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## Phospho-FoxO1 (Ser256) (E1F7T) Rabbit



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Applications: W, IP	<b>Reactivity:</b> H M R Mk	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 82	<b>Source/Isotype:</b> Rabbit IgG	UniProt ID: #Q12778	Entrez-Gene Id: 2308		
Product Usage Information		Application Western Blotting Immunoprecipitation			<b>Dilution</b> 1:1000 1:50			
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		Phospho-FoxO1 (Ser256) (E1F7T) recognizes endogenous levels of FoxO1 protein only when phosphorylated at Ser256. The antibody cross-reacts with overexpressed FoxO4 phosphorylated at Ser193 and may cross-react with overexpressed FoxO3a phosphorylated at Ser253. The antibody also cross-reacts with a protein of unknown origin around 160kD.						
Source / Purifi	ication	Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Ser256 of human FoxO1 protein.						
<b>Background</b> The Forkhead family of transcription factors is involved in tumorigenesis of rhabdomyosa acute leukemias (1-3). Within the family, three members (FoxO1, FoxO4, and FoxO3a) hav similarity to the nematode orthologue DAF-16, which mediates signaling via a pathway in PI3K, and Akt (4-6). Active forkhead members act as tumor suppressors by promoting cel and apoptosis. Increased expression of any FoxO member results in the activation of the inhibitor p27 Kip1. Forkhead transcription factors also play a part in TGF-β-mediated upre p21 Cip1, a process negatively regulated through PI3K (7). Increased proliferation results forkhead transcription factors are inactivated through phosphorylation by Akt at Thr24, S Ser319, which results in nuclear export and inhibition of transcription factor activity (8). Fr transcription factors can also be inhibited by the deacetylase sirtuin (SirT1) (9).				) have sequence vay involving IGFR1, g cell cycle arrest f the cell cycle upregulation of sults when '24, Ser256, and				
Background R	eferences	<ol> <li>Anderson, M.J. et al. (1998) <i>Genomics</i> 47, 187-99.</li> <li>Galili, N. et al. (1993) <i>Nat Genet</i> 5, 230-5.</li> <li>Borkhardt, A. et al. (1997) <i>Oncogene</i> 14, 195-202.</li> <li>Nakae, J. et al. (1999) <i>J Biol Chem</i> 274, 15982-5.</li> <li>Rena, G. et al. (1999) <i>J Biol Chem</i> 274, 17179-83.</li> <li>Guo, S. et al. (1999) <i>J Biol Chem</i> 274, 17184-92.</li> <li>Seoane, J. et al. (2004) <i>Cell</i> 117, 211-23.</li> <li>Arden, K.C. (2004) <i>Mol Cell</i> 14, 416-8.</li> <li>Yang, Y. et al. (2005) <i>EMBO J</i> 24, 1021-32.</li> </ol>						
Species Reacti	ivity	Species reactivity is determined by testing in at least one approved application (e.g., western blot).						
Western Blot I	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 <b>K</b>	(ey	W: Western Blotting IP: Immunoprecipitation						
Cross-Reactivi	ity Key	H: Human M: Mouse R: Rat Mk: Monkey						
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