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FEM1B Blocking Peptide (C-Term)

Synthetic peptide
Catalog # BP22084b

Specification

FEM1B Blocking Peptide (C-Term) - Product Information

Primary Accession	Q9UK73
Other Accession	Q9Z2G0, P0C6P7

FEM1B Blocking Peptide (C-Term) - Additional Information

Gene ID 10116

Other Names
Protein fem-1 homolog B, FEM1b, FEM1-beta, Fem-1-like death receptor-binding protein alpha, Fem-1-like in apoptotic pathway protein alpha, F1A-alpha, FEM1B, F1AA, KIAA0396

Target/Specificity
The synthetic peptide sequence is selected from aa 567-579 of HUMAN FEM1B

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.

FEM1B Blocking Peptide (C-Term) - Protein Information

Name FEM1B {ECO:0000303|PubMed:10623617, ECO:0000312|HGNC:HGNC:3649}

Function
Substrate-recognition component of a Cul2-RING (CRL2) E3 ubiquitin-protein ligase complex of the DesCEND (destruction via C-end degrons) pathway, which recognizes a C-degron located at the extreme C terminus of target proteins, leading to their ubiquitination and degradation (PubMed:29779948, PubMed:33398170, PubMed:33398168). The C-degron recognized by the DesCEND pathway is usually a motif of less than ten residues and can be present in full-length proteins, truncated proteins or proteolytically cleaved forms (PubMed:29779948, PubMed:33398170, PubMed:33398168). The CRL2(FEM1B) complex specifically recognizes proteins ending with -Gly-Leu-Asp-Arg, such as CDK5R1, leading to their ubiquitination and degradation (PubMed:<a

href="http://www.uniprot.org/citations/33398170" target="_blank">33398170, PubMed:33398168). Also acts as a regulator of the reductive stress response by mediating ubiquitination of reduced FNIP1: in response to reductive stress, the CRL2(FEM1B) complex specifically recognizes a conserved Cys degron in FNIP1 when this degron is reduced, leading to FNIP1 degradation and subsequent activation of mitochondria to recalibrate reactive oxygen species (ROS) (By similarity). Mechanistically, recognizes and binds reduced FNIP1 through two interface zinc ions, which act as a molecular glue that recruit reduced FNIP1 to FEM1B (By similarity). Promotes ubiquitination of GLI1, suppressing GLI1 transcriptional activator activity (PubMed:24076122). Promotes ubiquitination and degradation of ANKRD37 (By similarity). Promotes ubiquitination and degradation of SLBP (PubMed:28118078). Involved in apoptosis by acting as a death receptor-associated protein that mediates apoptosis (PubMed:10542291). Also involved in glucose homeostasis in pancreatic islet (By similarity). May also act as an adapter/mediator in replication stress-induced signaling that leads to the activation of CHEK1 (PubMed:19330022).

Cellular Location
Cytoplasm. Nucleus Note=In the nucleus, the protein level increased slightly after camptothecin (CPT) treatment (PubMed:19330022). Associated with chromatin (PubMed:19330022).

Tissue Location
Widely expressed (PubMed:10542291). Highly expressed in testis (PubMed:10542291). Weakly expressed in other tissues (PubMed:10542291).

FEM1B Blocking Peptide (C-Term) - Protocols

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

- Blocking Peptides

FEM1B Blocking Peptide (C-Term) - Images

FEM1B Blocking Peptide (C-Term) - Background

Component of an E3 ubiquitin-protein ligase complex, in which it may act as a substrate recognition subunit. Involved in apoptosis by acting as a death receptor-associated protein that mediates apoptosis. Also involved in glucose homeostasis in pancreatic islet. Functions as an adapter/mediator in replication stress-induced signaling that leads to the activation of CHEK1.

FEM1B Blocking Peptide (C-Term) - References

Chan S.-L.,et al.J. Biol. Chem. 274:32461-32468(1999).
Ventura-Holman T.,et al.Biochem. Biophys. Res. Commun. 267:317-320(2000).
Ishikawa K.,et al.DNA Res. 4:307-313(1997).
Nakajima D.,et al.DNA Res. 9:99-106(2002).
Ota T.,et al.Nat. Genet. 36:40-45(2004).