

DESCRIPTION

Source Chinese Hamster Ovary cell line, CHO-derived
Met1-Lys389
Accession # NP_035848

N-terminal Sequence Analysis Asn29

Predicted Molecular Mass 40.1 kDa

SPECIFICATIONS

SDS-PAGE 42 kDa, reducing conditions

Activity Measured in a cell proliferation/survival assay using IEC-18 rat small intestinal epithelial cells.
The ED₅₀ for this effect is 0.3-1.2 µg/mL.

Endotoxin Level <0.10 EU per 1 µg of the protein by the LAL method.

Purity >85%, by SDS-PAGE under reducing conditions and visualized by silver stain.

Formulation Lyophilized from a 0.2 µm filtered solution in PBS, NaCl, EDTA and CHAPS with BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE

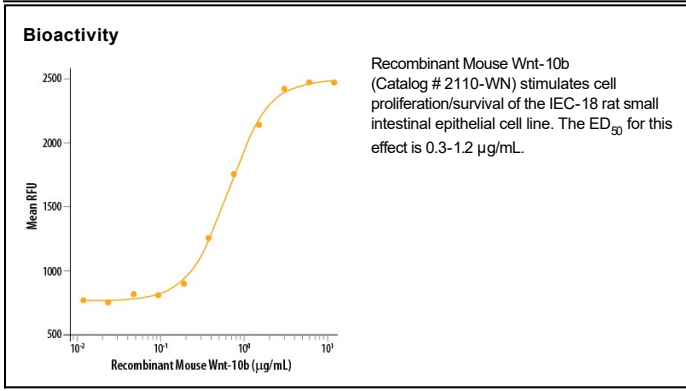
Reconstitution Reconstitute at 100 µg/mL in PBS.

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.

DATA



BACKGROUND

Wnt-10b (also known as Wnt-12) is a 42-44 kDa member of the Wnt family of secreted, highly conserved, cysteine-rich glycoproteins that play important roles in vertebrate pattern formation, cell fate decision, axon guidance, and tumor formation (1-3). Mouse Wnt-10b cDNA encodes a 361 amino acid (aa) precursor that contains a 28 aa signal sequence plus a 361 aa mature protein that contains two glycosylation sites, three potential phosphorylation sites, and a potential palmitoylation site (3, 4). A short isoform is reported that lacks aa 143-238 of the precursor (1, 4). Mouse Wnt-10b shares 97-98% aa identity with human, rat, equine, porcine and canine Wnt-10b. Wnt-10b plays a critical role in maintaining mesenchymal stem cells and determining whether they differentiate to adipocytes or osteoblasts (5-7). Mouse Wnt-10b deletion produces age-dependent loss of bone mass due to defective production of osteoblasts, while transgenic over-expression increases postnatal osteoblast differentiation and inhibits adipocyte differentiation (5-7). Ectopic expression of Wnt-10b in an obesity and diabetes-prone background, such as the ob/ob mouse, inhibits obesity (8). In mouse skeletal muscle, Wnt-10b is expressed inversely with SREBP1c and increases insulin sensitivity (9). In humans, a mis-sense polymorphism is responsible for a malformation of hands and feet, while a C256Y inactivating mutation is associated with severe early-onset obesity (10, 11). Wnt-10 is mainly produced by stem cells and pre-osteoblasts, but also by adult bone marrow CD8⁺ T lymphocytes stimulated with parathyroid hormone (12). In some hepatocellular carcinomas, Wnt-10b can inhibit cancer cell growth, but in others, it can act synergistically with FGFs to stimulate cell growth (13). Several Wnts, including Wnt-10b, are expressed in both normal and/or malignant colon tissues (14). As a key regulator, Wnt signaling plays a major role in the process of colon carcinogenesis (15).

References:

1. Wang, J. and G.M. Shackleford (1996) *Oncogene* **13**:1537.
2. Kato, M. (2008) *Curr. Drug Targets* **9**:565.
3. Hausmann, G. *et al.* (2007) *Nat. Rev. Mol. Cell Biol.* **8**:331.
4. Swiss Prot Accession # P48614.
5. Stevens, J.R. *et al.* (2010) *J. Bone Miner. Res.* **25**:2138.
6. Bennett, C.N. *et al.* (2007) *J. Bone Miner. Res.* **22**:1924.
7. Bennett, C.N. *et al.* (2005) *Proc. Natl. Acad. Sci. USA* **102**:3324.
8. Wright, W.S. *et al.* (2007) *Diabetes* **56**:295.
9. Abiola, M. *et al.* (2009) *PLoS ONE* **4**:e8509.
10. Ugur, S.A. and A. Tolun (2008) *Hum. Mol. Genet.* **17**:2644.
11. Christodoulides, C. *et al.* (2006) *Diabetologia* **49**:678.
12. Terauchi, M. *et al.* (2009) *Cell Metab.* **10**:229.
13. Yoshikawa, H. *et al.* (2007) *Mol. Biol. Cell* **18**:4292.
14. Holcombe, R.F. *et al.* (2002) *J. Clin. Pathol. Mol. Pathol.* **55**:220.
15. Cynthia K. *et al.* (2007) *Proc. Natl. Acad. Sci. USA*.