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# RIP (D94C12) XP<sup>®</sup> Rabbit mAb (PE Conjugate)

Store at +4C  
#14577

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

<b>Applications:</b> FC-FP	<b>Reactivity:</b> H M R Hm Mk	<b>Sensitivity:</b> Endogenous	<b>Source/Isotype:</b> Rabbit IgG	<b>UniProt ID:</b> #Q13546	<b>Entrez-Gene Id:</b> 8737
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## Product Usage Information

### Application

Flow Cytometry (Fixed/Permeabilized)

### Dilution

1:50

## 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 antibodies. Protect from light. Do not freeze.

## Specificity/Sensitivity

RIP (D94C12) XP<sup>®</sup> Rabbit mAb (PE Conjugate) detects endogenous levels of total RIP (RIP1) protein. It has not been shown to cross-react with other RIP family members.

## Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Leu190 of human RIP protein.

## Description

This Cell Signaling Technology antibody is conjugated to phycoerythrin (PE) and tested in-house for direct flow cytometry analysis in human cells. The antibody is expected to exhibit the same species cross-reactivity as the unconjugated RIP (D94C12) XP<sup>®</sup> Rabbit mAb #3493.

## 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

**FC-FP:** Flow Cytometry (Fixed/Permeabilized)

## Cross-Reactivity Key

**H:** Human **M:** Mouse **R:** Rat **Hm:** Hamster **Mk:** Monkey

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