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PERK Rabbit mAb

Catalog # AP75893

Product Information

Application	WB
Primary Accession	Q9NZJ5
Reactivity	Rat, Human, Mouse
Host	Rabbit
Clonality	Monoclonal Antibody
Isotype	IgG
Conjugate	Unconjugated
Purification	Affinity Purified
Calculated MW	125216

Additional Information

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

Protein Information

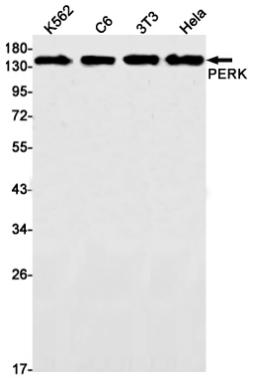
Name	EIF2AK3 {ECO:0000303|PubMed:10932183, ECO:0000312|HGNC:HGNC:3255}
Function	Metabolic-stress sensing protein kinase that phosphorylates the alpha subunit of eukaryotic translation initiation factor 2 (EIF2S1/eIF-2-alpha) in response to various stress, such as unfolded protein response (UPR) (PubMed:10026192, 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.

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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