

Store at  
-20°C  
#10419**GITR (D5V7P) Rabbit mAb**

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

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, IP, IHC-Bond, IHC-P, IF-IC, FC-FP, FC-L	H	Endogenous	25	Rabbit IgG	#Q9Y5U5	8784

**Product Usage Information****Application**

Western Blotting  
Immunoprecipitation  
IHC Leica Bond  
Immunohistochemistry (Paraffin)  
Immunofluorescence (Immunocytochemistry)  
Flow Cytometry (Fixed/Permeabilized)  
Flow Cytometry (Live)

**Dilution**

1:1000  
1:50  
1:100  
1:100  
1:400  
1:200  
1:200

**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.

For a carrier free (BSA and azide free) version of this product see product #24200.

**Specificity/Sensitivity**

GITR (D5V7P) Rabbit mAb recognizes endogenous levels of total GITR protein.

**Source / Purification**

Monoclonal antibody is produced by immunizing animals with recombinant protein specific to the amino terminus 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<sup>+</sup>CD25<sup>+</sup> 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-κB pathway in T cells (7). GITR ligation has been shown to play a role in CD8<sup>+</sup> 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

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11. Petrillo, M.G. et al. (2015) *Autoimmun Rev* 14, 117-26.
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13. Valzasina, B. et al. (2005) *Blood* 105, 2845-51.
14. Stephens, G.L. et al. (2004) *J Immunol* 173, 5008-20.
15. Nishikawa, H. et al. (2008) *Cancer Res* 68, 5948-54.
16. Knee, D.A. et al. (2016) *Eur J Cancer* 67, 1-10.

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## 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 **IHC-Bond:** IHC Leica Bond **IHC-P:** Immunohistochemistry (Paraffin) **IF-IC:** Immunofluorescence (Immunocytochemistry) **FC-FP:** Flow Cytometry (Fixed/Permeabilized) **FC-L:** Flow Cytometry (Live)

## Cross-Reactivity Key

**H:** Human

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