10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

PERK (EIF2AK3) Antibody (C-term) Blocking peptide

Synthetic peptide
Catalog # BP8150b

Specification

PERK (EIF2AK3) Antibody (C-term) Blocking peptide - Product Information

Primary Accession Q9NZJ5

PERK (EIF2AK3) Antibody (C-term) Blocking peptide - 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 used to generate the antibody AP8150b was selected from the C-term region of human EIF2AK3 . A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

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) Antibody (C-term) Blocking peptide - Protein Information

Name EIF2AK3

Synonyms PEK, PERK

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 conditions. Key activator of the integrated stress response (ISR) required for adaptation to various stress, such as unfolded protein response (UPR) and low amino acid availability (By similarity). 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:33384352). 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 (By similarity).

Cellular Location
Endoplasmic reticulum membrane; Single-pass type I membrane protein

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

PERK (EIF2AK3) Antibody (C-term) Blocking peptide - Protocols

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

- Blocking Peptides

PERK (EIF2AK3) Antibody (C-term) Blocking peptide - Images

PERK (EIF2AK3) Antibody (C-term) Blocking peptide - 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.

PERK (EIF2AK3) Antibody (C-term) Blocking peptide - 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).