

FIP1L1 Polyclonal Antibody

Catalog Number:E-AB-61550

Note: Centrifuge before opening to ensure complete recovery of vial contents.

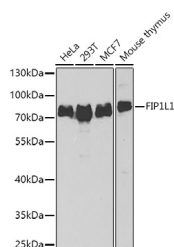
Description

Reactivity	Human,Mouse,Rat
Immunogen	Recombinant fusion protein of human FIP1L1 (NP_001128409.1).
Host	Rabbit
Isotype	IgG
Purification	Affinity purification
Conjugation	Unconjugated
Formulation	PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

Applications Recommended Dilution

WB	1:500-1:2000
IHC	1:50-1:200
IF	1:50-1:100

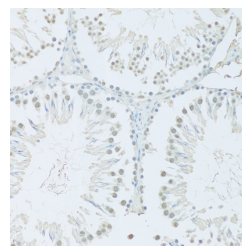
Data



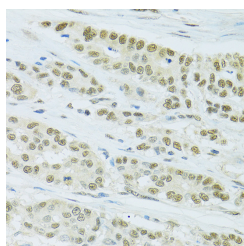
Western blot analysis of extracts of various cell lines using FIP1L1 Polyclonal Antibody at dilution of 1:1000.

Observed Mw:80kDa

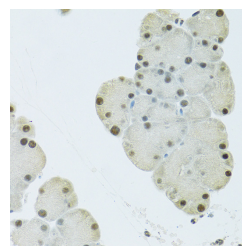
Calculated Mw:40kDa/58kDa/65kDa/66kDa



Immunohistochemistry of paraffin-embedded Rat testis using FIP1L1 Polyclonal Antibody at dilution of 1:100 (20x lens).



Immunohistochemistry of paraffin-embedded Human colon carcinoma using FIP1L1 Polyclonal Antibody at dilution of 1:100 (40x lens).



Immunohistochemistry of paraffin-embedded Mouse pancreas using FIP1L1 Polyclonal Antibody at dilution of 1:100 (40x lens).

For Research Use Only

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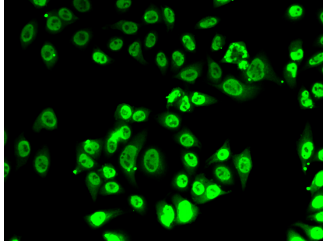
Tel: 1-832-243-6086

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Immunofluorescence analysis of A-549 cells using
FIP1L1 Polyclonal Antibody

Preparation & Storage

Storage Store at -20°C. Avoid freeze / thaw cycles.

Background

This gene encodes a subunit of the CPSF (cleavage and polyadenylation specificity factor) complex that polyadenylates the 3' end of mRNA precursors. This gene, the homolog of yeast Fip1 (factor interacting with PAP), binds to U-rich sequences of pre-mRNA and stimulates poly(A) polymerase activity. Its N-terminus contains a PAP-binding site and its C-terminus an RNA-binding domain. An interstitial chromosomal deletion on 4q12 creates an in-frame fusion of human genes FIP1L1 and PDGFRA (platelet-derived growth factor receptor, alpha). The FIP1L1-PDGFRA fusion gene encodes a constitutively activated tyrosine kinase that joins the first 233 amino acids of FIP1L1 to the last 523 amino acids of PDGFRA. This gene fusion and chromosomal deletion is the cause of some forms of idiopathic hypereosinophilic syndrome (HES). This syndrome, recently reclassified as chronic eosinophilic leukemia (CEL), is responsive to treatment with tyrosine kinase inhibitors. Alternative splicing results in multiple transcript variants encoding distinct isoforms.

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