

AUH Antibody (C-term) Blocking peptide
Synthetic peptide
Catalog # BP10124b**Specification**

AUH Antibody (C-term) Blocking peptide - Product Information

Primary Accession [O13825](#)
Other Accession [NP_001689.1](#)

AUH Antibody (C-term) Blocking peptide - Additional Information

Gene ID 549

Other Names

Methylglutaconyl-CoA hydratase, mitochondrial, AU-specific RNA-binding enoyl-CoA hydratase, AU-binding protein/enoyl-CoA hydratase, AUH

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.

AUH Antibody (C-term) Blocking peptide - Protein Information**Name AUH****Function**

Catalyzes the fifth step in the leucine degradation pathway, the reversible hydration of 3-methylglutaconyl-CoA (3-MG-CoA) to 3-hydroxy-3-methylglutaryl-CoA (HMG-CoA) (PubMed: [11738050](http://www.uniprot.org/citations/11738050), PubMed: [12434311](http://www.uniprot.org/citations/12434311), PubMed: [12655555](http://www.uniprot.org/citations/12655555), PubMed: [16640564](http://www.uniprot.org/citations/16640564)). Can catalyze the reverse reaction but at a much lower rate in vitro (PubMed: [16640564](http://www.uniprot.org/citations/16640564)). HMG-CoA is then quickly degraded by another enzyme (such as HMG-CoA lyase) to give acetyl-CoA and acetoacetate (PubMed: [16640564](http://www.uniprot.org/citations/16640564)). Uses other substrates such as (2E)-glutaconyl-CoA efficiently in vitro, and to a lesser extent 3-methylcrotonyl-CoA (3-methyl-(2E)-butenoyl-CoA), crotonyl-CoA ((2E)-butenoyl-CoA) and 3-hydroxybutanoyl-CoA (the missing carboxylate reduces affinity to the active site) (PubMed: [16640564](http://www.uniprot.org/citations/16640564)). Originally it was identified as an RNA-binding protein as it binds to AU-rich elements (AREs) in vitro (PubMed: [7892223](http://www.uniprot.org/citations/7892223))

target="_blank">7892223). AREs direct rapid RNA degradation and mRNA deadenylation (PubMed:7892223). Might have itaconyl-CoA hydratase activity, converting itaconyl-CoA into citramalyl-CoA in the C5-dicarboxylate catabolism pathway (PubMed:29056341). The C5-dicarboxylate catabolism pathway is required to detoxify itaconate, an antimicrobial metabolite and immunomodulator produced by macrophages during certain infections, that can act as a vitamin B12-poisoning metabolite (PubMed:29056341).

Cellular Location

Mitochondrion {ECO:0000250|UniProtKB:Q9JLZ3}.

AUH Antibody (C-term) Blocking peptide - Protocols

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

- [Blocking Peptides](#)

AUH Antibody (C-term) Blocking peptide - Images

AUH Antibody (C-term) Blocking peptide - Background

The methylglutaconyl-CoA hydratase, mitochondrial protein binds to the AU-rich element (ARE), a common element found in the 3' UTR of rapidly decaying mRNA such as c-fos, c-myc and granulocyte/macrophage colony stimulating factor. ARE elements are involved in directing RNA to rapid degradation and deadenylation. AUH is also homologous to enol-CoA hydratase, an enzyme involved in fatty acid degradation, and has been shown to have intrinsic hydratase enzymatic activity. AUH is thus a bifunctional chimera between RNA binding and metabolic enzyme activity. A possible subcellular localization in the mitochondria has been demonstrated for the mouse homolog of this protein which shares 92% identity with the human protein. It has been suggested that AUH may have a novel role as a mitochondrial located AU-binding protein. Human AUH is expressed as a single mRNA species of 1.8 kb, and translated as a 40-kDa precursor protein which is subsequently processed to a 32-kDa mature form.

AUH Antibody (C-term) Blocking peptide - References

Kurimoto, K., et al. *Proteins* 75(2):360-372(2009)
Vieira, A.R., et al. *Genet. Med.* 10(9):668-674(2008)
Mack, M., et al. *FEBS J.* 273(9):2012-2022(2006)
Illsinger, S., et al. *Pediatr. Neurol.* 30(3):213-215(2004)
Ly, T.B., et al. *Hum. Mutat.* 21(4):401-407(2003)