

# PERK Rabbit mAb

Catalog # AP75893

## Product Information

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<b>Application</b>	WB
<b>Primary Accession</b>	<a href="#">Q9NZJ5</a>
<b>Reactivity</b>	Rat, Human, Mouse
<b>Host</b>	Rabbit
<b>Clonality</b>	Monoclonal Antibody
<b>Isotype</b>	IgG
<b>Conjugate</b>	Unconjugated
<b>Purification</b>	Affinity Purified
<b>Calculated MW</b>	125216

## Additional Information

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<b>Gene ID</b>	9451
<b>Other Names</b>	EIF2AK3
<b>Dilution</b>	WB~~1:2000-1:10000
<b>Format</b>	1xPBS(pH 7.4), 150mM NaCl, 50% Glycerol, 0.02% Sodium azide and 0.05% BSA
<b>Storage</b>	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.

## Protein Information

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<b>Name</b>	EIF2AK3 {ECO:0000303   PubMed:10932183, ECO:0000312   HGNC:HGNC:3255}
<b>Function</b>	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: <a href="#">10026192</a> , PubMed: <a href="#">10677345</a> , PubMed: <a href="#">11907036</a> , PubMed: <a href="#">12086964</a> , PubMed: <a href="#">25925385</a> , PubMed: <a href="#">31023583</a> ). 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: <a href="#">10677345</a> , PubMed: <a href="#">27917829</a> , PubMed: <a href="#">31023583</a> ). 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: <a href="#">10026192</a> ,

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](#)}

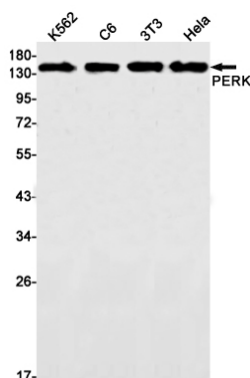
### Tissue Location

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

## 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.

## Images



Western blot analysis of PERK in K562, C6, 3T3, HeLa lysates using PERK antibody.

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