

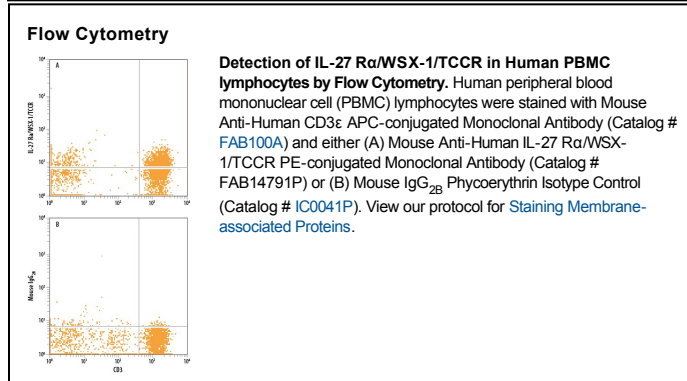
DESCRIPTION	
<b>Species Reactivity</b>	Human
<b>Specificity</b>	Detects human IL-27 R $\alpha$ /WSX-1/TCCR in direct ELISAs and Western blots. In direct ELISAs, no cross-reactivity with recombinant human gp130 or recombinant mouse IL-27 Ra is observed.
<b>Source</b>	Monoclonal Mouse IgG <sub>2B</sub> Clone # 191106
<b>Purification</b>	Protein A or G purified from hybridoma culture supernatant
<b>Immunogen</b>	Mouse myeloma cell line NS0-derived recombinant human IL-27 R $\alpha$ /WSX-1/TCCR Gly34-Lys516 Accession # Q6UWB1
<b>Conjugate</b>	Phycoerythrin Excitation Wavelength: 488 nm Emission Wavelength: 565-605 nm
<b>Formulation</b>	Supplied in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details.  *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

### 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
<b>Flow Cytometry</b>	10 $\mu$ L/10 <sup>6</sup> cells	See Below

### DATA



### PREPARATION AND STORAGE

<b>Shipping</b>	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
<b>Stability &amp; Storage</b>	<b>Protect from light. Do not freeze.</b> <ul style="list-style-type: none"> <li>12 months from date of receipt, 2 to 8 °C as supplied.</li> </ul>

**BACKGROUND**

IL-27 R $\alpha$  (also known as WSX-1 and TCCR) is a 96–100 kDa member of the type I, group 2 cytokine receptor family (1 - 6). Mature IL-27 R $\alpha$  is a type I transmembrane glycoprotein that contains a 484 amino acid (aa) extracellular region, a 21 aa transmembrane segment and a 99 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). One potential alternate splice form has been hypothesized that involves a 58 aa addition to the cytoplasmic domain and, based on mouse, a soluble 33 kDa splice form that shows a 20 aa substitution for aa 257–636 may also occur in human (3, 7). The human IL-27 R $\alpha$  extracellular region shares 63% amino acid identity with the mouse IL-27 R $\alpha$  extracellular domain (2, 3). IL-27 R $\alpha$  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 $\alpha$  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 $\alpha$  is reported to complex with CNTFR $\alpha$  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 $\alpha$ /WSX-1<sup>-/-</sup> 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<sup>+</sup> and CD8<sup>+</sup>, and which express the IL-27 receptor, can be induced by IL-27 to form a double-positive CD25<sup>+</sup> FoxP3<sup>-</sup> IFN- $\gamma$  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. & Y. Miyazaki (2008) *Int. J. Biochem. Cell Biol.* **40**:2379.
6. Yoshida, H. & 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.