#65746 Store at -20C

Phospho-RIP (Ser166) (D1L3S) Rabbit mAb



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Applications: WB, W-S	Reactivity: H	Sensitivity: Endogenous	MW (kDa): 78-82	Source/Isotype: Rabbit IgG	UniProt ID: #Q13546	Entrez-Gene Id: 8737	
Product Usage Information	Application			Dilution			
	Western Blotting			1:1000			
	Sin	nple Western™		1:10 - 1:50			
Storage		Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 μ g/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.					
Specificity / Sensi		Phospho-RIP (Ser166) (D1L3S) Rabbit mAb recognizes endogenous levels of RIP protein only when phosphorylated at Ser166.					
Source / Purificati	-	Monoclonal antibody is produced by immunizing animals with a synthetic phosphopeptide corresponding to residues surrounding Ser166 of human RIP 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-kB, 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-kB 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 IkB phosphorylation and degradation (6,7). Overexpression of RIP induces both NF-kB activation and apoptosis (2,3). Caspase-8-dependent cleavage of the RIP death domain can trigger the apoptotic activity of RIP (8).

Necroptosis, a regulated pathway for necrotic cell death, is triggered by a number of inflammatory signals including cytokines in the tumor necrosis factor (TNF) family, pathogen sensors such as toll-like receptors (TLRs), and ischemic injury (9,10). The process is negatively regulated by caspases and is initiated through a complex containing the RIP and RIP3 kinases, typically referred to as the necrosome. Necroptosis is inhibited by a small molecule inhibitor of RIP, necrostatin-1 (Nec-1) (11). Research studies show that necroptosis contributes to a number of pathological conditions, and Nec-1 has been shown to provide neuroprotection in models such as ischemic brain injury (12). RIP is phosphorylated at several sites within the kinase domain that are sensitive to Nec-1, including Ser14, Ser15, Ser161, and Ser166 (13).

Background References

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- 4. Ting, A.T. et al. (1996) EMBO J 15, 6189-96.
- 5. Kelliher, M.A. et al. (1998) Immunity 8, 297-303.
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- 7. Zhang, S.Q. et al. (2000) Immunity 12, 301-11.
- 8. Lin, Y. et al. (1999) Genes Dev 13, 2514-26.
- 9. Christofferson, D.E. and Yuan, J. (2010) Curr Opin Cell Biol 22, 263-8.
- 10. Kaczmarek, A. et al. (2013) Immunity 38, 209-23.
- 11. Degterev, A. et al. (2008) Nat Chem Biol 4, 313-21.
- 12. Degterev, A. et al. (2005) Nat Chem Biol 1, 112-9.
- 13. Ofengeim, D. and Yuan, J. (2013) Nat Rev Mol Cell Biol 14, 727-36.

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

WB: Western Blotting **W-S:** Simple Western™

Cross-Reactivity Key

H: human M: mouse R: rat Hm: hamster Mk: monkey Vir: virus Mi: mink C: chicken Dm: D. melanogaster X: Xenopus Z: zebrafish B: bovine Dg: dog Pg: pig Sc: S. cerevisiae Ce: C. elegans Hr: horse

GP: Guinea Pig Rab: rabbit All: all species expected

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