

RIP3 (Phospho- Ser316) Antibody

Catalog No: #12840



Package Size: #12840-1 50ul #12840-2 100ul

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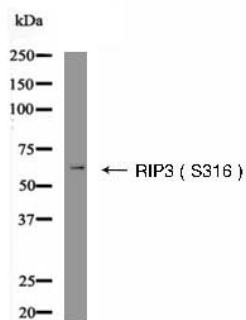
Description

Product Name	RIP3 (Phospho- Ser316) Antibody
Brief Description	Rabbit Polyclonal
Host Species	Rabbit
Clonality	Polyclonal
Applications	WB
Species Reactivity	Hu Ms Rt
Specificity	Phospho-RIP3 (S316) Antibody detects endogenous levels of RIP3 only when phosphorylated at S316
Immunogen Type	Peptide-KLH
Immunogen Description	A synthesized peptide derived from human RIP3 (Phospho- Ser316)
Other Names	Receptor interacting protein 3 antibody Receptor interacting serine threonine kinase 3 antibody Receptor interacting serine threonine protein kinase 3 antibody Receptor-interacting protein 3 antibody Receptor-interacting serine threonine-protein kinase 3 antibody RIP 3 antibody RIP like protein kinase 3 antibody RIP-3 antibody RIP-like protein kinase 3 antibody RIPK 3 antibody RIPK3 antibody RIPK3_HUMAN antibody
Accession No.	Swiss-Prot#:Q9QZL0 NCBI Gene ID56532
Calculated MW	46-62
Concentration	1.0mg mL
Formulation	Rabbit IgG in phosphate buffered saline (without Mg2+ and Ca2+) pH 7.4 150mM NaCl 0.02% sodium azide and 50% glycerol.
Storage	Store at -20°C

Application Details

WB dilution:1:1000

Images



Western blot analysis RIP3 (Phospho- Ser316) using EGF treated MCF7 whole cell lysates

Product Description

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- κ B, 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- κ B 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 I κ B phosphorylation and degradation (6,7). Overexpression of RIP induces both NF- κ B activation and apoptosis (2,3). Caspase-8-dependent cleavage of the RIP death domain can trigger the apoptotic activity of RIP (8).

Note: This product is for in vitro research use only and is not intended for use in humans or animals.