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## Cleaved RIP (Asp324) (D5P6D) Rabbit mAb



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Applications: WB	Reactivity: H	Sensitivity: Endogenous	<b>MW (kDa):</b> 30	Source/Isotype: Rabbit IgG	UniProt ID: #Q13546	Entrez-Gene Id 8737	
Product Usage	Aŗ	Application			Dilution		
Information	We	estern Blotting		1:1000			
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 Cleaved RIP (Asp324) (D5P6D) Rail RIP protein only when cleaved at As			` ,	it mAb recognizes endogenous levels of the amino-terminal end of 324.			
Source / Purifica	<b>Purification</b> Monoclonal antibody is produced by immunizing residues surrounding Asp324 of human RIP prot				synthetic peptide corre	esponding to	

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**

Background

- 1. Meylan, E. and Tschopp, J. (2005) Trends Biochem Sci 30, 151-9.
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- 3. Stanger, B.Z. et al. (1995) Cell 81, 513-23.
- 4. Ting, A.T. et al. (1996) *EMBO J* 15, 6189-96.
- 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. 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.

3/23/24, 11:24 AM Cleaved RIP (Asp324) (D5P6D) Rabbit mAb (#77565) Datasheet Without Images Cell Signaling Technology

**Applications Key** 

WB: Western Blotting

**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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