SMAD5 Protein, Mouse (sf9)

Cat. No.:	HY-P74540
Synonyms:	Mothers against decapentaplegic homolog 5; Dwarfin-C; SMAD 5
Species:	Mouse
Source:	Sf9 insect cells
Accession:	P97454 (T2-S465)
Gene ID:	17129
Molecular Weight:	Approximately 57 kDa

PROPERTIES

Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of 20 mM Tris, 500 mM Nacl,10% Glycerol, pH8.0. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O.
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background	SMAD5 is a receptor activated Smad (R-Smad), also known as dwarfin C, JV5-1 and MAD5, which belong to the dwarfin/SMAD family. The SMAD5 protein consists of an N-terminal domain responsible for DNA binding, a C-terminal domain primarily used for protein-protein interactions, and a junction region containing moths necessary for ubiquitination degradation. SMAD5 is activated by an activated type I TGF-β receptor, and phosphorylated SMAD5 binds to SMAD4 (common SMAD) to form heterotrimer complexes that enter the nucleus as transcription factors. At the same time, SMAD4 is also bound by inhibitory SMAD (SMAD6 and SMAD7), making R-Smad compete for receptors or interact with Smad4, and target degradation and negative regulation of TGF-β signaling. SMAD5 mainly responds to BMP signaling pathways. During embryonic development, SMAD5 is widely expressed in mice from gastrum formation. During this period, the loss of SMAD5 leads to mutations in the embryo, or defects in the yolk sac outside the embryo, and death in the second trimester. Furthermore, the BMP/SMAD5 pathway can regulate osteogenesis by up-regulating transcription of the transcription factor Runx2, or by targeting multiple cis-acting promoter elements in osteogenic specific genes, including osteocalcin and alkaline phosphatase (ALP). Alps bind to Smad interacting protein 1 (SIP1) and inhibit Smad5-induced SIP1 activation. A significant expansion of primitive multipotent progenitor cells showing normal terminal maturation was reported in embryonic stem cells from Smad5-deficient mice, suggesting that conditional activation or deactivation of Smad5-specific signaling pathways may contribute to hematopoietic and osteogenic tissue engineering[1].

REFERENCES

[1]. Liu B, et al. Smad5: signaling roles in hematopoiesis and osteogenesis. Int J Biochem Cell Biol. 2004 May;36(5):766-70.

[2]. Umans L, et al. Generation of a floxed allele of Smad5 for cre-mediated conditional knockout in the mouse. Genesis. 2003 Sep;37(1):5-11.

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