

## TNFRSF8/CD30 (E1A6Y) Rabbit mAb



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## For Research Use Only. Not for Use in Diagnostic Procedures.

<b>Applications:</b> W, IP	Reactivity: H	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 90, 120	<b>Source/Isotype:</b> Rabbit IgG	UniProt ID: #P28908	Entrez-Gene Id: 943
Product Usage Information		<b>Application</b> Western Blotting Immunoprecipitation			<b>Dilution</b> 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/Sensitivity		TNFRSF8/CD30 (E1A6Y) Rabbit mAb recognizes endogenous levels of total TNFRSF8/CD30 protein.				
Source / Purification		Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Leu500 of human TNFRSF8/CD30 protein.				
Background		TNFRSF8/CD30 is a type-I transmembrane glycoprotein that is a member of the TNFR superfamily. CD30 is synthesized as a precursor protein that undergoes extensive post-translational modification before becoming embedded in the plasma membrane as a 120-kDa transmembrane protein (1,2). The expression of CD30 is upregulated in activated T cells and may trigger costimulatory signaling pathways upon its engagement (3,4). While its expression is normally restricted to subsets of activated T cells and B cells, CD30 expression is robustly upregulated in hematologic malignancies, such as Hodgkin lymphoma (HL), anaplastic large cell lymphoma (ALCL), and adult T-cell leukemia, thus making it an attractive target for therapeutic intervention (5,6). Research studies have suggested that in certain disease contexts, CD30 recruits TRAF2 and TRAF5 adaptor proteins to drive NF-kappa B activation, aberrant cell growth, and cytokine production (7-9). CD30 signaling is also regulated by TACE-dependent proteolytic cleavage of its ectodomain, which results in reduced CD30L-dependent activation of CD30+cells (10,11).				
Background References		1. Froese, P. et al. (1987) <i>J Immunol</i> 139, 2081-7. 2. Nawrocki, J.F. et al. (1988) <i>J Immunol</i> 141, 672-80. 3. Del Prete, G. et al. (1995) <i>J Exp Med</i> 182, 1655-61. 4. Gilfillan, M.C. et al. (1998) <i>J Immunol</i> 160, 2180-7. 5. Stein, H. et al. (1985) <i>Blood</i> 66, 848-58. 6. Chiarle, R. et al. (1999) <i>Clin Immunol</i> 90, 157-64. 7. Horie, R. et al. (2002) <i>Am J Pathol</i> 160, 1647-54. 8. Horie, R. et al. (2002) <i>Oncogene</i> 21, 2493-503. 9. Horie, R. et al. (2004) <i>Cancer Cell</i> 5, 353-64. 10. Hansen, H.P. et al. (2000) <i>J Immunol</i> 165, 6703-9. 11. Gruss, H.J. et al. (1997) <i>Immunol Today</i> 18, 156-63.				

**Species Reactivity** Species reactivity is determined by testing in at least one approved application (e.g., western blot).

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

Cross-Reactivity Key H: Human

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KARPAS cell line source: Dr. Abraham Karpas at the University of Cambridge.

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