

**FZR Antibody**  
**Purified Mouse Monoclonal Antibody (Mab)**  
**Catalog # AM8522b**

**Specification**

---

**FZR Antibody - Product Information**

Application	WB,E
Primary Accession	<a href="#">O9UM11</a>
Reactivity	Human
Host	Mouse
Clonality	monoclonal
Isotype	IgG1,k
Calculated MW	55179

**FZR Antibody - 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 FZR antibody is generated from a mouse immunized with a recombinant protein between 1-496 amino acids from human FZR.

**Dilution**

WB ~ 1:2000

**Format**

Purified monoclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein G column, followed by dialysis against PBS.

**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**

FZR Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

**FZR Antibody - 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

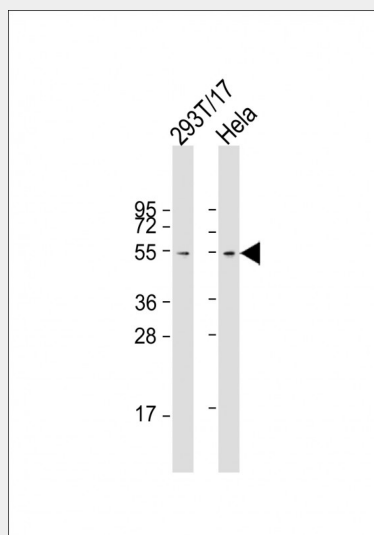
Isoform 2 is expressed at high levels in heart, liver, spleen and some cancer cell lines whereas isoform 3 is expressed only at low levels in these tissues.

## FZR Antibody - 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](#)

## FZR Antibody - Images



All lanes : Anti-FZR Antibody at 1:2000 dilution Lane 1: 293T/17 whole cell lysate Lane 2: HeLa whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-mouse IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 55 kDa Blocking/Dilution buffer: 5% NFD/MTBST.

## FZR Antibody - 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.

#### **FZR Antibody - References**

- Kramer E.R., et al. *Curr. Biol.* 8:1207-1210(1998).  
Kotani S., et al. Submitted (APR-1998) to the EMBL/GenBank/DDBJ databases.  
Sudo T., et al. Submitted (JUL-1998) to the EMBL/GenBank/DDBJ databases.  
Zhou Y., et al. *Biochem. J.* 374:349-358(2003).  
Nagase T., et al. *DNA Res.* 6:337-345(1999).