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Rubicon (D9F7) Rabbit mAb



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Applications: W	Reactivity: H M	Sensitivity: Endogenous	MW (kDa): 130	Source/Isotype: Rabbit IgG	UniProt ID: #Q92622	Entrez-Gene Id: 9711		
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		Rubicon (D9F7) Rabbit mAb recognizes endogenous levels of total Rubicon protein. A band of unknown origin is detected at 55 kDa.						
Source / Purifi	cation	Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Leu210 of human Rubicon protein.						
Background Background Re	 Autophagy is a catabolic process for the autophagosomic-lysosomal degradation of bulk cytoplasmic contents (1,2). Autophagy is generally activated by conditions of nutrient deprivation but is also associated with a number of physiological processes, including development, differentiation, neurodegeneration, infection, and cancer (3). The molecular machinery of autophagy was largely discovered in yeast and is directed by a number of autophagy-related (Atg) genes. These proteins are involved in the formation of autophagosomes, which are cytoplasmic vacuoles that are delivered to lysosomes for degradation. The class III type phosphoinositide 3-kinase (PI3K) Vps34 regulates vacuolar trafficking and autophagy (4,5). Multiple proteins associate with Vps34, including p105/Vps1 Beclin-1, UVRAG, Atg14, and Rubicon (6-12). Atg14 and Rubicon were identified based on their ability bind to Beclin-1 and participate in unique complexes with opposing functions (9-12). Rubicon, which localizes to the endosome and lysosome, inhibits Vps34 lipid kinase activity; knockdown of Rubicon enhances autophagy and endocytic trafficking (11,12). In contrast, Atg14 localizes to autophagosome isolation membranes, and ER and can enhance Vps34 activity. Knockdown of Atg14 inhibits starvation induced autophagy (11,12). 1. Reggiori, F. and Klionsky, D.J. (2002) <i>Eukaryot Cell</i> 1, 11-21. 2. Codogno, P. and Meijer, A.J. (2005) <i>Cell Death Differ</i> 12 Suppl 2, 1509-18. 					but is also ntiation, y was largely hese proteins are are delivered to regulates uding p105/Vps15, d on their ability to Rubicon, which wwn of Rubicon autophagosomes,		
		 Coudghi, P. and Weijel, X.J. (2005) <i>J Clin Invest 115</i>, 2679-88. Levine, B. and Yuan, J. (2005) <i>J Clin Invest</i> 115, 2679-88. Corvera, S. (2001) <i>Traffic</i> 2, 859-66. Yan, Y. and Backer, J.M. (2007) <i>Biochem Soc Trans</i> 35, 239-41. Stack, J.H. et al. (1995) <i>J Cell Biol</i> 129, 321-34. Zeng, X. et al. (2006) <i>J Cell Sci</i> 119, 259-70. Liang, C. et al. (2006) <i>Nat Cell Biol</i> 8, 688-99. Itakura, E. et al. (2008) <i>Mol Biol Cell</i> 19, 5360-72. Sun, Q. et al. (2008) <i>Proc Natl Acad Sci USA</i> 105, 19211-6. Zhong, Y. et al. (2009) <i>Nat Cell Biol</i> 11, 468-76. Matsunaga, K. et al. (2009) <i>Nat Cell Biol</i> 11, 385-96. 						
Species Reactivity		Species reactivity is determined by testing in at least one approved application (e.g., western blot).						
Western Blot E	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.				1 5% w/v BSA, 1X		
Applications K	ations Key W: Western Blotting							
Cross-Reactivity Key H: Human M: Mouse								
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