

c-Rel (D5G1A) Rabbit mAb (ChIP Formulated)



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Applications: ChIP, ChIP-seq	Reactivity:	Sensitivity: Endogenous	Source/Isotype: Rabbit IgG	UniProt ID: #Q04864	Entrez-Gene Id: 5966	
Product Usage Information		For optimal ChIP and ChIP-seq results, use 5 μ l of antibody and 10 μ g of chromatin (approximately 4 x 10 ⁶ cells) per IP. This antibody has been validated using SimpleChIP [®] Enzymatic Chromatin IP Kits.				
		Application Chromatin IP Chromatin IP-seq			Dilution 1:100 1:100	
Storage		Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 μ g/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.				
Specificity/Sensitivity		c-Rel (D5G1A) Rabbit mAb (ChIP Formulated) recognizes endogenous levels of c-Rel in ChIP analysis.				
Species predicted to react based on 100% sequence homology		Rat, Monkey				
Source / Purification		Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Leu65 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 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). c-Rel contains an amino-terminal DNA-binding domain referred to as the REL homology domain (REH) and carboxy-terminal transactivation domains. The c-Rel protein is typically inhibited in unstimulated cells by IκBα and IκBβ. 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		2. Baeuerle, P.A. and Balt 3. Haskill, S. et al. (1991) 4. Thompson, J.E. et al. (1 5. Whiteside, S.T. et al. (1 6. Traenckner, E.B. et al. (7. Scherer, D.C. et al. (1996) 8. Chen, Z.J. et al. (1996) 9. Senftleben, U. et al. (2011) 10. Coope, H.J. et al. (2001) 11. Xiao, G. et al. (2001)	. Baeuerle, P.A. and Henkel, T. (1994) <i>Annu Rev Immunol</i> 12, 141-79. 2. Baeuerle, P.A. and Baltimore, D. (1996) <i>Cell</i> 87, 13-20. 3. Haskill, S. et al. (1991) <i>Cell</i> 65, 1281-9. 4. Thompson, J.E. et al. (1995) <i>Cell</i> 80, 573-82. 5. Whiteside, S.T. et al. (1997) <i>EMBO J</i> 16, 1413-26. 5. Traenckner, E.B. et al. (1995) <i>EMBO J</i> 14, 2876-83. 7. Scherer, D.C. et al. (1995) <i>Proc Natl Acad Sci USA</i> 92, 11259-63. 8. Chen, Z.J. et al. (1996) <i>Cell</i> 84, 853-62. 9. Senftleben, U. et al. (2001) <i>Science</i> 293, 1495-9. 9. Coope, H.J. et al. (2002) <i>EMBO J</i> 21, 5375-85. 1. Xiao, G. et al. (2001) <i>Mol Cell</i> 7, 401-9. 2. Gilmore, T.D. and Gerondakis, S. (2011) <i>Genes Cancer</i> 2, 695-711. 3. Fullard, N. et al. (2012) <i>Int J Biochem Cell Biol</i> 44, 851-60.			

Species Reactivity

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

Applications Key

ChIP: Chromatin IP ChIP-seq: Chromatin IP-seq

Cross-Reactivity Key

H: Human

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