

#54105 Store at -20°C

## PhosphoPlus® RIP3 (Ser227) Antibody Duet



**Cell Signaling**  
TECHNOLOGY®

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

**UniProt ID:**  
#Q9Y572

**Entrez-Gene Id:**  
11035

Product Includes	Product #	Quantity	Mol. Wt.	Isotype/Source
RIP3 (E1Z1D) Rabbit mAb	13526	100 µl	46-62 kDa	Rabbit IgG
Phospho-RIP3 (Ser227) (D6W2T) Rabbit mAb	93654	100 µl	46-62 kDa	Rabbit IgG

Please visit [cellsignal.com](http://cellsignal.com) for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

### Description

PhosphoPlus® Duets from Cell Signaling Technology (CST) provide a means to assess protein activation status. Each Duet contains an activation-state and total protein antibody to your target of interest. These antibodies have been selected from CST's product offering based upon superior performance in specified applications.

### 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.*

### 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). Subsequently, it has 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). In mice, RIP3 is phosphorylated at Thr231 and Ser232, leading to association with MLKL and necroptosis (15).

### Background References

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