

**DESCRIPTION**

<b>Source</b>	<i>E. coli</i> -derived human TGF-beta 3 protein Ala301-Ser412 Accession # P10600.1
<b>N-terminal Sequence Analysis</b>	Ala301
<b>Predicted Molecular Mass</b>	13 kDa

**SPECIFICATIONS**

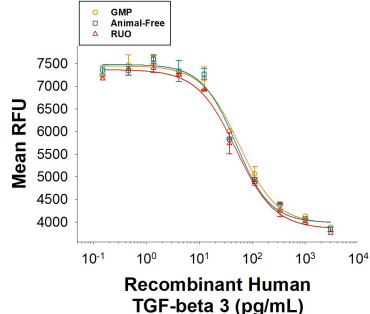
<b>SDS-PAGE</b>	10 kDa, under reducing conditions.
<b>Activity</b>	Measured by its ability to inhibit the IL-4-dependent proliferation of HT-2 mouse T cells. Tsang, M. <i>et al.</i> (1995) Cytokine 7:389. The ED <sub>50</sub> for this effect is 7.50-75.0 pg/mL.
<b>Endotoxin Level</b>	<0.10 EU per 1 µg of the protein by the LAL method.
<b>Purity</b>	>97%, by SDS-PAGE with quantitative densitometry by Coomassie® Blue Staining.
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in Acetonitrile and TFA with Trehalose. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

<b>Reconstitution</b>	Reconstitute the 20 µg size at 100 µg/mL in 4mM HCl. Reconstitute all other sizes at 500 µg/mL in 4mM HCl.
<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>	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <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>• 3 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

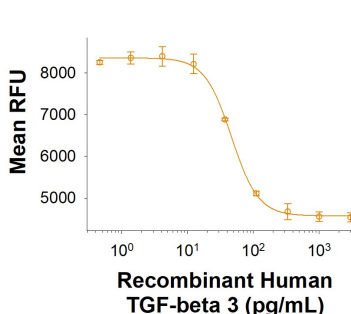
**DATA**

**Bioactivity**



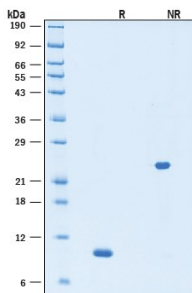
**Equivalent Bioactivity of GMP, Animal-Free, and RUO grades of Recombinant Human TGF-beta 3.** Equivalent bioactivity of GMP (Catalog # BT-TGFB3-GMP), Animal-Free (Catalog # BT-TGFB3-AFL) and RUO (Catalog # BT-TGFB3) grades of Recombinant Human TGF-beta 3 as measured by its ability to inhibit the IL-4-dependent proliferation of HT-2 mouse T cells (orange, green, red, respectively).

**Bioactivity**



**Recombinant Human TGF-beta 3 Protein Bioactivity.** Recombinant Human TGF-beta 3 Protein (Catalog # BT-TGFB3) inhibits recombinant mouse IL-4 induced proliferation in the HT-2 mouse T cell line. The ED<sub>50</sub> for this effect is 7.50-75.0 pg/mL.

**SDS-PAGE**



**Recombinant Human TGF-beta 3 Protein SDS-PAGE.** 2 µg/lane of Recombinant Human TGF-beta 3 Protein (Catalog # BT-TGFB3) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 10 kDa, under reducing conditions.

**BACKGROUND**

TGFβ3 (transforming growth factor-beta 3) is a member of a TGF-β superfamily subgroup that is defined by their structural and functional similarities (1-5). TGF-β3 and its closely related proteins, TGF-β1 and -β2, act as cellular switches to regulate immune function, cell proliferation, and epithelial-mesenchymal transition (4, 6, 7). The non-redundant biological effects of TGF-β3 include involvement in palatogenesis, chondrogenesis, and pulmonary development (1, 2, 7-9). Human TGF-β3 cDNA encodes a 412 amino acid (aa) precursor that contains a 20 aa signal peptide and a 392 aa proprotein. The proprotein is processed by a furinlike convertase to generate a 220 aa latency-associated peptide (LAP) and a 112 aa mature TGF-β3 (10, 11). Mature human TGF-β3 shows 100%, 99%, and 98% aa identity with mouse/dog/horse, rat, and pig TGF-β3, respectively. TGF-β3 is secreted as a complex with LAP. This latent form of TGF-β3 becomes active upon cleavage by plasmin, matrix metalloproteinases, thrombospondin-1, and a subset of integrins (12). TGF-β3 binds with high affinity to TGF-β RII, a type II serine/threonine kinase receptor. This receptor then phosphorylates and activates type I serine/threonine kinase receptors, TGF-β RI or ALK-1, to modulate transcription through Smad phosphorylation (13-15). The divergent biological effects exerted by individual TGF-β isoforms is dependent upon the recruitment of co-receptors (TGF-β RIII and endoglin) and the subsequent initiation of Smad-dependent or -independent signaling pathways (14, 16, 17).

**References:**

1. Barrio, M.C. *et al.* (2014) *Cells Tissues Organs*. [Epub ahead of print; PMID 24861080].
2. Doetschman, T. *et al.* (2012) *Genesis* **50**:59.
3. Mittl, P.R. *et al.* (1996) *Protein Sci.* **5**:1261.
4. Sporn, M.B. (2006) *Cytokine Growth Factor Rev.* **17**:3.
5. Wahl, S.M. *et al.* (2006) *Immunol. Rev.* **213**:213.
6. Chang, H. *et al.* (2002) *Endocr. Rev.* **23**:787.
7. Dunker, N. and K. Kriegelstein (2000) *Eur. J. Biochem.* **267**:6982.
8. Jin, J.Z. *et al.* (2014) *Dev. Dyn.* [Epub ahead of print; PMID 25104574].
9. Tang, Q.O. *et al.* (2009) *Expert Opin. Biol Ther.* **9**:689.
10. Derynck, R. *et al.* (1988) *EMBO J.* **7**:3737.
11. Miyazono, K. *et al.* (1988) *J. Biol. Chem.* **263**:6407.
12. Oklu, R. and R. Hesketh (2000) *Biochem. J.* **352 Pt 3**:601.
13. Cui, X.M. and C.F. Shuler (2000) *Int. J. Dev. Biol.* **44**:397.
14. de Caestecker, M. (2004) *Cytokine Growth Factor Rev.* **15**:1.
15. Nakajima, A. *et al.* (2007) *Dev. Dyn.* **236**:791.
16. Iwata, J. *et al.* (2012) *J. Clin. Invest.* **122**:873.
17. Gatzka, C.E. *et al.* (2010) *Cell. Signal.* **22**:1163.