CTLA-4 (D4E9I) Rabbit mAb (Alexa Fluor® 488 Conjugate)



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Applications: FC-FP, FC-L	Reactivity: H	Sensitivity: Endogenous	Source/Isotype: Rabbit IgG	UniProt ID: #P16410	Entrez-Gene Id: 1493
Product Usage Information		Application Flow Cytometry (Fixed/P Flow Cytometry (Live)	ermeabilized)		Dilution 1:50 1:50
Storage		Supplied in PBS (pH 7.2), less than 0.1% sodium azide and 2 mg/ml BSA. Store at 4°C. Do not aliquot the antibody. Protect from light. Do not freeze.			
Specificity/Sensitivity		CTLA-4 (D4E9I) Rabbit mAb (Alexa Fluor [®] 488 Conjugate) recognizes endogenous levels of total CTLA-4 protein.			
Source / Purification		Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Asp100 of human CTLA-4 protein.			
Description		This Cell Signaling Technology antibody is conjugated to Alexa Fluor [®] 488 fluorescent dye and tested in-house for direct flow cytometric analysis in human cells. This antibody is expected to exhibit the same species cross-reactivity as the unconjugated CTLA-4 (D4E9I) Rabbit mAb #15119.			
Background		Cytotoxic T-lymphocyte protein 4 (CTLA-4, CD152) is an Ig superfamily member that negatively regulates early T cell activation (1-4). The CTLA-4 protein is primarily expressed on T cells, including CD8+ cytotoxic T cells, CD4+ helper T cells, and CD4+/FoxP3+ regulatory T cells (1,2). CTLA-4 protein competes with CD28 for B7.1 (CD80) and B7.2 (CD86) binding at the cell surface, which results in the downregulation of T cell activity (5). The activation of SHP-2 and PP2A downstream of CTLA-4 attenuates TCR signaling (6). Research studies indicate that <i>CTLA4</i> knockout mice display lymphoproliferative disorders leading to early death, confirming the role of CTLA-4 as a negative regulator of T cells (7). Mutations in the corresponding <i>CTLA4</i> gene are associated with multiple disorders, including insulin-dependent diabetes mellitus, Graves' disease, Hashimoto thyroiditis, celiac disease, systemic lupus erythematosus, and type V autoimmune lymphoproliferative syndrome (8,9). Additional studies demonstrate that CTLA-4 blockade is an effective strategy for tumor immunotherapy (10-12).			
Background References		1. Brunet, J.F. et al. (1987) <i>Nature</i> 328, 267-70. 2. Brunet, J.F. et al. (1988) <i>Immunol Rev</i> 103, 21-36. 3. Dariavach, P. et al. (1988) <i>Eur J Immunol</i> 18, 1901-5. 4. Linsley, P.S. (1995) <i>J Exp Med</i> 182, 289-92. 5. Collins, A.V. et al. (2002) <i>Immunity</i> 17, 201-10. 6. Rudd, C.E. et al. (2009) <i>Immunol Rev</i> 229, 12-26. 7. Waterhouse, P. et al. (1995) <i>Science</i> 270, 985-8. 8. Romo-Tena, J. et al. (2013) <i>Autoimmun Rev</i> 12, 1171-6. 9. Wang, J. et al. (2014) <i>PLoS One</i> 9, e85982. 10. Egen, J.G. et al. (2002) <i>Nat Immunol</i> 3, 611-8. 11. Hodi, F.S. et al. (2003) <i>Proc Natl Acad Sci U S A</i> 100, 4712-7. 12. Pardoll, D.M. (2012) <i>Nat Rev Cancer</i> 12, 252-64.			

Species Reactivity

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

Applications Key

FC-FP: Flow Cytometry (Fixed/Permeabilized) FC-L: Flow Cytometry (Live)

Cross-Reactivity Key

H: Human

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