Revision 1

#12636 Store at -20C

RIP4 Antibody

Cell Signaling TECHNOLOGY®

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

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source:	UniProt ID:	Entrez-Gene Id:
WB, IP	H	Endogenous	86	Rabbit	#P57078	54101

Product Usage Information

Application	Dilution
Western Blotting	1:1000
Immunoprecipitation	1:50

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

RIP4 Antibody recognizes endogenous levels of total RIP4 protein.

Species predicted to react based on 100% sequence homology:

Monkey

Source / Purification

Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to residues near the carboxy terminus of human RIP4 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 serine-threonine kinase 4 (RIP4, ANKRD3, DIK, PKK, or RIPK4) is a membrane-associated, ankyrin repeat-containing member of the RIP family first identified in HaCat cells (9,10). RIP4 has been shown to be involved in keratinocyte differentiation *in vivo* as well as wound repair (11-13). Studies indicate that siRNA knockdown of RIP4 in human xenografted tumor cells suppresses Wnt-dependent growth while over-expression of RIP4 *in vitro* stabilized β-catenin and lead to an increase in Wnt-dependent gene expression (14).

Background References

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6. Devin, A. et al. (2000) *Immunity* 12, 419-29.
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11. Rountree, R.B. et al. (2010) *J Invest Dermatol* 130, 102-12.
12. Adams, S. et al. (2007) *J Invest Dermatol* 127, 538-44.
13. Holland, P. et al. (2002) *Curr Biol* 12, 1424-8.
14. Huang, X. et al. (2013) *Science* 339, 1441-5.

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 **IP:** Immunoprecipitation

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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Limited Uses