

FZR1 Antibody (N-term)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP19009a

Specification

FZR1 Antibody (N-term) - Product Information

Application	WB,E
Primary Accession	O9UM11
Other Accession	O9R1K5 , NP_057347.2
Reactivity	Human
Predicted	Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Antigen Region	53-81

FZR1 Antibody (N-term) - Additional Information

Gene ID 51343

Other Names

Fizzy-related protein homolog, Fzr, CDC20-like protein 1, Cdh1/Hct1 homolog, hCDH1, FZR1, CDH1, FYR, FZR, KIAA1242

Target/Specificity

This FZR1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 53-81 amino acids from the N-terminal region of human FZR1.

Dilution

WB~~1:1000

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

FZR1 Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

FZR1 Antibody (N-term) - Protein Information

Name FZR1 ([HGNC:24824](#))

Function Substrate-specific adapter for the anaphase promoting complex/cyclosome (APC/C) E3

ubiquitin-protein ligase complex. Associates with the APC/C in late mitosis, in replacement of CDC20, and activates the APC/C during anaphase and telophase. The APC/C remains active in degrading substrates to ensure that positive regulators of the cell cycle do not accumulate prematurely. At the G1/S transition FZR1 is phosphorylated, leading to its dissociation from the APC/C. Following DNA damage, it is required for the G2 DNA damage checkpoint: its dephosphorylation and reassociation with the APC/C leads to the ubiquitination of PLK1, preventing entry into mitosis. Acts as an adapter for APC/C to target the DNA-end resection factor RBBP8/CtIP for ubiquitination and subsequent proteasomal degradation. Through the regulation of RBBP8/CtIP protein turnover, may play a role in DNA damage response, favoring DNA double-strand repair through error-prone non-homologous end joining (NHEJ) over error-free, RBBP8-mediated homologous recombination (HR) (PubMed:[25349192](#)).

Cellular Location

[Isoform 2]: Nucleus

Tissue Location

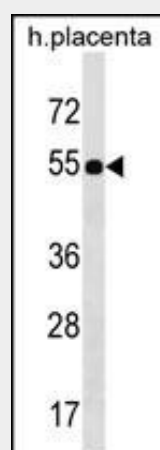
Isoform 2 is expressed at high levels in heart, liver, spleen and some cancer cell lines whereas isoform 3 is expressed only at low levels in these tissues.

FZR1 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](#)

FZR1 Antibody (N-term) - Images



FZR1 Antibody (N-term) (Cat. #AP19009a) western blot analysis in human placenta tissue lysates (35ug/lane). This demonstrates the FZR1 antibody detected the FZR1 protein (arrow).

FZR1 Antibody (N-term) - Background

Key regulator of ligase activity of the anaphase promoting complex/cyclosome (APC/C), which

confers substrate specificity upon the complex. Associates with the APC/C in late mitosis, in replacement of CDC20, and activates the APC/C during anaphase and telophase. The APC/C remains active in degrading substrates to ensure that positive regulators of the cell cycle do not accumulate prematurely. At the G1/S transition FZR1 is phosphorylated, leading to its dissociation from the APC/C. Following DNA damage, it is required for the G2 DNA damage checkpoint: its dephosphorylation and reassociation with the APC/C leads to the ubiquitination of PLK1, preventing entry into mitosis.

FZR1 Antibody (N-term) - References

- Olson, J.E., et al. Breast Cancer Res. Treat. 125(1):221-228(2011)
Colombo, S.L., et al. Proc. Natl. Acad. Sci. U.S.A. 107(44):18868-18873(2010)
Naoe, H., et al. Mol. Cell. Biol. 30(16):3994-4005(2010)
Sigl, R., et al. J. Cell. Sci. 122 (PT 22), 4208-4217 (2009) :
Bassermann, F., et al. Cell 134(2):256-267(2008)