abcam

Product datasheet

Recombinant human Noggin protein ab198773

2 Images

Description

Product name	Recombinant human Noggin protein
Biological activity	Measured by ability of ab198773 to inhibit BMP-4-induced alkaline phosphatase production by C2C12 mouse myoblast cells. The ED_{50} is <20 ng/ml in the presence of 30 ng/ml of Human BMP-4
Purity	> 70 % SDS-PAGE.
Endotoxin level	< 1.000 Eu/µg
Expression system	Freestyle 293-F cells
Accession	Q13253
Protein length	Full length protein
Animal free	No
Nature	Recombinant
Species	Human
Sequence	QHYLHIRPAPSDNLPLVDLIEHPDPIFDPKEKDLNETLLRSL LGGHYDPG FMATSPPEDRPGGGGGAAGGAEDLAELDQLLRQRPSGA MPSEIKGLEFSE GLAQGKKQRLSKKLRRKLQMWLWSQTFCPVLYAWNDLG SRFWPRYVKVGS CFSKRSCSVPEGMVCKPSKSVHLTVLRWRCQRRGGQR CGWIPIQYPIISE CKCSC
Predicted molecular weight	28 kDa
Amino acids	28 to 232
Additional sequence information	Mature disulfide-linked homodimer without signal peptide. NM_005450.

Specifications

Our Abpromise guarantee covers the use of **ab198773** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Applications	Functional Studies
	SDS-PAGE

Form	Lyophilized

Preparation and Storage

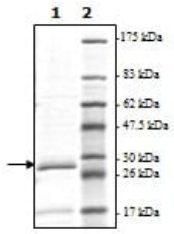
Stability and Storage	Shipped at 4°C. Store at -20°C or -80°C. Avoid freeze / thaw cycle. pH: 8.00 Constituents: 0.61% Tris, 6.96% Sodium chloride, 0.02% Potassium chloride, 0.1% BSA Lyophilized from a 0.2 μm filtered solution This product is an active protein and may elicit a biological response in vivo, handle with caution.
Reconstitution	Reconstitute in sterile water with 0.1% BSA to a final concentration of 0.1 mg/ml. Final formulation will be 50 mM Tris, pH 8.0, 1.2 M NaCl, 2.7 mM KCl, 0.1% BSA. This solution can then be diluted into other buffers. To maximize product collection from vial surface, vortex briefly and then spin down to recollect the liquid. Store reconstituted protein in aliquots at -20°C to -80°C for up to 6 months. Avoid freeze/thaw cycles.

General Info

Function	Essential for cartilage morphogenesis and joint formation. Inhibitor of bone morphogenetic proteins (BMP) signaling which is required for growth and patterning of the neural tube and somite.
Involvement in disease	Defects in NOG are a cause of symphalangism proximal syndrome (SYM1) [MIM:185800]. SYM1 is characterized by the hereditary absence of the proximal interphalangeal (PIP) joints (Cushing symphalangism). Severity of PIP joint involvement diminishes towards the radial side. Distal interphalangeal joints are less frequently involved and metacarpophalangeal joints are rarely affected whereas carpal bone malformation and fusion are common. In the lower extremities, tarsal bone coalition is common. Conducive hearing loss is seen and is due to fusion of the stapes to the petrous part of the temporal bone. Defects in NOG are the cause of multiple synostoses syndrome type 1 (SYNS1) [MIM:186500]; also known as synostoses, multiple, with brachydactyly/symphalangism-brachydactyly syndrome. SYNS1 is characterized by tubular-shaped (hemicylindrical) nose with lack of alar flare, otosclerotic deafness, and multiple progressive joint fusions commencing in the hand. The joint fusions are progressive, commencing in the fifth proximal interphalangeal joint in early childhood (or at birth in some individuals) and progressing in an ulnar-to-radial and proximal-to-distal direction. With increasing age, ankylosis of other joints, including the cervical vertebrae, hips, and humeroradial joints, develop. Defects in NOG are the cause of tarsal-carpal coalition syndrome (TCC) [MIM:186570]. TCC is an autosomal dominant disorder characterized by fusion of the carpals, tarsals and phalanges, short first metacarpals causing brachydactyly, and humeroradial fusion. TCC is allelic to SYM1, and different mutations in NOG can result in either TCC or SYM1 in different families. Defects in NOG are a cause of stapes ankylosis with broad thumb and toes (SABTS) [MIM:184460]; also known as Teunissen-Cremers syndrome. SABTS is a congenital autosomal dominant disorder that includes hyperopia, a hemicylindrical nose, broad thumbs, great toes, and other minor skeletal anomalies but lacked carpal and tarsal fusion and symphalangism. Defects in NOG are the cause of brachydactyly type B2 (BDB2) [MIM:611377]. BDB2 is a subtype of brachydactyly characterized by hypoplasia/aplasia of distal phalanges in combination with distal symphalangism, fusion of carpal/tarsal bones, and partial cutaneous syndactyly.
Sequence similarities	Belongs to the noggin family.

Cellular localization	Secreted.

Images

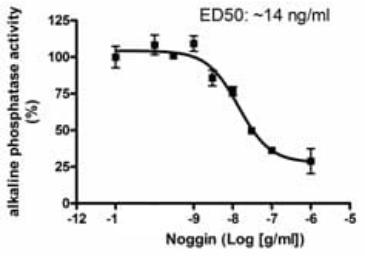


SDS-PAGE - Recombinant human Noggin protein (ab198773)

4-20% SDS-PAGE analysis of ab198773 with Coomassie staining.

Lane 1: 3 µg ab198773

Lane 2: Protein marker



Functional Studies - Recombinant human Noggin protein (ab198773)

Functional analysis of ab198773.

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