

RIP4 Antibody



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

Applications: W, IP	Reactivity: H	Sensitivity: Endogenous	MW (kDa): 86	Source/Isotype: Rabbit	UniProt ID: #P57078	Entrez-Gene Id: 54101
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Product Usage Information

Application

Western Blotting
Immunoprecipitation

Dilution

1:1000
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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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**W:** Western Blotting **IP:** Immunoprecipitation**Cross-Reactivity Key****H:** Human**Trademarks and Patents**

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