MCM3 (D47B6) Rabbit mAb



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Applications: W	Reactivity: H M R Mk	Sensitivity: Endogenous	MW (kDa): 100	Source/Isotype: Rabbit IgG	UniProt ID: #P25205	Entrez-Gene Id: 4172
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		MCM3 (D47B6) Rabbit mAb detects endogenous levels of total MCM3 protein.				
Source / Purification		Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to amino-terminal residues of human MCM3.				
Background		The minichromosome maintenance (MCM) 2-7 proteins are a family of six related proteins required for initiation and elongation of DNA replication. MCM2-7 bind together to form the heterohexameric MCM complex that is thought to act as a replicative helicase at the DNA replication fork (1-5). This complex is a key component of the pre-replication complex (pre-RC) (reviewed in 1). Cdc6 and CDT1 recruit the MCM complex to the origin recognition complex (ORC) during late mitosis/early G1 phase forming the pre-RC and licensing the DNA for replication (reviewed in 2). Licensing of the chromatin permits the DNA to replicate only once per cell cycle, thereby helping to ensure that genetic alterations and malignant cell growth do not occur (reviewed in 3). Phosphorylation of the MCM2, MCM3, MCM4, and MCM6 subunits appears to regulate MCM complex activity and the initiation of DNA synthesis (6-8). CDK1 phosphorylation of MCM3 at Ser112 during late mitosis/early G1 phase has been shown to initiate complex formation and chromatin loading <i>in vitro</i> (8). Phosphorylation of MCM2 at serine 139 by cdc7/dbf4 coincides with the initiation of DNA replication (9). MCM proteins are removed during DNA replication, causing chromatin to become unlicensed through inhibition of pre-RC reformation. Studies have shown that the MCM complex is involved in checkpoint control by protecting the structure of the replication fork and assisting in restarting replication by recruiting checkpoint proteins after arrest (reviewed in 3,10).				
Background References		1. Lei, M. and Tye, B.K. (2001) <i>J Cell Sci</i> 114, 1447-54. 2. Lygerou, Z. and Nurse, P. (2000) <i>Science</i> 290, 2271-3. 3. Forsburg, S.L. (2004) <i>Microbiol Mol Biol Rev</i> 68, 109-31. 4. Tye, B.K. and Sawyer, S. (2000) <i>J Biol Chem</i> 275, 34833-6. 5. Labib, K. et al. (2000) <i>Science</i> 288, 1643-7. 6. Charych, D.H. et al. (2008) <i>J Cell Biochem</i> 104, 1075-86. 7. Masai, H. et al. (2006) <i>J Biol Chem</i> 281, 39249-61. 8. Lin, D.I. et al. (2008) <i>Proc Natl Acad Sci USA</i> 105, 8079-84. 9. Tsuji, T. et al. (2006) <i>Mol Biol Cell</i> 17, 4459-72. 10. Bailis, J.M. et al. (2008) <i>Mol Cell Biol</i> 28, 1724-38.				

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

Cross-Reactivity Key H: Human M: Mouse R: Rat Mk: Monkey

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