

DESCRIPTION

Source Chinese Hamster Ovary cell line, CHO-derived
Asn30-Lys364
Accession # Q9QYS1

N-terminal Sequence Analysis Asn30

Predicted Molecular Mass 38 kDa

SPECIFICATIONS

SDS-PAGE 43-63 kDa, reducing conditions

Activity Measured by its ability to induce alkaline phosphatase production by C3H10T1/2 mouse embryonic fibroblast cells.
The ED₅₀ for this effect is 2-12 µg/mL.

Endotoxin Level <0.10 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 PBS, NaCl, EDTA and CHAPS with BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE

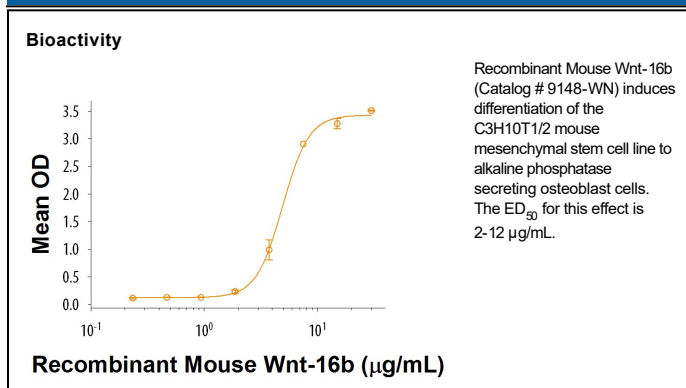
Reconstitution Reconstitute at 500 µg/mL in PBS containing at least 0.1% human or bovine serum albumin.

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-16 is a 40 kDa protein within the Wnt family of secreted, highly conserved, cysteine-rich, palmitoylated cell signaling glycoproteins that play important roles in vertebrate developmental pattern formation, cell fate decision, axon guidance, and tumor formation (1-3). Mature mouse Wnt-16 shares 92% and 97% aa sequence identity with human and rat Wnt-16, respectively. Wnt-16 is expressed by uterine stromal cells adjacent to the luminal epithelium during implantation (4). It is up-regulated during the first embryonic lymphoid progenitor differentiation (5). Congenital heart defects correlate with elevated Wnt-16 in mouse embryos and human amniotic fluid (6). It is secreted by osteoblasts and inhibits monocyte differentiation into osteoclasts, thereby contributing to cortical bone thickness and bone mineral density [1279, 7]. Wnt-16 is over-expressed in cells undergoing replicative senescence, and it is up-regulated in articular cartilage by injury and osteoarthritis (9, 10). Wnt-16b expression in skin is up-regulated and enhances cell survival in human basal cell carcinomas (11). Its expression is also up-regulated by DNA damage (radiation and chemotherapy) in stroma surrounding prostate tumors, causing enhanced survival and treatment resistance in the tumor cells (12). Pre-B acute lymphoblastic leukemia with t(1;19) translocation, creating an E2A-Pbx1 fusion protein, also causes up-regulation of Wnt-16 that confers resistance to apoptosis (13, 14). Wnt-16 signaling through both canonical and JNK-mediated (non-canonical) pathways is reported (9-11).

References:

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