Revision 4

Store at +4C

#47477

Phospho-RIP3 (Thr231/Ser232) (E7S1R) Rabbit mAb (Alexa Fluor® 488 Conjugate)

Cell Signaling TECHNOLOGY®

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Applications:	Reactivity:	Sensitivity:	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
IF-IC	M	Endogenous	Rabbit IgG	#Q9QZL0	56532

Product Usage Information

Application	Dilution
Immunofluorescence (Immunocytochemistry)	1:200

Storage

Supplied in PBS (pH 7.2), less than 0.1% sodium azide and 2 mg/ml BSA. Store at 4°C. *Do not aliquot the antibody. Protect from light. Do not freeze.*

Specificity/Sensitivity

Phospho-RIP3 (Thr231/Ser232) (E7S1R) Rabbit mAb (Alexa Fluor® 488 Conjugate) recognizes endogenous levels of RIP3 protein only when phosphorylated at Thr231/Ser232.

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic phospho-peptide corresponding to residues surrounding Thr231/Ser232 of mouse RIP3 protein. This antibody may not recognize RIP3 when only singly phosphorylated at Thr231 or Ser232.

Description

This Cell Signaling Technology antibody is conjugated to Alexa Fluor® 488 fluorescent dye and tested in-house for direct immunofluorescent analysis in mouse cells. This antibody is expected to exhibit the same species cross-reactivity as the unconjugated Phospho-RIP3 (Thr231/Ser232) (E7S1R) Rabbit mAb #91702.

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).

Background References

1. Meylan, E. and Tschopp, J. (2005) *Trends Biochem Sci* 30, 151-9.
2. Hsu, H. et al. (1996) *Immunity* 4, 387-96.
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.

Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Applications Key

IF-IC: Immunofluorescence (Immunocytochemistry)

Cross-Reactivity Key

M: Mouse

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