function**

Metabolic-stress sensing protein kinase that phosphorylates the alpha subunit of eukaryotic translation initiation factor 2 (EIF2S1/eIF-2-alpha) in response to various stress, such as unfolded protein response (UPR) (PubMed:10026192, PubMed:10677345, PubMed:11907036, PubMed:12086964, PubMed:25925385, PubMed:31023583). Key effector of the integrated stress response (ISR) to unfolded proteins: EIF2AK3/PERK specifically recognizes and binds misfolded proteins, leading to its activation and EIF2S1/eIF-2-alpha phosphorylation (PubMed:10677345, PubMed:27917829, PubMed:31023583). EIF2S1/eIF-2-alpha phosphorylation in response to stress converts EIF2S1/eIF-2-alpha in a global

protein synthesis inhibitor, leading to a global attenuation of cap-dependent translation, while concomitantly initiating the preferential translation of ISR-specific mRNAs, such as the transcriptional activators ATF4 and QRICH1, and hence allowing ATF4- and QRICH1-mediated reprogramming (PubMed:10026192, PubMed:10677345, PubMed:31023583, PubMed:33384352). The EIF2AK3/PERK- mediated unfolded protein response increases mitochondrial oxidative phosphorylation by promoting ATF4-mediated expression of COX7A2L/SCAF1, thereby increasing formation of respiratory chain supercomplexes (PubMed:31023583). In contrast to most subcellular compartments, mitochondria are protected from the EIF2AK3/PERK-mediated unfolded protein response due to EIF2AK3/PERK inhibition by ATAD3A at mitochondria-endoplasmic reticulum contact sites (PubMed:39116259). In addition to EIF2S1/eIF-2-alpha, also phosphorylates NFE2L2/NRF2 in response to stress, promoting release of NFE2L2/NRF2 from the BCR(KEAP1) complex, leading to nuclear accumulation and activation of NFE2L2/NRF2 (By similarity). Serves as a critical effector of unfolded protein response (UPR)-induced G1 growth arrest due to the loss of cyclin-D1 (CCND1) (By similarity). Involved in control of mitochondrial morphology and function (By similarity).

Cellular Location

Endoplasmic reticulum membrane {ECO:0000250|UniProtKB:Q9Z2B5}; Single-pass type I membrane protein. Note=Localizes to the Localizes to endoplasmic reticulum membrane (By similarity). Also present at mitochondria-endoplasmic reticulum contact sites; where it interacts with ATAD3A (PubMed:39116259). {ECO:0000250|UniProtKB:Q9Z2B5, ECO:0000269|PubMed:39116259}

Tissue Location

Ubiquitous. A high level expression is seen in secretory tissues.

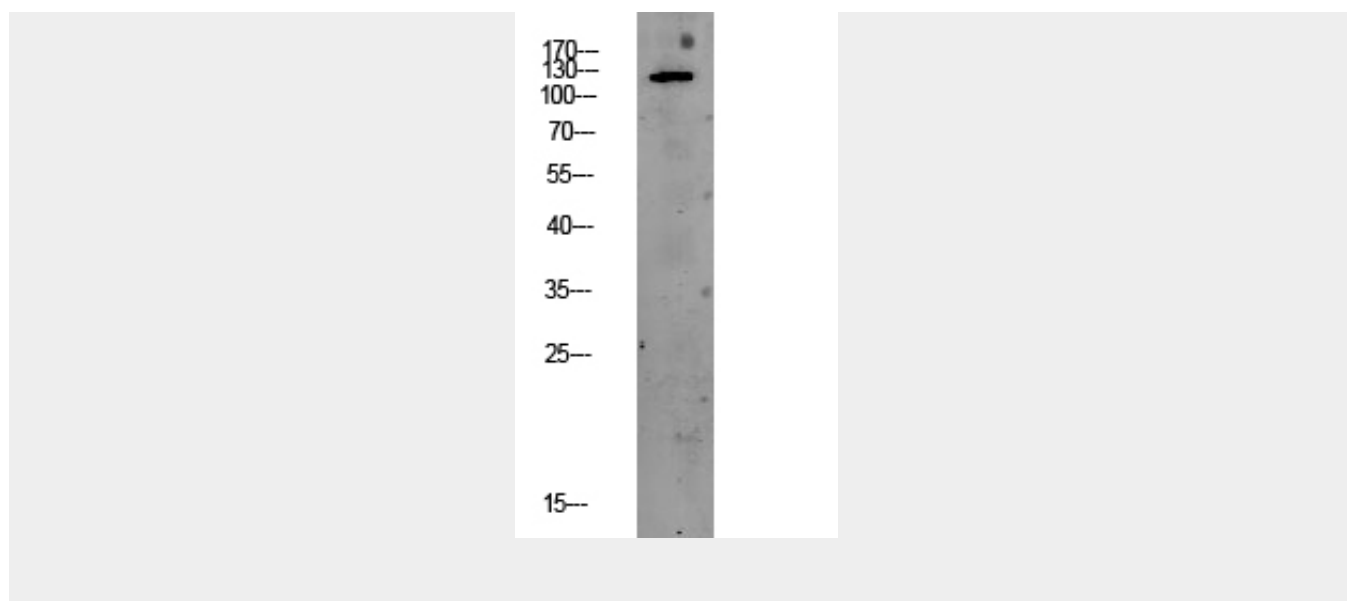
PEK/PERK Polyclonal Antibody - Protocols

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](#)

PEK/PERK Polyclonal Antibody - Images



**PEK/PERK Polyclonal Antibody - Background**

Metabolic-stress sensing protein kinase that phosphorylates the alpha subunit of eukaryotic translation initiation factor 2 (eIF-2-alpha/EIF2S1) on 'Ser-52' during the unfolded protein response (UPR) and in response to low amino acid availability. Converts phosphorylated eIF-2-alpha/EIF2S1 either in a global protein synthesis inhibitor, leading to a reduced overall utilization of amino acids, or to a translation initiation activator of specific mRNAs, such as the transcriptional activator ATF4, and hence allowing ATF4-mediated reprogramming of amino acid biosynthetic gene expression to alleviate nutrient depletion. Serves as a critical effector of unfolded protein response (UPR)- induced G1 growth arrest due to the loss of cyclin-D1 (CCND1). Involved in control of mitochondrial morphology and function.