

**DESCRIPTION**

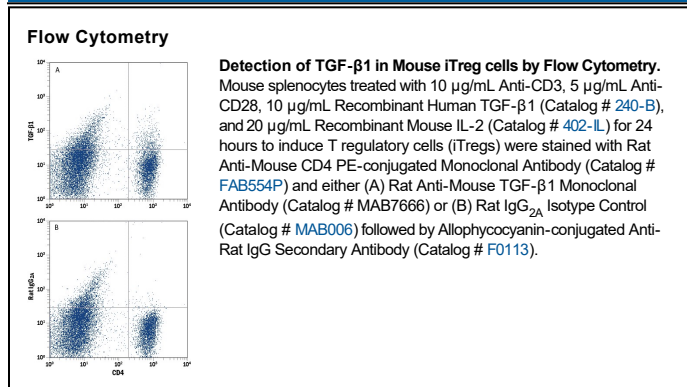
<b>Species Reactivity</b>	Mouse
<b>Specificity</b>	Detects mouse TGF-β1.
<b>Source</b>	Monoclonal Rat IgG <sub>2A</sub> Clone # 860206
<b>Purification</b>	Protein A or G purified from hybridoma culture supernatant
<b>Immunogen</b>	Chinese hamster ovary cell line CHO-derived recombinant mouse TGF-β1 Met1-Ser390 Accession # P04202
<b>Formulation</b>	Lyophilized from a 0.2 μm 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 μm 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.

	<b>Recommended Concentration</b>	<b>Sample</b>
<b>Flow Cytometry</b>	0.25 μg/10 <sup>6</sup> cells	See Below
<b>CyTOF-ready</b>	Ready to be labeled using established conjugation methods. No BSA or other carrier proteins that could interfere with conjugation.	

**DATA**



**PREPARATION AND STORAGE**

<b>Reconstitution</b>	Reconstitute at 0.5 mg/mL in sterile PBS.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
<b>Stability &amp; Storage</b>	<b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b> <ul style="list-style-type: none"> <li>● 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>● 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>● 6 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

**BACKGROUND**

TGF- $\beta$ 1 (Transforming Growth Factor beta 1) is one of three closely related mammalian members of the large TGF- $\beta$  superfamily that share a characteristic cystine knot structure (1-7). TGF- $\beta$ 1, -2 and -3 are highly pleiotropic cytokines that are proposed to act as cellular switches that regulate processes such as immune function, proliferation and epithelial-mesenchymal transition (1-4). Each TGF- $\beta$  isoform has some non-redundant functions; for TGF- $\beta$ 1, mice with targeted deletion show defects in hematopoiesis and endothelial differentiation, and die of overwhelming inflammation (2). Human TGF- $\beta$ 1 cDNA encodes a 390 amino acid (aa) precursor that contains a 29 aa signal peptide and a 361 aa proprotein (8). A furin-like convertase processes the proprotein to generate an N-terminal 249 aa Latency-Associated Peptide (LAP) and a C-terminal 112 aa mature TGF- $\beta$ 1 (8, 9). Disulfide-linked homodimers of LAP and TGF- $\beta$ 1 remain non-covalently associated after secretion, forming the small latent TGF- $\beta$ 1 complex (8-10). Covalent linkage of LAP to one of three latent TGF- $\beta$  binding proteins (LTBPs) creates a large latent complex that may interact with the extracellular matrix (9, 10). TGF- $\beta$  is activated from latency by pathways that include actions of the protease plasmin, matrix metalloproteases, thrombospondin 1 and a subset of integrins (10). Mature human TGF- $\beta$ 1 shares 100% aa identity with pig, dog and cow TGF- $\beta$ 1, and 99% aa identity with mouse, rat and horse TGF- $\beta$ 1. It demonstrates cross-species activity (1). TGF- $\beta$ 1 signaling begins with high-affinity binding to a type II ser/thr kinase receptor termed TGF- $\beta$  RII. This receptor then phosphorylates and activates a second ser/thr kinase receptor, TGF- $\beta$  RI, also known as Activin Receptor-Like Kinase 5 (ALK-5), or alternatively, ALK-1. This complex phosphorylates and activates Smad proteins that regulate transcription (3, 11, 12). Contributions of the accessory receptors Betaglycan (also known as TGF- $\beta$  RIII) and Endoglin, or use of Smad-independent signaling pathways, allow for disparate actions observed in response to TGF- $\beta$  in different contexts (11).

**References:**

1. Derynck, R. and K. Miyazono (2008) Cold Spring Harbor Laboratory Press, 29.
2. Dunker, N. and K. Kriegelstein (2000) Eur. J. Biochem. **267**:6982.
3. Wahl, S.M. (2006) Immunol. Rev. **213**:213.
4. Chang, H. *et al.* (2002) Endocr. Rev. **23**:787.
5. Lin, J.S. *et al.* (2006) Reproduction **132**:179.
6. Hinck, A.P. *et al.* (1996) Biochemistry **35**:8517.
7. Mittl, P.R.E. *et al.* (1996) Protein Sci. **5**:1261.
8. Derynck, R. *et al.* (1985) Nature **316**:701.
9. Miyazono, K. *et al.* (1988) J. Biol. Chem. **263**:6407.
10. Oklu, R. and R. Hesketh (2000) Biochem. J. **352**:601.
11. de Caestecker, M. *et al.* (2004) Cytokine Growth Factor Rev. **15**:1.
12. Zuniga, J.E. *et al.* (2005) J. Mol. Biol. **354**:1052.