p70 S6 Kinase (49D7) Rabbit mAb (Biotinylated)	Cell TEC	Cell Signaling Technology®	
Store		877-616-CELL (2355) ders@cellsignal.com	
	Support:	877-678-TECH (8324)	
#5707	Web:	info@cellsignal.com cellsignal.com	
<b>1</b> 2	3 Trask Lane   Danvers   Massachu	usetts   01923   USA	
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Applications: W	<b>Reactivity:</b> H M R Mk	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 70, 85	<b>Source/Isotype:</b> Rabbit IgG	<b>UniProt ID:</b> #P23443	Entrez-Gene Id 6198		
Product Usage Information		<b>Application</b> Western Blotting			<b>Dilution</b> 1:1000			
Storage		Supplied in 140 mM NaCl, 3 mM KCI, 10 mM sodium phosphate (pH 7.4) dibasic, 2 mM potassium phosphate monobasic, 2 mg/mL BSA, and 50% glycerol. Store at –20°C. <i>Do not aliquot the antibody.</i>						
Specificity/Sensitivity		p70 S6 Kinase (49D7) Rabbit mAb (Biotinylated) detects endogenous levels of total p70 S6 kinase protein. The antibody also recognizes p85 S6 kinase.						
Source / Purification		Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding the amino terminus of human p70 S6 kinase.						
Description		This Cell Signaling Technology antibody is conjugated to biotin under optimal conditions. The biotinylated antibody is expected to exhibit the same species cross-reactivity as the unconjugated p70 S6 Kinase (49D7) Rabbit mAb #2708.						
Background		p70 S6 kinase is a mitogen activated Ser/Thr protein kinase that is required for cell growth and G1 cell cycle progression (1,2). p70 S6 kinase phosphorylates the S6 protein of the 40S ribosomal subunit and is involved in translational control of 5' oligopyrimidine tract mRNAs (1). A second isoform, p85 S6 kinase, is derived from the same gene and is identical to p70 S6 kinase except for 23 extra residues at the amino terminus, which encode a nuclear localizing signal (1). Both isoforms lie on a mitogen activated signaling pathway downstream of phosphoinositide-3 kinase (PI-3K) and the target of rapamycin, FRAP/mTOR, a pathway distinct from the Ras/MAP kinase cascade (1). The activity of p70 S6 kinase is controlled by multiple phosphorylation events located within the catalytic, linker and pseudosubstrate domains (1). Phosphorylation of Thr229 in the catalytic domain and Thr389 in the linker domain are most critical for kinase function (1). Phosphorylation of Thr389, however, most closely correlates with p70 kinase activity <i>in vivo</i> (3). Prior phosphorylation of Thr389, however, most closely correlates with p70 kinase activity <i>in vivo</i> (3). Prior phosphorylation of Thr389 is required for the action of phosphoinositide 3-dependent protein kinase 1 (PDK1) on Thr229 (4,5). Phosphorylation of this site is stimulated by growth factors such as insulin, EGF and FGF, as well as by serum and some G-protein-coupled receptor ligands, and is blocked by wortmannin, LY294002 (PI-3K inhibitor) and rapamycin (FRAP/mTOR inhibitor) (1,6,7). Ser411, Thr421 and Ser424 lie within a Ser-Pro-rich region located in the pseudosubstrate suppression (1,2). Another LY294002 and rapamycin sensitive phosphorylation site, Ser371, is an <i>in vitro</i> substrate for mTOR and correlates well with the activity of a partially rapamycin resistant mutant p70 S6 kinase (8).						
Background Re	ferences	1. Pullen, N. and Tho 2. Dufner, A. and Tho 3. Weng, Q.P. et al. (1 4. Pullen, N. et al. (19 5. Alessi, D.R. et al. (1 6. Polakiewicz, R.D. e 7. Fingar, D.C. et al. (2 8. Saitoh, M. et al. (20	omas, G. (1999) <i>Exp</i> ( 998) <i>J Biol Chem</i> 27: 998) <i>Science</i> 279, 70 1998) <i>Curr Biol</i> 8, 69- t al. (1998) <i>J Biol Che</i> 2002) <i>Genes Dev</i> 16,	<i>Cell Res</i> 253, 100-9. 3, 16621-9. 7-10. -81. <i>em</i> 273, 23534-41. 1472-87.				
Species Reactiv	vity	Species reactivity is o	determined by testir	ig in at least one approve	ed application (e.g.,	western blot).		
Western Blot B	uffer	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 Ke	ications Key W: Western Blotting							
Cross-Reactivit	y Key	H: Human M: Mouse R: Rat Mk: Monkey						
Trademarks an	d Patents	Cell Signaling Technology is a trademark of Cell Signaling Technology, Inc.						

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