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HRI (EIF2AK1) Antibody (C-term) Blocking peptide

Synthetic peptide
Catalog # BP8114b

Specification

HRI (EIF2AK1) Antibody (C-term) Blocking peptide - Product Information

Primary Accession Q9BQI3

HRI (EIF2AK1) Antibody (C-term) Blocking peptide - Additional Information

Gene ID 27102

Other Names
Eukaryotic translation initiation factor 2-alpha kinase 1, Heme-controlled repressor, HCR, Heme-regulated eukaryotic initiation factor eIF-2-alpha kinase, Heme-regulated inhibitor, Hemin-sensitive initiation factor 2-alpha kinase, EIF2AK1, HRI, KIAA1369

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP8114b was selected from the C-term region of human EIF2AK1. 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.

HRI (EIF2AK1) Antibody (C-term) Blocking peptide - Protein Information

Name EIF2AK1 (HGNC:24921)

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 (PubMed:32132706, PubMed:32132707, PubMed:37327776). Key activator of the integrated stress response (ISR) required for adaptation to various stress, such as heme deficiency, oxidative stress, osmotic shock, mitochondrial dysfunction and heat shock (PubMed:32132706, PubMed:32132707, PubMed:37327776).

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 activator ATF4, and hence allowing ATF4-mediated reprogramming (PubMed:32132706, PubMed:32132707, PubMed:37327776). Acts as a key sensor of heme-deficiency: in normal conditions, binds hemin via a cysteine thiolate and histidine nitrogenous coordination, leading to inhibit the protein kinase activity (By similarity). This binding occurs with moderate affinity, allowing it to sense the heme concentration within the cell: heme depletion relieves inhibition and stimulates kinase activity, activating the ISR (By similarity). Thanks to this unique heme-sensing capacity, plays a crucial role to shut off protein synthesis during acute heme-deficient conditions (By similarity). In red blood cells (RBCs), controls hemoglobin synthesis ensuring a coordinated regulation of the synthesis of its heme and globin moieties (By similarity). It thereby plays an essential protective role for RBC survival in anemias of iron deficiency (By similarity). Iron deficiency also triggers activation by full-length DELE1 (PubMed:37327776). Also activates the ISR in response to mitochondrial dysfunction: HRI/EIF2AK1 protein kinase activity is activated upon binding to the processed form of DELE1 (S-DELE1), thereby promoting the ATF4-mediated reprogramming (PubMed:32132706, PubMed:32132707).

HRI (EIF2AK1) Antibody (C-term) Blocking peptide - Protocols

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

- Blocking Peptides

HRI (EIF2AK1) Antibody (C-term) Blocking peptide - Images

HRI (EIF2AK1) Antibody (C-term) Blocking peptide - Background

Eukaryotic translation initiation factor 2-alpha kinase 1 (EIF2AK1) belongs to the protein kinase superfamily, Ser/Thr protein kinase family, GCN2 subfamily. It mediates down-regulation of protein synthesis in response to various stress conditions by the phosphorylation of EIF2S1 at Ser-48 and Ser-51. Hemin inactivates EIF2AK1 by promoting the formation of a disulfide-linked homodimer. The homodimer is non-covalently bound in the absence of hemin, and converted to an inactive disulfide linked homodimer in the presence of hemin. Binding of nitric oxide (NO) to the heme iron in the N-terminal heme-binding domain activates the kinase activity, while binding of carbon monoxide (CO) suppresses kinase activity. EIF2AK1 is activated by autophosphorylation; phosphorylated predominantly on serine and threonine residues, but also on tyrosine residues.

HRI (EIF2AK1) Antibody (C-term) Blocking peptide - References

Hwang S.-Y., Mol. Cells 10:584-591(2000).Omasa T., DNA Seq. 13:133-137(2002).