

GITR (D9I9D) Rabbit mAb



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Applications: W, IHC-Bond, IHC-P	Reactivity: H	Sensitivity: Endogenous	MW (kDa): 25	Source/Isotype: Rabbit IgG	UniProt ID: #Q9Y5U5	Entrez-Gene Id: 8784
Product Usage Information		ApplicationDilutionWestern Blotting1:1000IHC Leica Bond1:400Immunohistochemistry (Paraffin)1:400				1000 400
Storage		Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 μ g/ml BSA, 50% glycerol and less than 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 #77770				
Specificity/Sensitivity		GITR (D9I9D) Rabbit mAb #68014 recognizes endogenous levels of total GITR protein.				
Species predicted to react based on 100% sequence homology		Monkey				
Source / Purification		Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Val142 of human GITR protein.				
Background		TNFRSF18, also known as glucocorticoid-induced tumor necrosis factor-receptor (TNFR)-related protein (GITR) and activation-inducible TNFR family receptor, encodes a type 1 membrane protein of the TNF-receptor superfamily (1). Three alternatively spliced transcript variants encoding distinct isoforms have been reported (2). GITR is an immune cell co-stimulatory receptor expressed constitutively at high levels on CD4 ⁺ CD25 ⁺ T regulatory cells (Tregs), at low levels on naïve and memory T cells, and is induced upon T cell activation (3-5). Studies show GITR can also be induced on NK cells, macrophages, and DCs (3,4,6). Although GITR does not have intrinsic enzymatic activity, TNFSF18 (also known as GITRL) expressed on antigen presenting cells binds to GITR, resulting in recruitment of TNFR-associated factor family members and activation of the NF-kB pathway in T cells (7). GITR ligation has been shown to play a role in CD8 ⁺ T cell activation, cytotoxicity, and memory T cell survival (8-10). In the thymus, GITR is thought to play a key role in dominant immunological self-tolerance through thymic Treg differentiation and expansion (11). Of note, GITR ligation inhibits Treg suppressive function (12-13) and promotes effector T cell resistance to Treg suppression (14-15). Due to the combined effects on both Treg suppression and effector cell activation, GITR represents a unique opportunity for immunotherapeutic intervention in cancer (16).				
Background References		 Nocentini, G. et al. (1997) Proc Natl Acad Sci U S A 94, 6216-21. Nocentini, G. et al. (2000) Cell Death Differ 7, 408-10. Shimizu, J. et al. (2002) Nat Immunol 3, 135-42. Nocentini, G. and Riccardi, C. (2009) Adv Exp Med Biol 647, 156-73. McHugh, R.S. et al. (2002) Immunity 16, 311-23. Hanabuchi, S. et al. (2006) Blood 107, 3617-23. Snell, L.M. et al. (2011) Immunol Rev 244, 197-217. Ronchetti, S. et al. (2007) J Immunol 179, 5916-26. Kim, I.K. et al. (2015) Nat Med 21, 1010-7. Snell, L.M. et al. (2012) J Immunol 188, 5915-23. Petrillo, M.G. et al. (2015) Autoimmun Rev 14, 117-26. Kanamaru, F. et al. (2004) J Immunol 172, 7306-14. Valzasina, B. et al. (2004) J Immunol 173, 5008-20. Nishikawa, H. et al. (2008) Cancer Res 68, 5948-54. 				

16. Knee, D.A. et al. (2016) Eur J Cancer 67, 1-10.

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 **IHC-Bond**: IHC Leica Bond **IHC-P**: Immunohistochemistry (Paraffin)

Cross-Reactivity Key H: Human

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