Product datasheet

Anti-Noggin antibody ab16054

Overview

Product name: Anti-Noggin antibody
Description: Rabbit polyclonal to Noggin
Host species: Rabbit
Tested applications: Suitable for: ICC/IF, IHC-P, WB
Unsuitable for: IHC-Fr
Species reactivity: Reacts with: Mouse, Human
Predicted to work with: Horse, Chicken, Xenopus laevis
Immunogen: Synthetic peptide corresponding to Human Noggin aa 1-100 (internal sequence) conjugated to keyhole limpet haemocyanin.
(Peptide available as ab16380)

Positive control

Purchase matching WB positive control:
Recombinant human Noggin protein

This antibody gave a positive signal in both Human and Mouse Noggin Recombinant protein.

Properties

Form: Liquid
Storage instructions: Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C or -80°C. Avoid freeze / thaw cycle.
Storage buffer: pH: 7.40
Preservative: 0.02% Sodium azide
Constituent: PBS

Purity: Immunogen affinity purified
Clonality: Polyclonal
Isotype: IgG
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. Conductive 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.
**Western blot - Anti-Noggin antibody (ab16054)**

**All lanes**: Anti-Noggin antibody (ab16054) at 1 µg/ml

**Lane 1**: Noggin Human Recombinant Protein

**Lane 2**: Noggin Mouse Recombinant Protein

Lysates/proteins at 0.1 µg per lane.

**Secondary**

**All lanes**: Goat polyclonal to Rabbit IgG - H&L - Pre-Adsorbed (HRP) at 1/3000 dilution

Developed using the ECL technique.

Performed under reducing conditions.

**Predicted band size**: 26 kDa

**Observed band size**: 26,35 kDa

*why is the actual band size different from the predicted?*

**Exposure time**: 1 minute

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**Western blot - Anti-Noggin antibody (ab16054)**

**All lanes**: Anti-Noggin antibody (ab16054) at 1 µg/ml

**Lane 1**: Noggin Mouse Recombinant Protein

**Lane 2**: Noggin Mouse Recombinant Protein with Human Noggin peptide (ab16380) at 1 µg/ml

Lysates/proteins at 0.01 µg per lane.

**Predicted band size**: 26 kDa
Western blot - Anti-Noggin antibody (ab16054)

Anti-Noggin antibody (ab16054) at 1 µg/ml + Recombinant human Noggin protein (ab73756) at 1 µg

**Secondary**
Goat Anti-Rabbit IgG H&L (HRP) preadsorbed (ab97080) at 1/5000 dilution

Developed using the ECL technique.

Performed under reducing conditions.

**Predicted band size:** 26 kDa

**Exposure time:** 4 minutes

ab16054 staining Noggin in paraffin-embedded human liver tissue, showing a cytoplasmic and/or membranous distribution in both hepatocytes and bile duct cells. Paraffin embedded tissue was incubated with ab16054 (1/175 dilution) for 30 minutes at room temperature. Antigen retrieval was performed by heat induction in citrate buffer pH 6. ab16054 was tested in a tissue microarray (TMA) containing a wide range of normal and cancer tissues as well as a cell microarray consisting of a range of commonly used, well characterised human cell lines.
Immunohistochemical analysis of human small intestine tissue, labeling Noggin with ab16054. Tissue was formaldehyde fixed, treated with EDTA (pH 8.6) at 100°C for 20 minutes for heat-mediated antigen retrieval and blocked with 3% Hydrogen Peroxide for 10 minutes at 25°C. Incubation with ab16054 (diluted 1/400) was performed for 20 minutes at 25°C.

Immunohistochemical analysis of mouse kidney tissue, labeling Noggin with ab16054. Tissue was paraformaldehyde fixed, treated with Citrate buffer for heat-mediated antigen retrieval and blocked with Serum Free Protein Block for 20 minutes. Incubation with ab16054 (diluted 1/2500) was performed for 15 hours at 4°C.
ICC/IF image of Noggin stained primary myoblast cells. The cells were 4% PFA fixed (10 min) and then incubated in 10% normal donkey serum / 0.3M glycine in 0.1% PBS-Tritonx100 for 1h to permeabilise the cells and block non-specific protein-protein interactions. The cells were then incubated with the antibody (ab16054, 1µg/ml) overnight at +4°C. The secondary antibody (green) was Alexa Fluor® 488 donkey anti-rabbit IgG (H+L) used at a 1/1000 dilution for 1h. DAPI was used to stain the cell nuclei (blue) at a concentration of 1.43µM.

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