Product Name: Recombinant Human SMAD3 (N-6His-Flag) Catalog #: PEH1176



Summary

Name	Mothers Against Decapentaplegic Homolog 3/SMAD3
Purity	Greater than 95% as determined by reducing SDS-PAGE
Endotoxin level	<1 EU/ μ g as determined by LAL test.
Construction	Recombinant Human Mothers Against Decapentaplegic Homolog 3 is produced by our E.coli expression system and the target gene encoding Ser2-Ser425 is expressed with a 6His, Flag tag at the N-terminus.
Accession #	P84022
Host	E.coli
Species	Human
Predicted Molecular Mass	50.5 KDa
Formulation	Supplied as a 0.2 μm filtered solution of 20mM Tris-HCl, 500mM NaCl, 10% Glycerol, 2mM EDTA, pH 8.0.
Shipping	The product is shipped on dry ice/polar packs. Upon receipt, store it immediately at the temperature listed below.
Stability&Storage	Store at \leq -70°C, stable for 6 months after receipt. Store at \leq -70°C, stable for 3 months under sterile conditions after opening. Please minimize freeze-thaw cycles.
Reconstitution	•

SDS-PAGE image



Background

Alternative Names	Mothers against decapentaplegic homolog 3; MAD homolog 3; Mad3; Mothers against DPP homolog 3; hMAD-3; JV15-2; SMAD family member 3; SMAD 3;
Background	Smad3; hSMAD3; SMAD3; MADH3 Mothers against decapentaplegic homolog 3(SMAD3) is a cytoplasm protein which



belongs to the dwarfin/SMAD family. Smad proteins undergo rapid nuclear translocation upon stimulation by transforming growth factor and in so doing transduce the signal into the nucleus. Receptor-regulated SMAD is an intracellular signal transducer and transcriptional modulator activated by TGF-beta and activin type 1 receptor kinases. SMAD3 binds the TRE element in the promoter region of many genes that are regulated by TGF-beta and, on formation of the SMAD3/SMAD4 complex, activates transcription. It also can form a SMAD3/SMAD4/JUN/FOS complex at the AP-1/SMAD site to regulate TGF-beta-mediated transcription. SMAD3 has an inhibitory effect on wound healing probably by modulating both growth and migration of primary keratinocytes and by altering the TGF-mediated chemotaxis of monocytes. This effect on wound healing appears to be hormone-sensitive.

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Note

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