

HUS1 Antibody (C-term)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP16922b**Specification**

HUS1 Antibody (C-term) - Product Information

Application	WB,E
Primary Accession	O60921
Other Accession	NP_004498.1
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	31691
Antigen Region	199-226

HUS1 Antibody (C-term) - Additional Information**Gene ID** 3364**Other Names**

Checkpoint protein HUS1, hHUS1, HUS1

Target/Specificity

This HUS1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 199-226 amino acids from the C-terminal region of human HUS1.

Dilution

WB~~1:1000

E~~Use at an assay dependent concentration.

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

HUS1 Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

HUS1 Antibody (C-term) - Protein Information**Name** HUS1**Function** Component of the 9-1-1 cell-cycle checkpoint response complex that plays a major role

in DNA repair (PubMed:[21659603](#)). The 9-1-1 complex is recruited to DNA lesion upon damage by the RAD17-replication factor C (RFC) clamp loader complex (PubMed:[21659603](#)). Acts then as a sliding clamp platform on DNA for several proteins involved in long-patch base excision repair (LP-BER) (PubMed:[21659603](#)). The 9-1-1 complex stimulates DNA polymerase beta (POLB) activity by increasing its affinity for the 3'-OH end of the primer-template and stabilizes POLB to those sites where LP-BER proceeds; endonuclease FEN1 cleavage activity on substrates with double, nick, or gap flaps of distinct sequences and lengths; and DNA ligase I (LIG1) on long-patch base excision repair substrates (PubMed:[21659603](#)). The 9-1-1 complex is necessary for the recruitment of RHNO1 to sites of double-stranded breaks (DSB) occurring during the S phase (PubMed:[21659603](#)).

Cellular Location

Nucleus. Cytoplasm, cytosol. Note=In discrete nuclear foci upon DNA damage (PubMed:11077446). According to PubMed:11077446, localized also in the cytoplasm (PubMed:11077446). DNA damage induces its nuclear translocation (PubMed:11077446). Shuttles between the nucleus and the cytoplasm (PubMed:11077446).

Tissue Location

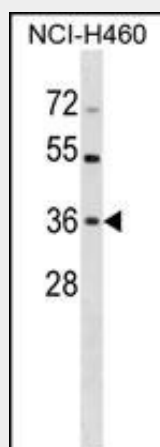
Ubiquitous..

HUS1 Antibody (C-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](#)

HUS1 Antibody (C-term) - Images



HUS1 Antibody (C-term) (Cat. #AP16922b) western blot analysis in NCI-H460 cell line lysates (35ug/lane). This demonstrates the HUS1 antibody detected the HUS1 protein (arrow).

HUS1 Antibody (C-term) - Background

The protein encoded by this gene is a component of an evolutionarily conserved, genotoxin-activated checkpoint complex that is involved in the cell cycle arrest in response to DNA damage. This protein forms a heterotrimeric complex with checkpoint proteins RAD9 and RAD1. In response to DNA damage, the trimeric complex interacts with another protein complex consisting of checkpoint protein RAD17 and four small subunits of the replication factor C (RFC), which loads the combined complex onto the chromatin. The DNA damage induced chromatin binding has been shown to depend on the activation of the checkpoint kinase ATM, and is thought to be an early checkpoint signaling event. [provided by RefSeq].

HUS1 Antibody (C-term) - References

Liu, C.Y., et al. Carcinogenesis 31(7):1259-1263(2010)
Takeishi, Y., et al. Genes Cells 15(7):761-771(2010)
Bai, H., et al. DNA Repair (Amst.) 9(5):478-487(2010)
Guey, L.T., et al. Eur. Urol. 57(2):283-292(2010)
Hosgood, H.D. III, et al. Respir Med 103(12):1866-1870(2009)