

## FLIP (D5J1E) Rabbit mAb



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## For Research Use Only. Not for Use in Diagnostic Procedures.

W, IP	Reactivity: H M R	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 55, 25	<b>Source/Isotype:</b> Rabbit IgG	UniProt ID: #O15519	Entrez-Gene Id: 8837
Product Usage Information	•	<b>Application</b> Western Blotting Immunoprecipitation			<b>Dilution</b> 1:1000 1:100	
Storage		Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 $\mu$ g/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.				
Specificity/Sensitivity		FLIP (D5J1E) Rabbit mAb recognizes endogenous levels of total FLIP protein. This antibody recognizes both the long and short isoforms of FLIP.				
Source / Purification		Monoclonal antibody is produced by immunizing animals with recombinant protein specific to the amino terminus of human FLIP protein.				
Background		Cellular FLIP (FLICE inhibitory protein) is a regulator of apoptosis that has various names, such as c-FLIP (1), Casper (2), CLARP (3), FLAME (4), I-FLICE (5), MRIT (6), CASH (7), and Usurpin (8). FLIP is expressed as two alternative splice isoforms, FLIP short (FLIP <sub>S</sub> ) and FLIP long (FLIP <sub>L</sub> ). FLIP <sub>S</sub> contains two death effector domains (DEDs) like those found on the death receptor adaptor protein FADD and the prodomain of caspase-8. FLIP <sub>L</sub> shares significant homology with caspase-8 (FLICE), and contains an additional death effector domain, but FLIP <sub>L</sub> lacks the catalytic active site of the caspases and does not have protease activity. Both FLIP isoforms have been reported to interact with FADD and pro-caspase-8. The role of FLIP in apoptosis is controversial as some research studies have reported it to be antiapoptotic, while others claim that it is pro-apoptotic. Overexpression of FLIP <sub>L</sub> can lead to caspase-8 heterodimers that produce an active protease, resulting in apoptosis. However, at physiological levels, it is thought that the binding of FLIP to the DED of FADD results in inhibition of caspase-8 processing. Reduction of FLIP by siRNA or gene targeting sensitizes cells to death receptor-mediated apoptosis. FLIP has also been implicated in the resistance of cancer cells to apoptosis and is upregulated in some cancer types including Hodgkin's lymphoma and ovarian and colon carcinomas (9).				
		have protease activity The role of FLIP in apo apoptotic, while other heterodimers that pro it is thought that the b Reduction of FLIP by s FLIP has also been im	tor domain, but FLI . Both FLIP isoform optosis is controver is claim that it is pro- oduce an active pro- oinding of FLIP to the iRNA or gene targe plicated in the resis	P <sub>L</sub> lacks the catalytic act is have been reported to sial as some research sto p-apoptotic. Overexpress tease, resulting in apopt the DED of FADD results i ting sensitizes cells to d tance of cancer cells to	ive site of the caspa interact with FADD udies have reported sion of FLIP <sub>L</sub> can lea osis. However, at pl n inhibition of casp eath receptor-medi apoptosis and is up	ases and does not and pro-caspase-8. d it to be anti- ad to caspase-8 hysiological levels, ase-8 processing. iated apoptosis.

**Species Reactivity** 

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

**Western Blot Buffer** 

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

**Applications Key** 

W: Western Blotting IP: Immunoprecipitation

**Cross-Reactivity Key** 

H: Human M: Mouse R: Rat

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