Revision 4

Store at -20C

#8510

FLIP (D16A8) Rabbit mAb

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Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, IP	H M R	Endogenous	55	Rabbit IgG	#O15519	8837

Product Usage Information

Application	Dilution
Western Blotting	1:1000
Immunoprecipitation	1:50

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.

Specificity/Sensitivity

FLIP (D16A8) Rabbit mAb recognizes endogenous levels of total FLIP protein. The antibody detects a band of unknown origin at around 70 kDa.

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding His160 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 ($FLIP_S$) and FLIP long ($FLIP_L$). $FLIP_S$ contains two death effector domains (DEDs) like those found on the death receptor adaptor protein FADD and the pro-domain of caspase-8. $FLIP_L$ shares significant homology with caspase-8 (FLICE), and contains an additional death effector domain, but $FLIP_L$ 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 anti-apoptotic, while others claim that it is pro-apoptotic. Overexpression of $FLIP_L$ 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).

Background References

1. Irmler, M. et al. (1997) *Nature* 388, 190-5.
2. Shu, H.B. et al. (1997) *Immunity* 6, 751-63.
3. Inohara, N. et al. (1997) *Proc Natl Acad Sci U S A* 94, 10717-22.
4. Srinivasula, S.M. et al. (1997) *J Biol Chem* 272, 18542-5.
5. Hu, S. et al. (1997) *J Biol Chem* 272, 17255-7.
6. Han, D.K. et al. (1997) *Proc Natl Acad Sci U S A* 94, 11333-8.
7. Rasper, D.M. et al. (1998) *Cell Death Differ* 5, 271-88.
8. Goltsev, Y.V. et al. (1997) *J Biol Chem* 272, 19641-4.
9. Kataoka, T. (2005) *Crit Rev Immunol* 25, 31-58.

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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