Revision 8

Store at -20C

#95702

RIP3 (D4G2A) Rabbit mAb

Cell Signaling TECHNOLOGY®

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

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, W-S, IP, IF-IC, FC-FP	M	Endogenous	46-62	Rabbit IgG	#Q9QZL0	56532

Product Usage Information

Application	Dilution
Western Blotting	1:1000
Simple Western™	1:10 - 1:50
Immunoprecipitation	1:100
Immunofluorescence (Immunocytochemistry)	1:400 - 1:1600
Flow Cytometry (Fixed/Permeabilized)	1:800 - 1:1600

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.

For a carrier free (BSA and azide free) version of this product see product #74771.

Specificity/Sensitivity

RIP3 (D4G2A) Rabbit mAb recognizes endogenous levels of total RIP3 protein from mouse.

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Val370 of mouse RIP3 protein.

Background

The receptor-interacting protein (RIP) family of serine-threonine kinases (RIP, RIP2, RIP3, and RIP4) are important regulators of cellular stress that trigger pro-survival and inflammatory responses through the activation of NF-κB, as well as pro-apoptotic pathways (1). In addition to the kinase domain, RIP contains a death domain responsible for interaction with the death domain receptor Fas and recruitment to TNF-R1 through interaction with TRADD (2,3). RIP-deficient cells show a failure in TNF-mediated NF-κB activation, making the cells more sensitive to apoptosis (4,5). RIP also interacts with TNF-receptor-associated factors (TRAFs) and can recruit IKKs to the TNF-R1 signaling complex via interaction with NEMO, leading to IκB phosphorylation and degradation (6,7). Overexpression of RIP induces both NF-κB activation and apoptosis (2,3). Caspase-8-dependent cleavage of the RIP death domain can trigger the apoptotic activity of RIP (8).
Receptor-interacting protein 3 (RIP3) was originally found to interact with RIP and the TNF receptor complex to induce apoptosis and activation of NF-κB (9,10). It has subsequently been shown that the association between RIP and RIP3 is a key component of a signaling pathway that results in programmed necrosis (necroptosis), a necrotic-like cell death induced by TNF in the presence of caspase inhibitors (11-13). RIP3 is phosphorylated at Ser227 and targets the phosphorylation of mixed lineage kinase domain-like protein (MLKL), which is critical for necroptosis (14).

Background References

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3. Stanger, B.Z. et al. (1995) *Cell* 81, 513-23.
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5. Kelliher, M.A. et al. (1998) *Immunity* 8, 297-303.
6. Devin, A. et al. (2000) *Immunity* 12, 419-29.
7. Zhang, S.Q. et al. (2000) *Immunity* 12, 301-11.
8. Lin, Y. et al. (1999) *Genes Dev* 13, 2514-26.
9. Yu, P.W. et al. (1999) *Curr Biol* 9, 539-42.
10. Sun, X. et al. (1999) *J Biol Chem* 274, 16871-5.
11. Zhang, D.W. et al. (2009) *Science* 325, 332-6.
12. He, S. et al. (2009) *Cell* 137, 1100-11.
13. Cho, Y.S. et al. (2009) *Cell* 137, 1112-23.
14. Sun, L. et al. (2012) *Cell* 148, 213-27.

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 **W-S:** Simple Western™ **IP:** Immunoprecipitation **IF-IC:** Immunofluorescence (Immunocytochemistry) **FC-FP:** Flow Cytometry (Fixed/Permeabilized)

Cross-Reactivity Key

M: Mouse

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Limited Uses