

FAS Antibody (C-term Y291)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP12467b

Specification

FAS Antibody (C-term Y291) - Product Information

Application	WB,E
Primary Accession	P25445
Other Accession	NP_690610.1 , NP_690611.1 , NP_000034.1
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	37732
Antigen Region	269-298

FAS Antibody (C-term Y291) - Additional Information

Gene ID 355

Other Names

Tumor necrosis factor receptor superfamily member 6, Apo-1 antigen, Apoptosis-mediating surface antigen FAS, FASLG receptor, CD95, FAS, APT1, FAS1, TNFRSF6

Target/Specificity

This FAS antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 269-298 amino acids from the C-terminal region of human FAS.

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

FAS Antibody (C-term Y291) is for research use only and not for use in diagnostic or therapeutic procedures.

FAS Antibody (C-term Y291) - Protein Information

Name FAS

Synonyms APT1, FAS1, TNFRSF6

Function Receptor for TNFSF6/FASLG. The adapter molecule FADD recruits caspase CASP8 to the activated receptor. The resulting death-inducing signaling complex (DISC) performs CASP8 proteolytic activation which initiates the subsequent cascade of caspases (aspartate-specific cysteine proteases) mediating apoptosis. FAS-mediated apoptosis may have a role in the induction of peripheral tolerance, in the antigen- stimulated suicide of mature T-cells, or both. The secreted isoforms 2 to 6 block apoptosis (in vitro).

Cellular Location

[Isoform 1]: Cell membrane; Single-pass type I membrane protein. Membrane raft [Isoform 3]: Secreted. [Isoform 5]: Secreted.

Tissue Location

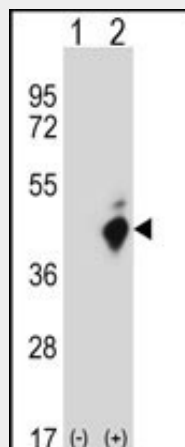
Isoform 1 and isoform 6 are expressed at equal levels in resting peripheral blood mononuclear cells. After activation there is an increase in isoform 1 and decrease in the levels of isoform 6.

FAS Antibody (C-term Y291) - 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](#)

FAS Antibody (C-term Y291) - Images



Western blot analysis of FAS (arrow) using rabbit polyclonal FAS Antibody (pY291) (Cat. #AP12467b). 293 cell lysates (2 ug/lane) either nontransfected (Lane 1) or transiently transfected (Lane 2) with the FAS gene.

FAS Antibody (C-term Y291) - Background

The protein encoded by this gene is a member of the TNF-receptor superfamily. This receptor contains a death domain. It has been shown to play a central role in the physiological regulation of programmed cell death, and has been implicated in the

pathogenesis of various malignancies and diseases of the immune system. The interaction of this receptor with its ligand allows the formation of a death-inducing signaling complex that includes Fas-associated death domain protein (FADD), caspase 8, and caspase 10. The autoproteolytic processing of the caspases in the complex triggers a downstream caspase cascade, and leads to apoptosis. This receptor has been also shown to activate NF-kappaB, MAPK3/ERK1, and MAPK8/JNK, and is found to be involved in transducing the proliferating signals in normal diploid fibroblast and T cells. At least eight alternatively spliced transcript variants have been described, some of which are candidates for nonsense-mediated decay (NMD). The isoforms lacking the transmembrane domain may negatively regulate the apoptosis mediated by the full length isoform.

FAS Antibody (C-term Y291) - References

- Cao, Y., et al. Mol. Carcinog. 49(11):944-950(2010)
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Gizinger, O.A., et al. Vopr Kurortol Fizioter Lech Fiz Kult 3, 29-31 (2010) :
Dubikov, A.I., et al. Scand. J. Rheumatol. 39(5):368-372(2010)
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