

Polyclonal Antibody to FAP-1



11175 Flintkote Ave., Suite E, San Diego, CA 92121
Tel: (858) 642-0978 Fax (858) 642-0937
Toll free: 1-888-723-GENE
E-mail: info@imgenex.com
web site: <http://www.imgenex.com>

Polyclonal Antibody to FAP-1

Catalog No : IMG-5726
Formulation : 50 ul neat serum containing 0.05% sodium azide.
Sodium azide is highly toxic.
Isotype : Rabbit Ig
Clone : N/A
Purification : Neat Serum
Species React : Human
Host : Rabbit

Application
Western blot analysis: 1:1000-1:2000
IHC (paraffin): 1:1000-1:5000
IHC (frozen): Users should optimize according to model and immunodetection system used (secondary reagents)
IP: 1:50-1:200
IF/ICC: 1:500-1:3000

Storage
Aliquot and store at -20°C. Avoid repeated freeze-thaw cycles.

Recommended Positive Control: spleen, lymphoid, many cancer cell lines

Background

FAP-1 (also known as Fas-associated phosphatase-1, PTPN13, PTP-BAS, hPTPIE, and PTPL1) is a member of the protein tyrosine phosphatase (PTP) family (reviewed in Meinhold-Heerlein et al, 2001; Savaskan et al, 2005; Foehr et al, 2005; and Ivanov et al, 2006). PTPs are enzymes that catalyze the removal of a phosphate group attached to a tyrosine residue. Most intracellular signaling involves reversible phosphorylation events; therefore, PTPs are central to the dynamic regulation of signaling cascades that underlie cell functions. For example, PTPs play key roles in regulating cell growth, differentiation, proliferation, inflammation, and oncogenic transformation. PTPs are emerging as a promising class of signaling targets for diseases such as cancer, neurodegeneration, diabetes, and inflammation. A key challenge is to identify specific PTPs that are involved in a disease process and develop therapeutics to modulate the PTP. FAP-1 phosphatase is thought to be important in the Fas signaling pathway. FAP-1 binds to the cytosolic tail of the Fas receptor (Apo1, CD95) and inhibits Fas-induced apoptosis. Increased FAP-1 protein levels in some tumor cell lines and tumor tissues correlates with resistance to Fas-mediated apoptosis. In general, FAP-1 expression has been found to be highest in cell lines and tissues that are relatively resistant to Fas-mediated apoptosis. Gene transfer-mediated elevations in FAP-1 partially abolished Fas-induced apoptosis in a T cell line which is consistent with an inhibitory effect of FAP-1 on Fas signal transductions. Additionally, FAP-1 expression correlates with Fas resistant in ovarian cancer cell lines and FAP-1 is commonly expression in ovarian cancers. Human Fas has a putative consensus tyrosine phosphorylation site (Tyrosine 275) suggesting that Fas surface expression or signaling may be regulated by phosphorylation. FAP-1 has been shown to directly bind to Fas and may dephosphorylate Fas as part of the down regulation of the apoptotic pathway. It is thought that development of therapeutics to inhibit FAP-1 may increase the ability of tumor cells with upregulated FAP-1 to undergo apoptosis. FAP-1 is a large ~270 kDa protein. Multiple alternatively spliced FAP-1 transcript variants which encode distinct proteins have been reported, including shorter forms. Please see Application Notes section for additional details on FAP-1 isoforms. IMG-5726 recognizes FAP-1, including FAP-1 isoforms listed in the Application Notes section.

Antigen

A recombinant protein corresponding to amino acids 1279 to 1883 of human FAP-1 protein was used as immunogen; GenBank no. [NP_542414.1](#). This peptide sequence is conserved in multiple isoforms of FAP-1. These FAP-1 isoforms are of varying amino acid (aa) lengths and include:

- (1) [NP_542414.1](#) = 2485 aa, FAP-1 isoform 1.
- (2) [BAD92141.1](#) = 2434 aa, FAP-1 isoform 2.
- (3) [NP_542415.1](#) = 2294 aa, FAP-1 isoform 3.
- (4) [NP_542416.1](#) = 2490 aa, FAP-1 isoform 4.
- (5) [AAH39610.1](#) = 604 aa, FAP-1.
- (6) [AAF63474.1](#) = 499 aa, FAP-1.

For additional information on FAP-1 isoforms, users are encouraged to use the NCBI data bases such as AceView (<http://www.ncbi.nlm.nih.gov/IEB/Research/AceView/>) or BLAST (<http://www.ncbi.nlm.nih.gov/BLAST/>).

Application Notes

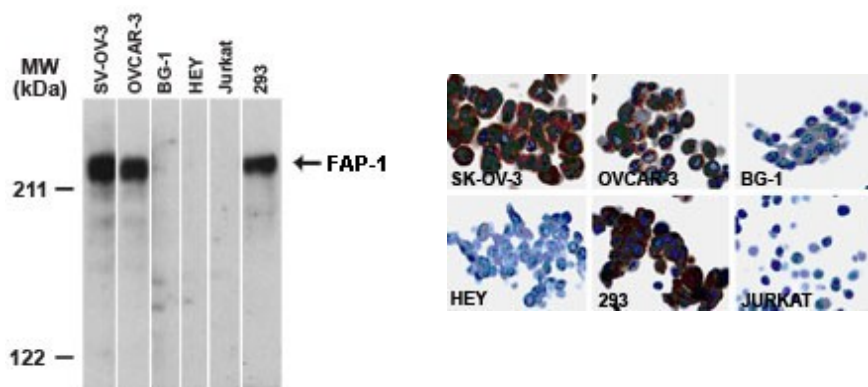
1. FAP-1 typically migrates at ~270 kDa on western blots. Thus for SDS-PAGE and western blot, the researcher should use techniques optimized for the transfer of proteins with high molecular weights.

2. The actual molecular weight of FAP-1 observed depends on the isoform expression. Isoforms of varying amino acid (aa) lengths have been described and denoted under antigen section.

Genebank Info (Protein)

NP_542416

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Western blot analysis of FAP-1 using IMG-5726 at 1:2000. In ovarian carcinoma cell lines FAP-1 expression was detected in SK-OV-3 and OVCAR-3, but not in BG-1 or HEY. Human Jurkat T and 293 kidney cell lines were used as negative and positive controls, respectively.

Immunocytochemical analysis of FAP-1 in cell lines using IMG-5726 at 1:2000. In ovarian carcinoma cell lines FAP-1 expression was detected in SK-OV-3 and OVCAR-3, but not in BG-1 or HEY. Human 293 kidney and Jurkat T cell lines were used as positive and negative controls, respectively. The staining data correlates with the western blot data (figure to the left).

Immunohistochemistry of FAP-1 in formalin-fixed, paraffin embedded ovarian carcinoma cores from a tissue microarray using IMG-5726 at 1:2000. A-D, samples are from four different patients. A1-D1 are high magnification images from A-D, respectively. Hematoxylin-eosin counterstain.

Reference

1. Sakaskan E, R Ravid, F Meier, F Muller-Spahn, and R Jockers. 2005. Immunohistochemical localization of Fas-associated phosphatase-1 (FAP-1) in Alzheimer Disease hippocampus. *Appl Immunohistochem Mol Morphol* 13:190-193.
2. Foehr ED, G Lorente, V Vincent, K Nikolich, and R Urfer. 2005. FAS associated phosphatase (FAP-1) blocks apoptosis of astrocytomas through dephosphorylation of FAS. *J Neuro-Oncology*. 74:241-248.
3. Ivanov VN, Z Ronai, and TK Hei. 2005. Opposite roles of FAP-1 and dynamin in the regulation of Fas (CD95) translocation of the cell surface and susceptibility to Fas ligand-mediated apoptosis. *J Biol Chem*. 281:1840-1852.

Product Citations

1. Expression and potential role of Fas-associated phosphatase-1 in ovarian cancer. *American J Pathol*. 158:1335-1344. Meinhold-Heerlein I, F Stenner-Liewen, H Liewen, S Kitada, M Krajewska, S Krajewski, JM Zapata, A Monks, DA Scudiero, T Bauknecht and JC Reed. 2001.

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Toll free: 1-888-723-4363

Fax: 1-858-642-0937

www.imgenex.com

info@imgenex.com

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