

PERK Antibody (N-term Q163)
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP8054A**Specification**

PERK Antibody (N-term Q163) - Product Information

Application	IHC-P, WB,E
Primary Accession	Q9NZJ5
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Antigen Region	148-175

PERK Antibody (N-term Q163) - 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

Target/Specificity

This PERK antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 148-175 amino acids from the N-terminal region of human PERK.

Dilution

IHC-P~~1:50~100

WB~~1:1000

E~~Use at an assay dependent concentration.

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.

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

PERK Antibody (N-term Q163) is for research use only and not for use in diagnostic or therapeutic procedures.

PERK Antibody (N-term Q163) - 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 QRIH1, and hence allowing ATF4- and QRIH1-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.

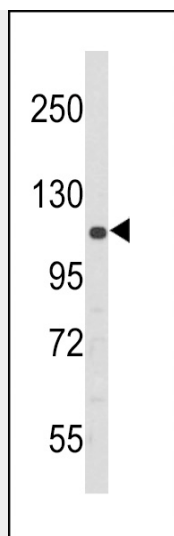
PERK Antibody (N-term Q163) - Protocols

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

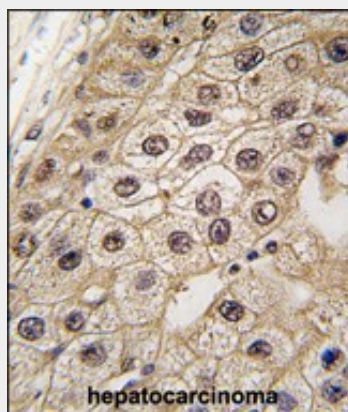
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

PERK Antibody (N-term Q163) - Images





Western blot analysis of PERK Antibody (N-term Q163) (Cat. #AP8054a) in 293 cell line lysates (35ug/lane). PERK (arrow) was detected using the purified Pab.



Formalin-fixed and paraffin-embedded human hepatocarcinoma tissue reacted with PERK antibody (N-term Q163) (Cat.#AP8054a), which was peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated.

PERK Antibody (N-term Q163) - Background

PERK, a member of the GCN2 subfamily of Ser/Thr protein kinases, 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. It likely serves as a critical effector of unfolded protein response (UPR)-induced G1 growth arrest due to the loss of cyclin D1. Perturbation in protein folding in the endoplasmic reticulum (ER) promotes reversible dissociation from HSPA5/BIP and oligomerization, resulting in transautophosphorylation and kinase activity induction. Expression of this Type I membrane protein is ubiquitous, with highest levels seen in secretory tissues. Defects in EIF2AK3 are the cause of Wolcott-Rallison syndrome (WRS), also known as multiple epiphyseal dysplasia with early-onset diabetes mellitus. WRS is a rare autosomal recessive disorder, characterized by permanent neonatal or early infancy insulin-dependent diabetes and, at a later age, epiphyseal dysplasia, osteoporosis, growth retardation and other multisystem manifestations, such as hepatic and renal dysfunctions, mental retardation and cardiovascular abnormalities.

PERK Antibody (N-term Q163) - References

Delepine, M., et al., Nat. Genet. 25(4):406-409 (2000). Shi, Y., et al., J. Biol. Chem. 274(9):5723-5730 (1999). Sood, R., et al., Biochem. J. 346 Pt 2, 281-293 (2000).

PERK Antibody (N-term Q163) - Citations

- [Molecular mechanisms of the LPS-induced non-apoptotic ER stress-CHOP pathway.](#)