ម្ត<mark>ីខ្ល</mark>ុ BNIP3 (D7U1T) Rabbit mAb





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Applications: W, IP, IHC-P, IF-IC	Reactivity: H	Sensitivity: Endogenous	MW (kDa): 22-28, 50-55	Source/Isotype: Rabbit IgG	UniProt ID: #Q12983	Entrez-Gene Id: 664	
Product Usage Information Storage		ApplicationDilutionWestern Blotting1:1000Immunoprecipitation1:100Immunohistochemistry (Paraffin)1:50 - 1:200Immunofluorescence (Immunocytochemistry)1:800Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less			:1000 :100 :50 - 1:200 :800		
-		0.02% sodium azide. Store at –20°C. Do not aliquot the antibody. For a carrier free (BSA and azide free) version of this product see product #34048.					
Specificity/Sens	itivity	BNIP3 (D7U1T) Rabbit mAb recognizes endogenous levels of total BNIP3 protein.					
Source / Purifica	ation	Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues near the amino terminus of human BNIP3 protein.				orresponding to	
Background		BNIP3 (Bcl-2/E1B-19kDa interacting protein 3) is a pro-apoptotic mitochondrial protein and Bcl-2 family member that contains a Bcl-2 homology 3 (BH3) domain and a carboxyl-terminal transmembrane (TM) domain (1-3). While BNIP3 has a predicted molecular weight of about 22 kDa, it runs anomalously on SDS-PAGE and includes a band of around 60 kDa that may be a dimeric form that is not reduced (2). BNIP3 associates with anti-apoptotic family members Bcl-2, Bcl-xL, and the adenovirus homologue E1B-19kDa. BNIP3 is distinct from other Bcl-2 family members that contain only the BH3 domain in that the TM domain, and not the BH3 domain, is required for mitochondrial targeting and pro-apoptotic activity (4). In addition to apoptosis, BNIP3 has been implicated in necrosis (5) and autophagy (6-11). In hypoxic conditions, BNIP3 can also promote mitophagy by triggering the translocation of the E3 ubiquitin ligase Parkin to the mitochondria (10) or by directly binding LC3 on the autophagosome (11). BNIP3 may also localize to the endoplasmic reticulum (ER) where it can selectively induce the autophagic clearance of ER (ERphagy) (11). Increased expression of BNIP3 under hypoxic conditions is mainly regulated by the transcription factor HIF-1α (12-14). Silencing of the BNIP3 promoter by methylation has been observed in several types of cancer cells and may play an important role in their survival (14-18).					
Background Ref	round References 1. Boyd, J.M. et al. (1994) Cell 79, 341-51. 2. Chen, G. et al. (1997) J Exp Med 186, 1975-83. 3. Yasuda, M. et al. (1998) J Biol Chem 273, 12415-21. 4. Ray, R. et al. (2000) J Biol Chem 275, 1439-48. 5. Vande Velde, C. et al. (2000) Mol Cell Biol 20, 5454-68. 6. Daido, S. et al. (2004) Cancer Res 64, 4286-93. 7. Tracy, K. et al. (2007) Mol Cell Biol 27, 6229-42. 8. Quinsay, M.N. et al. (2010) Autophagy 6, 855-62. 9. Bellot, G. et al. (2001) Cancer Res 64, 928. 10. Lee, Y. et al. (2011) Am J Physiol Heart Circ Physiol 301, H1924-31. 11. Hanna, R.A. et al. (2012) J Biol Chem 287, 19094-104. 12. Bruick, R.K. (2000) Proc Natl Acad Sci USA 97, 9082-7. 13. Guo, K. et al. (2001) Cell Death Differ 8, 367-76. 14. Sowter, H.M. et al. (2001) Cancer Res 61, 6669-73. 15. de Angelis, P.M. et al. (2004) Cancer Res 64, 5338-46. 17. Murai, M. et al. (2005) Clin Cancer Res 11, 1021-7. 18. Murai, M. et al. (2005) Br J Cancer 92, 1165-72.						

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 IP: Immunoprecipitation IHC-P: Immunohistochemistry (Paraffin) IF-IC: Immunofluorescence (Immunocytochemistry)		
Cross-Reactivity Key	H: Human		
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