

EIF2AK3 Antibody (N-term)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP14551A**Specification**

EIF2AK3 Antibody (N-term) - Product Information

Application	WB, IHC-P,E
Primary Accession	O9NZJ5
Other Accession	NP_004827.4
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	125216
Antigen Region	73-102

EIF2AK3 Antibody (N-term) - 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 EIF2AK3 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 73-102 amino acids from the N-terminal region of human EIF2AK3.

Dilution

WB~~1:1000

IHC-P~~1:10~50

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

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

EIF2AK3 Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

EIF2AK3 Antibody (N-term) - 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 QRIH1, and hence allowing ATF4- and QRIH1-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

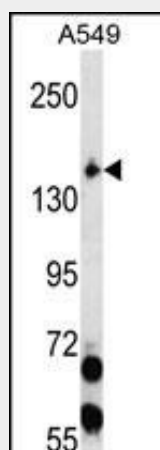
Tissue Location

Ubiquitous. A high level expression is seen in secretory tissues

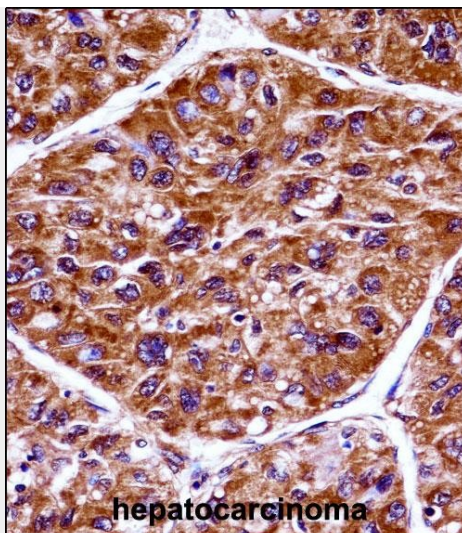
EIF2AK3 Antibody (N-term) - Protocols

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](#)

EIF2AK3 Antibody (N-term) - Images

EIF2AK3 Antibody (N-term) (Cat. #AP14551a) western blot analysis in A549 cell line lysates (35ug/lane). This demonstrates the EIF2AK3 antibody detected the EIF2AK3 protein (arrow).



EIF2AK3 Antibody (N-term) (AP14551a) immunohistochemistry analysis in formalin fixed and paraffin embedded human hepatocarcinoma followed by peroxidase conjugation of the secondary antibody and DAB staining. This data demonstrates the use of EIF2AK3 Antibody (N-term) for immunohistochemistry. Clinical relevance has not been evaluated.

EIF2AK3 Antibody (N-term) - Background

The protein encoded by this gene 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 is a type I membrane protein located in the endoplasmic reticulum (ER), where it is induced by ER stress caused by malformed proteins. Mutations in this gene are associated with Wolcott-Rallison syndrome.

EIF2AK3 Antibody (N-term) - References

Xu, H., et al. Toxicology 277 (1-3), 1-5 (2010) :
Bailey, S.D., et al. Diabetes Care 33(10):2250-2253(2010)
Rose, J.E., et al. Mol. Med. 16 (7-8), 247-253 (2010) :
Kim, K.W., et al. Oncogene 29(22):3241-3251(2010)
Lee do, Y., et al. PLoS ONE 5 (5), E10489 (2010) :