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TTK (D15B7) Rabbit mAb	Cell Signaling		
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Applications: W, IP	Reactivity: H	Sensitivity: Endogenous	MW (kDa): 95	Source/Isotype: Rabbit IgG	UniProt ID: #P33981	Entrez-Gene Id: 7272		
Product Usage Information		Application Western Blotting Immunoprecipitation			Dilution 1:1000 1:100			
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/Sen	sitivity	TTK (D15B7) Rabbit mAb detects endogenous levels of total TTK protein. A background band of unknown origin is detected at 70 kDa.						
Species predict based on 100% homology	ted to react sequence	Monkey						
Source / Purific	cation	Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to a region surrounding Ala560 of human TTK protein.						
Background		TTK (Mps1, PYT) is a cell cycle regulated dual specificity kinase present in rapidly proliferating tissues and cell lines (1-3). TTK localizes to kinetochores and centromeres and is an essential component of the mitotic spindle checkpoint as well as centrosome duplication (4-6). The mitotic checkpoint inhibits entry into anaphase until all chromosomes are attached to the spindle; inhibition of this process leads to genomic instability and tumorigenesis. Phosphorylation of the BLM helicase at Ser144 by TTK maintains chromosome stability during mitosis (7). Small molecule inhibitors of TTK can block the spindle checkpoint response, thereby making TTK a potential therapeutic target (8,9). TTK also participates in the DNA damage response by directly phosphorylating and activating the cell cycle checkpoint kinase Chk2 at Thr68. Two targets phosphorylated by Chk2 are the cell cycle phosphatase cdc25 and the transcription factor p53. Inactivation of cdc25 phosphatase results in the accumulation of inactive cyclin B and cell cycle arrest following DNA damage. Phosphorylation of p53 by active Chk2 stabilizes the transcription factor and promotes cell cycle arrest and apoptosis in response to DNA damage (10).						
Background Re	eferences	 Mills, G.B. et al. (1992) <i>J. Biol. Chem.</i> 267, 16000-16006. Stucke, V.M. et al. (2002) <i>EMBO J.</i> 21, 1723-1732. Lindberg, R.A. et al. (1993) <i>Oncogene</i> 8, 351-359. Fisk, H.A. et al. (2003) <i>Proc. Natl. Acad. Sci. USA</i> 100, 14875-14880. Dou, Z. et al. (2003) <i>Cell Res.</i> 13, 443-449. Abrieu, A. et al. (2001) <i>Cell</i> 106, 83-93. Leng, M. et al. (2006) <i>Proc. Natl. Acad. Sci. USA</i> 103, 11485-11490. Schmidt, M. et al. (2005) <i>EMBO Rep.</i> 6, 866-872. Dorer, R.K. et al. (2005) <i>Curr. Biol.</i> 15, 1070-1076. Wei, J.H. et al. (2005) <i>J. Biol. Chem.</i> 280, 7748-7757. 						
Species Reactiv	vitv	Species reactivity is de	etermined by testin	g in at least one approve	ed application (e.g.,	western blot).		
Western Blot B	Suffer	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 K	ey	W: Western Blotting I	P: Immunoprecipita	ation				
Cross-Reactivit	ту Кеу	H: Human						
Trademarks ar	nd Patents	Cell Signaling Technology is a trademark of Cell Signaling Technology, Inc.						

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