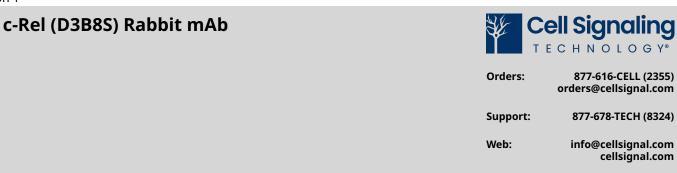
Store at -20C

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

Applications: W	Reactivity: H M	Sensitivity: Endogenous	MW (kDa): 68-78	Source/Isotype: Rabbit IgG	UniProt ID: #Q04864	Entrez-Gene Id: 5966
Product Usage Information		Application Western Blotting		Dilution 1:1000		
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.				
Specificity/Sensitivity		c-Rel (D3B8S) Rabbit mAb recognizes endogenous levels of total c-Rel protein.				
Source / Purification		Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Lys73 of human c-Rel 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 IkB inhibitory proteins (3-5). NF-κB-activating agents can induce the phosphorylation of IkB 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). c-Rel contains an amino-terminal DNA-binding domain referred to as the REL homology domain (RHD) and carboxy-terminal transactivation domains. The c-Rel protein is typically inhibited in unstimulated cells by IkBα and IkBβ. c-Rel expression is highest in hematopoietic cells with extensive research studies demonstrating its role in immune cell function and pathogenesis of disease (12,13).				
Background References		 Baeuerle, P.A. and Henkel, T. (1994) <i>Annu Rev Immunol</i> 12, 141-79. Baeuerle, P.A. and Baltimore, D. (1996) <i>Cell</i> 87, 13-20. Haskill, S. et al. (1991) <i>Cell</i> 65, 1281-9. Thompson, J.E. et al. (1995) <i>Cell</i> 80, 573-82. Whiteside, S.T. et al. (1997) <i>EMBO J</i> 16, 1413-26. Traenckner, E.B. et al. (1995) <i>Porb DJ</i> 14, 2876-83. Scherer, D.C. et al. (1996) <i>Cell</i> 84, 853-62. Senftleben, U. et al. (2001) <i>Science</i> 293, 1495-9. Coope, H.J. et al. (2001) <i>Mol Cell</i> 7, 401-9. Gilmore, T.D. and Gerondakis, S. (2011) <i>Genes Cancer</i> 2, 695-711. Fullard, N. et al. (2012) <i>Int J Biochem Cell Biol</i> 44, 851-60. 				
Species Reactivity		Species reactivity is de	etermined by testin	g in at least one approv	ed application (e.g.,	western blot).
Western Blot Buffer		IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v nonfat dry milk, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.				
Applications Key		W: Western Blotting				
Cross-Reactivity Key		H: Human M: Mouse				
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