

## RIP3 (D8J3L) Rabbit mAb



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

<b>Applications:</b> W, IP	<b>Reactivity:</b> M R	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 46-62	<b>Source/Isotype:</b> Rabbit IgG	UniProt ID: #Q9QZL0	Entrez-Gene Id: 56532
Product Usage Information		<b>Application</b> Western Blotting Immunoprecipitation	ı	<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		RIP3 (D8J3L) Rabbit mAb recognizes endogenous levels of total RIP3 protein from mouse and rat.				
Source / Purification		Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding His411 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		1. Meylan, E. and Tschopp, J. (2005) <i>Trends Biochem Sci</i> 30, 151-9.  2. Hsu, H. et al. (1996) <i>Immunity</i> 4, 387-96.  3. Stanger, B.Z. et al. (1995) <i>Cell</i> 81, 513-23.  4. Ting, A.T. et al. (1996) <i>EMBO J</i> 15, 6189-96.  5. Kelliher, M.A. et al. (1998) <i>Immunity</i> 8, 297-303.  6. Devin, A. et al. (2000) <i>Immunity</i> 12, 419-29.  7. Zhang, S.Q. et al. (2000) <i>Immunity</i> 12, 301-11.  8. Lin, Y. et al. (1999) <i>Genes Dev</i> 13, 2514-26.  9. Yu, P.W. et al. (1999) <i>Curr Biol</i> 9, 539-42.  10. Sun, X. et al. (1999) <i>J Biol Chem</i> 274, 16871-5.  11. Zhang, D.W. et al. (2009) <i>Science</i> 325, 332-6.  12. He, S. et al. (2009) <i>Cell</i> 137, 1100-11.  13. Cho, Y.S. et al. (2012) <i>Cell</i> 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 IP: Immunoprecipitation

**Cross-Reactivity Key** 

M: Mouse R: Rat

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