Recombinant Mouse Follistatin 288 (FS-288)

Catalog Number: 769-FS/CF

DESCRIPTION

Source	*Spodoptera frugiperda*, *Sf* 21 (baculovirus)-derived Gly30-Asn317, with an N-terminal Met Accession # P47931
N-terminal Sequence Analysis	Met
Predicted Molecular Mass	31 kDa

SPECIFICATIONS

SDS-PAGE	35-40 kDa, reducing conditions
Activity	Measured by its ability to neutralize Activin-mediated erythroid differentiation of K562 human chronic myelogenous leukemia cells. The ED_{50} for this effect is 0.1-0.4 µg/mL in the presence of 7.5 ng/mL rhActivin A.
Endotoxin Level	<0.01 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in Acetonitrile and TFA. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 100 µg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 3 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Follistatin (FS) was initially identified as a follicle-stimulating hormone inhibiting substance found in ovarian follicular fluid. It has since been shown that FS is a high-affinity activin-binding protein that can act as an activin antagonist. Two alternatively spliced follistatin mRNAs, encoding mature FS with 288 amino acid (aa) residues (FS-288) and 315 aa residues (FS-315), exist. Natural FS purified from porcine ovaries is primarily a carboxy-terminal truncated form of FS-315 composed of 300 aa residues. FS-288 binds with high-affinity to cell-surface heparan sulfate proteoglycans whereas FS-315 binds with low-affinity. Cell surface-associated FS has been suggested to play a role in the clearance and bioavailability of activin *in vivo*. Besides activin, FS has also been shown to bind with multiple BMPs and to inhibit BMP activity in early Xenopus embryos. FS deficient mice have been shown to have multiple embryonic defects that will result in death shortly after birth. Overexpression of FS can also cause reproductive defects in transgenic mice.

References:

1. Iemura, S. *et al.* (1998) Proc. Natl. Acad. Sci. USA **95**:9337.
2. Guo, Q. (1998) Mol. Endocrinol. **12**:96.
3. Hashimoto, O. *et al.* (1997) J. Biol. Chem. **272**:13835.

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