

Phospho-RIP3 (Thr231/Ser232) Antibody (Mouse Specific)



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Applications: W	Reactivity: M	Sensitivity: Endogenous	MW (kDa): 46-62	Source/Isotype: Rabbit	UniProt ID: #Q9QZL0	Entrez-Gene Id: 56532
Product Usage Information	9	Application Western Blotting			Dilution 1:1000	
Storage		Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 μg/ml BSA and 50% glycerol. Store at – 20°C. Do not aliquot the antibody.				
Specificity/Sensitivity		Phospho-RIP3 (Thr231/Ser232) Antibody (Mouse Specific) recognizes endogenous levels of RIP3 protein only when phosphorylated at Thr231/Ser232. This antibody may not react with single phosphorylation at either site.				
Source / Purification		Polyclonal antibodies are produced by immunizing animals with a synthetic phospho-peptide corresponding to residues surrounding Thr231/Ser232 of mouse RIP3 protein. Antibodies are purified by protein A and peptide affinity chromatography.				
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). In mice, RIP3 is phosphorylated at Thr231 and Ser232, leading to association with MLKL and necroptosis (15).				
Background References		2. Hsu, H. et al. (1996) 3. Stanger, B.Z. et al. (14. Ting, A.T. et al. (1996) 5. Kelliher, M.A. et al. (2007) 7. Zhang, S.Q. et al. (2008) 8. Lin, Y. et al. (1999) 10. Sun, X. et al. (1999) 11. Zhang, D.W. et al. (12. He, S. et al. (2009) 13. Cho, Y.S. et al. (2012)	n, E. and Tschopp, J. (2005) <i>Trends Biochem Sci</i> 30, 151-9. d. et al. (1996) <i>Immunity</i> 4, 387-96. er, B.Z. et al. (1995) <i>Cell</i> 81, 513-23. A.T. et al. (1996) <i>EMBO J</i> 15, 6189-96. er, M.A. et al. (1998) <i>Immunity</i> 8, 297-303. d., A. et al. (2000) <i>Immunity</i> 12, 419-29. d., S.Q. et al. (2000) <i>Immunity</i> 12, 301-11. et al. (1999) <i>Genes Dev</i> 13, 2514-26. N. et al. (1999) <i>Curr Biol</i> 9, 539-42. X. et al. (1999) <i>J Biol Chem</i> 274, 16871-5. d. D.W. et al. (2009) <i>Science</i> 325, 332-6. S. et al. (2009) <i>Cell</i> 137, 1100-11. Y.S. et al. (2012) <i>Cell</i> 148, 213-27. d., W. et al. (2013) <i>J Biol Chem</i> 288, 16247-61.			

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

Cross-Reactivity Key M: Mouse

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