Recombinant Mouse Exostosin-Like 2/EXTL2 Protein (His Tag)

Catalog Number: PKSM041014

Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

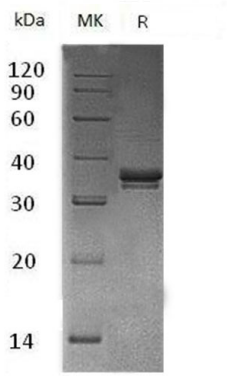
Description

Species	Mouse
Source	HEK293 Cells-derived Mouse Exostosin-Like 2/EXTL2 protein Asn43-Met330, with an N-terminal His
Calculated MW	33.6 kDa
Observed MW	35 kDa
Accession	Q9ES89
Bio-activity	Not validated for activity

Properties

Purity	> 95 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μg of the protein as determined by the LAL method.
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80 ℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from a 0.2 μm filtered solution of 20mM Tris-HCl, 150mM NaCl, pH 8.0. Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 95 % as determined by reducing SDS-PAGE.

Background

Exostosin-like 2 (EXTL2) is a member of the exostosin (EXT)-related family which contains five members: EXT1, EXT2, EXTL1, EXTL2, and EXTL3. Studies have shown that EXT gene family members have the activities of heparan sulfate-synthesizing glycosyltransferases. EXT1 and EXT2, which have been identified as causal genes for hereditary multiple exostoses, have HS-GlcAT-II and GlcNAcT-II activities. EXTL1 has GlcNAcT-II activity and EXTL3 has GlcNAcT-I and -II activities. EXTL2 has GlcNAcT-I and N-acetylgalactosaminyltransferase activities, and transfers a GlcNAc residue to the tetrasaccharide linkage region when this region is phosphorylated by a xylose kinase 1 (FAM20B) and thereby terminate chain elongation. In mice, lack of EXTL2 causes glycosaminoglycan (GAG) overproduction and structural changes of GAGs associated with pathological processes.