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PERK Antibody
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP51183

Specification

PERK Antibody - Product Information

Application	**WB, IP, ICC, IHC-P, E**
Primary Accession	Q9NZJ5
Reactivity	**Human**
Host	**Rabbit**
Clonality	**Polyclonal**
Calculated MW	**140 KDa**

PERK Antibody - Additional Information

Gene ID 9451

Other Names
Eukaryotic translation initiation factor 2-alpha kinase 3, PRKR-like endoplasmic reticulum kinase, Pancreatic eIF2-alpha kinase, HsPEK, EIF2AK3, PEK, PERK

Dilution
WB~~1:1000
IP~~N/A
ICC~~N/A
IHC-P~~N/A
E~~N/A

Format
0.01M PBS, pH 7.2, 0.09% (W/V) Sodium azide, Glycerol 50%

Storage
Store at -20 °C.Stable for 12 months from date of receipt

PERK 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.

PERK 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 Cytomety
- Cell Culture

PERK Antibody - Images

PERK Antibody - Background

Phosphorylates the alpha subunit of eukaryotic translation-initiation factor 2 (EIF2), leading to its inactivation and thus to a rapid reduction of translational initiation and repression of global protein

synthesis. Serves as a critical effector of unfolded protein response (UPR)-induced G1 growth arrest due to the loss of cyclin-D1 (CCND1) (By similarity).

PERK Antibody - References

Shi Y.,et al.J. Biol. Chem. 274:5723-5730(1999).
Sood R.,et al.Biochem. J. 346:281-293(2000).
Delepine M.,et al.Nat. Genet. 25:406-409(2000).
Ota T.,et al.Nat. Genet. 36:40-45(2004).
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