Recombinant Human Wnt-16b

Catalog Number: 7790-WN/CF

DESCRIPTION

Source	Chinese Hamster Ovary cell line, CHO-derived Asn30-Lys365 Accession # Q9UBV4
N-terminal Sequence Analysis	Asn30
Predicted Molecular Mass	37.6 kDa

SPECIFICATIONS

SDS-PAGE	45-65 kDa, reducing conditions
Activity	Measured in a cell proliferation/survival assay using 3T3-L1 mouse embryonic fibroblast adipose-like cells. The ED_{50} for this effect is 0.4-1.6 µg/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>80%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS, EDTA and CHAPS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 200 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.

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). Wnt-16a and Wnt-16b isoforms in humans differ in the signal sequence and the first two amino acids (aa) of the mature protein (2, 3). Wnt-16b is the more conserved isoform and is widely expressed, while Wnt-16a is expressed mainly in the human pancreas (3). Mature human Wnt-16b shares 92%, 93%, and 95% aa sequence identity with mouse/rat, rabbit/porcine/equine, and bovine Wnt-16, respectively. Wnt-16 expression is detected on uterine stroma 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). Low cortical bone thickness and bone mineral density correlate with deletion of Wnt-16 in mice and a Wnt-16 missense SNP in humans (7). Wnt-16 is over-expressed in cells undergoing replicative senescence, and is up-regulated in articular cartilage by injury and osteoarthritis (8, 9). Wnt-16b expression in skin is up-regulated in human basal cell carcinomas, enhancing cell survival (10). 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 (11). 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 (12, 13). Wnt-16 signaling through both canonical and JNK-mediated (non-canonical) pathways is reported (8-10).

References:

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