

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



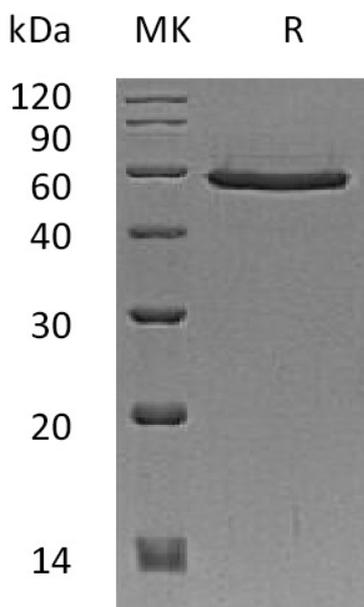
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## Summary

<b>Name</b>	Mothers Against Decapentaplegic Homolog 3/SMAD3
<b>Purity</b>	Greater than 95% as determined by reducing SDS-PAGE
<b>Endotoxin level</b>	<1 EU/μg as determined by LAL test.
<b>Construction</b>	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.
<b>Accession #</b>	P84022
<b>Host</b>	E.coli
<b>Species</b>	Human
<b>Predicted Molecular Mass</b>	50.5 KDa
<b>Formulation</b>	Supplied as a 0.2 μm filtered solution of 20mM Tris-HCl, 500mM NaCl, 10% Glycerol, 2mM EDTA, pH 8.0.
<b>Shipping</b>	The product is shipped on dry ice/polar packs. Upon receipt, store it immediately at the temperature listed below.
<b>Stability&amp;Storage</b>	Store at ≤-70°C, stable for 6 months after receipt. Store at ≤-70°C, stable for 3 months under sterile conditions after opening. Please minimize freeze-thaw cycles.
<b>Reconstitution</b>	

## SDS-PAGE image

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### 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; Smad3; hSMAD3; SMAD3; MADH3

### Background

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.

### Note

For Research Use Only , Not for Diagnostic Use.