

Store at  
-20C  
#95702**RIP3 (D4G2A) Rabbit mAb**

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

| Applications:            | Reactivity: | Sensitivity: | MW (kDa): | Source/Isotype: | UniProt ID: | Entrez-Gene Id: |
|--------------------------|-------------|--------------|-----------|-----------------|-------------|-----------------|
| W, W-S, IP, IF-IC, FC-FP | M           | Endogenous   | 46-62     | Rabbit IgG      | #Q9QZL0     | 56532           |

**Product Usage Information****Application**

Western Blotting  
Simple Western™  
Immunoprecipitation  
Immunofluorescence (Immunocytochemistry)  
Flow Cytometry (Fixed/Permeabilized)

**Dilution**

1:1000  
1:10 - 1:50  
1:100  
1:400 - 1:1600  
1:800 - 1:1600

**Storage**

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at -20°C. Do not aliquot the antibody.

For a carrier free (BSA and azide free) version of this product see product #74771.

**Specificity/Sensitivity**

RIP3 (D4G2A) Rabbit mAb recognizes endogenous levels of total RIP3 protein from mouse.

**Source / Purification**

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Val370 of mouse RIP3 protein.

**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 protein 3 (RIP3) was originally found to interact with RIP and the TNF receptor complex to induce apoptosis and activation of NF-κB (9,10). It has subsequently been shown that the association between RIP and RIP3 is a key component of a signaling pathway that results in programmed necrosis (necroptosis), a necrotic-like cell death induced by TNF in the presence of caspase inhibitors (11-13). RIP3 is phosphorylated at Ser227 and targets the phosphorylation of mixed lineage kinase domain-like protein (MLKL), which is critical for necroptosis (14).

**Background References**

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7. Zhang, S.Q. et al. (2000) *Immunity* 12, 301-11.
8. Lin, Y. et al. (1999) *Genes Dev* 13, 2514-26.
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14. Sun, L. et al. (2012) *Cell* 148, 213-27.

**Species Reactivity**

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

|                               |   |
|-------------------------------|---|
| <b>Western Blot Buffer</b>    | 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.  |
| <b>Applications Key</b>       | <b>W:</b> Western Blotting <b>W-S:</b> Simple Western™ <b>IP:</b> Immunoprecipitation <b>IF-IC:</b> Immunofluorescence (Immunocytochemistry) <b>FC-FP:</b> Flow Cytometry (Fixed/Permeabilized)   |
| <b>Cross-Reactivity Key</b>   | <b>M:</b> Mouse   |
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