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PERK (EIF2AK3) Blocking Peptide (Center)

Synthetic peptide
Catalog # BP8150c

Specification

PERK (EIF2AK3) Blocking Peptide (Center) - Product Information

Primary Accession	Q9NZJ5
Other Accession	Q9Z1Z1, Q9Z2B5

PERK (EIF2AK3) Blocking Peptide (Center) - 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
The synthetic peptide sequence is selected from aa 482~496 of HUMAN EIF2AK3

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

PERK (EIF2AK3) Blocking Peptide (Center) - 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:<a

href="http://www.uniprot.org/citations/31023583" target="_blank">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 (EIF2AK3) Blocking Peptide (Center) - Protocols

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

- Blocking Peptides

PERK (EIF2AK3) Blocking Peptide (Center) - Images

PERK (EIF2AK3) Blocking Peptide (Center) - Background

EIF2AK3, 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. This protein serves as a critical effector of unfolded protein response (UPR)-induced G1 growth arrest due to the loss of cyclin D1. It is proposed that 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 a high level expression 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.

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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).