

Human Transforming Growth Factor β 2 (hTGF- β 2)

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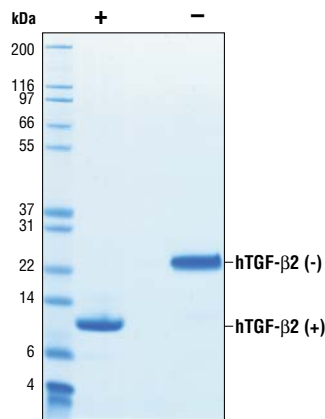
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Source: Recombinant human TGF- β 2 (hTGF- β 2) Ala303-Ser414 (Accession #NP_003229) was expressed in human 293 cells at Cell Signaling Technology.

Molecular Characterization: Recombinant hTGF- β 2 contains no "tags" and the nonglycosylated protein has a calculated MW of 12,719. DTT-reduced protein migrates as a 12 kDa polypeptide and the non-reduced cystine-linked homodimer migrates as a 24 kDa protein. The expected amino-terminal ALDAA of recombinant hTGF- β 2 was verified by amino acid sequencing.

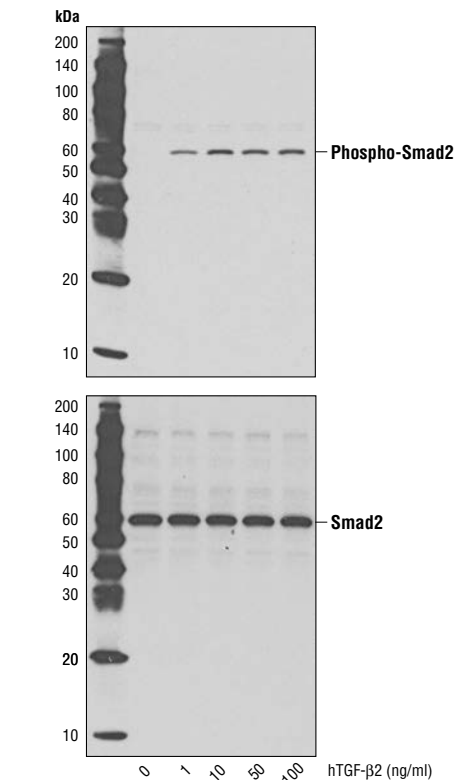
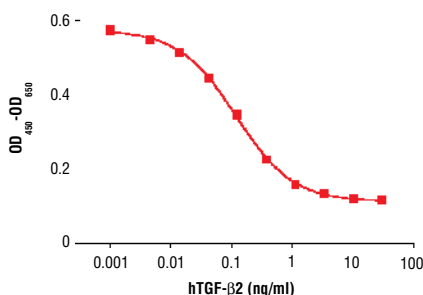
Endotoxin: Less than 0.01 ng endotoxin/1 μ g hTGF- β 2.

Purity: >98% as determined by SDS-PAGE of 6 μ g reduced (+) and non-reduced (-) recombinant hTGF- β 2. All lots are greater than 98% pure.



The purity of recombinant hTGF- β 2 was determined by SDS-PAGE of 6 μ g reduced (+) and non-reduced (-) recombinant hTGF- β 2 and staining overnight with Coomassie Blue.

Bioactivity: The bioactivity of recombinant hTGF- β 2 was determined by assessing inhibition of IL-4 induced HT-2 cell proliferation. The ED₅₀ of each lot is between 0.1-0.3 ng/ml.



Western blot analysis of extracts from HT-1080 cells untreated or treated with TGF- β 2 for 15 minutes, using Phospho-Smad2 (Ser465/467) (138D4) Rabbit mAb #3108 (upper) and Smad2 (86F7) Rabbit mAb #3122 (lower).

◀ The inhibition of IL-4 induced proliferation in HT-2 cells treated with increasing concentrations of hTGF- β 2 was assessed. After 48 hour treatment with hTGF- β 2, cells were incubated with a tetrazolium salt and the OD₄₅₀ - OD₆₅₀ was determined.

Formulation: With carrier: Lyophilized from a 0.22 μ m filtered solution of 35% acetonitrile and 0.1% trifluoroacetic acid containing 20 μ g BSA per 1 μ g hTGF- β 2.

Carrier free: Lyophilized from a 0.22 μ m filtered solution of 35% acetonitrile and 0.1% trifluoroacetic acid.

Reconstitution:

With carrier: Add sterile 4 mM HCl containing 0.1% BSA to a final hTGF- β 2 concentration of greater than 50 μ g/ml. Solubilize for 30 minutes at room temperature with occasional gentle vortexing.

Carrier free: Add sterile 4 mM HCl, or 4 mM HCl containing protein to minimize absorption of hTGF- β 2 to surfaces. Solubilize for 30 minutes at room temperature with occasional gentle vortexing. Stock hTGF- β 2 should be greater than 50 μ g/ml.

Storage: Stable in lyophilized state at -20°C for 1 year after receipt. Sterile stock solutions reconstituted with carrier protein are stable at 4°C for 2 months and at -20°C for 6 months. Avoid repeated freeze-thaw cycles.

Maintain sterility. Storage at -20°C should be in a manual defrost freezer.

Applications: Optimal concentration for the desired application should be determined by the user.

Background: TGF- β 2 is produced by eosinophils, astrocytes, glioblastoma and other cancer derived cell types (1-6). TGF- β 2 inhibits antigen-induced T-cell proliferation, inhibits epithelial cell proliferation, induces mesenchymal cell proliferation and has cell specific effects on apoptosis (1-8). TGF- β 2 binds to T β RII and binding is promoted by T β RIII. T β RI then complexes with T β RII and T β RIII (3,5). Signaling involves phosphorylation of the SMAD proteins (2,3,9). TGF- β 2 also activates Erk2, p38, and JNK pathways (9). Knockout of TGF- β 2 in mice severely impacts heart, lung and eye development (10).

Background References:

- (1) Balzar, S. et al. (2005) *J Allergy Clin Immunol* 115, 110-7.
- (2) Siegel, P.M. and Massagué, J. (2003) *Nat Rev Cancer* 3, 807-21.
- (3) Bierie, B. and Moses, H.L. (2006) *Nat Rev Cancer* 6, 506-20.
- (4) Hinz, S. et al. (2007) *Cancer Res* 67, 8344-50.
- (5) Damstrup, L. et al. (1993) *Br J Cancer* 67, 1015-21.
- (6) Constam, D.B. et al. (1992) *J Immunol* 148, 1404-10.
- (7) Zhang, H. et al. (2008) *Immunology* 124, 304-14.
- (8) Dufour, C. et al. (2008) *Am J Physiol Endocrinol Metab* 294, E794-801.
- (9) Moustakas, A. and Heldin, C.H. (2009) *Development* 136, 3699-714.
- (10) Dünker, N. and Kriegelstein, K. (2000) *Eur J Biochem* 267, 6982-8.