

DESCRIPTION

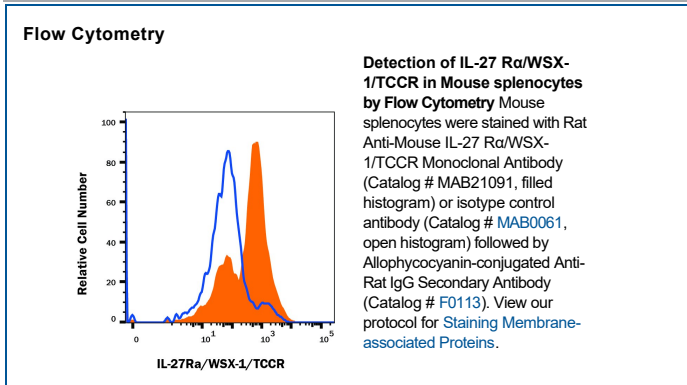
Species Reactivity	Mouse
Specificity	Detects mouse IL-27 R α /WSX-1/TCCR in direct ELISAs and Western blots. In direct ELISAs and Western blots, no cross-reactivity with recombinant mouse gp130 or recombinant human IL-27 R α is observed.
Source	Monoclonal Rat IgG _{2B} Clone # 263503
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse IL-27 R α /WSX-1/TCCR Gly29-Lys510 Accession # O70394
Formulation	Lyophilized from a 0.2 μ m filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 μ m filtered solution in PBS.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the [Technical Information](#) section on our website.

	Recommended Concentration	Sample
Western Blot	1 μ g/mL	Recombinant Mouse IL-27 R α /WSX-1/TCCR Fc Chimera (Catalog # 2109-TC)
Flow Cytometry	2.5 μ g/10 ⁶ cells	Mouse splenocytes
CyTOF-ready	Ready to be labeled using established conjugation methods. No BSA or other carrier proteins that could interfere with conjugation.	

DATA



PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS. For liquid material, refer to CoA for concentration.
Shipping	Lyophilized product is shipped at ambient temperature. Liquid small pack size (-SP) is shipped with polar packs. Upon receipt, store immediately at the temperature recommended below.
Stability & Storage	<p>Use a manual frost freezer and avoid repeated freeze-thaw cycles.</p> <ul style="list-style-type: none"> 12 months from date of receipt, -20 to -70 °C as supplied. 1 month, 2 to 8 °C under sterile conditions after reconstitution. 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

IL-27 R α (also known as WSX-1 and TCCR) is a 85-95 kDa member of the type I, group 2 cytokine receptor family (1-6). Mature IL-27 R α is a type I transmembrane glycoprotein that contains a 486 amino acid (aa) extracellular region, a 21 aa transmembrane segment and a 92 aa cytoplasmic domain. Consistent with type I cytokine receptors, the extracellular region contains four positionally conserved cysteine residues, a WSxWS motif (for receptor folding and ligand binding), and three fibronectin type III repeats. The intracellular domain contains a "box-1" motif that may be involved with Janus kinases (3). In mouse, a soluble 33 kDa splice form that shows a 20 aa substitution for aa 251-623 has been identified (7). The mouse IL-27 R α extracellular region shares 63% amino acid identity with the human IL-27 R α extracellular domain (2, 3). IL-27 R α is expressed in mast cells, endothelial cells, NK cells, macrophages, monocytes, B cells, dendritic cells, and naïve T cells (1, 2, 4, 8). Typical of other class I cytokine receptor chains, the ligand binding IL-27 R α molecule is known to heterodimerize with a signal-transducing subunit (gp130) to form a functional IL-27 receptor (9, 10). In addition, IL-27 R α is reported to complex with CNTFR α and gp130 form a humanin receptor on neurons (7, 11), and to complex with gp130 and IL-6 R to form a receptor for a p28:CLF heterodimeric cytokine on lymphocytes (12). Studies using IL-27 R α /WSX-1^{-/-} mice reveal that IL-27 has the ability to suppress T cell activity during infection, and to mediate an inhibition of both type 1 and type 2 T cell immunity (4, 13, 14). In particular, IL-27 is known to act on naïve T cells, blocking their differentiation into a Th17 phenotype. Notably, cells committed to a Th17 phenotype, although they express a functional IL-27 receptor, are unresponsive to the effects of IL-27 (15). Activated T cells that are CD4⁺ and CD8⁺, and which express the IL-27 receptor, can be induced by IL-27 to form a double-positive CD25⁺ FoxP3⁺ IFN- γ plus IL-10 secreting phenotype that both promotes and suppresses the inflammatory response (16).

References:

1. Villarino, A.V. *et al.* (2004) *J. Immunol.* **173**:715.
2. Chen, Q. *et al.* (2000) *Nature* **407**:916.
3. Sprecher, C.A. *et al.* (1998) *Biochem. Biophys. Res. Commun.* **246**:82.
4. Artis, D. *et al.* (2004) *J. Immunol.* **173**:5626.
5. Yoshida, H. and Y. Miyazaki (2008) *Int. J. Biochem. Cell Biol.* **40**:2379.
6. Yoshida, H. and M. Yoshiyuki (2008) *Immunol. Rev.* **226**:234.
7. Hashimoto, Y. *et al.* (2009) *Biochem. Biophys. Res. Commun.* **389**:95.
8. Holscher, C. *et al.* (2005) *J. Immunol.* **174**:3534.
9. Pflanz, S. *et al.* (2004) *J. Immunol.* **172**:2225.
10. Scheller, J. *et al.* (2005) *Biochem. Biophys. Res. Commun.* **326**:724.
11. Hashimoto, Y. *et al.* (2009) *Mol. Biol. Cell* **20**:2864.
12. Crabe, S. *et al.* (2009) *J. Immunol.* **183**:7692.
13. Villarino, A. *et al.* (2003) *J. Immunol.* **170**:645.
14. Hamano, S. *et al.* (2003) *Immunity* **19**:657.
15. El-behi, M. *et al.* (2009) *J. Immunol.* **183**:4957.
16. Fitzgerald, D.C. *et al.* (2007) *Nat. Immunol.* **8**:1372.