## Phospho-RelB (Ser552) (D41B9) XP® Rabbit



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

<b>Applications:</b> W, IP, IF-IC, FC-FP	Reactivity: H M	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 70	<b>Source/Isotype:</b> Rabbit IgG	UniProt ID: #Q01201	Entrez-Gene Id 5971
Product Usage Information		Application Western Blotting Immunoprecipitation Immunofluorescence Flow Cytometry (Fixed		istry)		<b>Dilution</b> 1:1000 1:100 1:400 1:800
Storage		Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 $\mu$ g/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) vers	sion of this product see	product #17616.	
Specificity/Sensitivity		Phospho-RelB (Ser552) (D41B9) $XP^{\otimes}$ Rabbit mAb detects endogenous levels of RelB only when phosphorylated at Ser552.				
Species predicted to react based on 100% sequence homology		Rat, Monkey, Bovine, [	Dog			
Source / Purification		Monoclonal antibody is produced by immunizing animals with a synthetic phosphopeptide corresponding to residues surrounding Ser552 of mouse RelB protein.				
Background		Transcription factors of the nuclear factor κB (NF-κB)/Rel family play a pivotal role in inflammatory and immune responses (1,2). There are five family members in mammals: RelA, c-Rel, RelB, NF-κB1 (p105/p50), and NF-κB2 (p100/p52). Both p105 and p100 are proteolytically processed by the proteasome to produce p50 and p52, respectively. Rel proteins bind p50 and p52 to form dimeric complexes that bind DNA and regulate transcription. In unstimulated cells, NF-κB is sequestered in the cytoplasm by IκB inhibitory proteins (3-5). NF-κB-activating agents can induce the phosphorylation of IκB proteins, targeting them for rapid degradation through the ubiquitin-proteasome pathway and releasing NF-κB to enter the nucleus where it regulates gene expression (6-8). NIK and IKKα (IKK1) regulate the phosphorylation and processing of NF-κB2 (p100) to produce p52, which translocates to the nucleus (9-11).  RelB, which is generally activated by non-canonical signaling, forms heterodimers with either p50 or p52 NF-κB subunits to regulate transcription (12,13). RelB null mice are significantly impaired in inflammatory responses and hematopoietic differentiation (14,15). Phosphorlyation at Thr84 and Ser552 results in proteosomal degradation (16).				
Background References		<ol> <li>Baeuerle, P.A. and Henkel, T. (1994) <i>Annu Rev Immunol</i> 12, 141-79.</li> <li>Baeuerle, P.A. and Baltimore, D. (1996) <i>Cell</i> 87, 13-20.</li> <li>Haskill, S. et al. (1991) <i>Cell</i> 65, 1281-9.</li> <li>Thompson, J.E. et al. (1995) <i>Cell</i> 80, 573-82.</li> <li>Whiteside, S.T. et al. (1997) <i>EMBO J</i> 16, 1413-26.</li> <li>Traenckner, E.B. et al. (1995) <i>EMBO J</i> 14, 2876-83.</li> <li>Scherer, D.C. et al. (1995) <i>Proc Natl Acad Sci USA</i> 92, 11259-63.</li> <li>Chen, Z.J. et al. (1996) <i>Cell</i> 84, 853-62.</li> <li>Senftleben, U. et al. (2001) <i>Science</i> 293, 1495-9.</li> <li>Coope, H.J. et al. (2002) <i>EMBO J</i> 21, 5375-85.</li> <li>Xiao, G. et al. (2001) <i>Mol Cell</i> 7, 401-9.</li> <li>Ryseck, R.P. et al. (1992) <i>Mol Cell Biol</i> 12, 674-84.</li> <li>Bours, V. et al. (1994) <i>Oncogene</i> 9, 1699-702.</li> <li>Weih, F. et al. (1995) <i>Cell</i> 80, 331-40.</li> <li>Mariosfeld, R. et al. (1995) <i>Nature</i> 333, 531-6.</li> <li>Mariosfeld, R. et al. (1995) <i>Cell</i> 80, 373, 531-6.</li> </ol>				

16. Marienfeld, R. et al. (2001) Oncogene 20, 8142-7.

**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 IF-IC: Immunofluorescence (Immunocytochemistry) FC-

FP: Flow Cytometry (Fixed/Permeabilized)

Cross-Reactivity Key H: Human M: Mouse

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