abcam

Product datasheet

Recombinant Human Collagen XI alpha 2/COL11A2 protein ab158168

1 Image

Description

Product name	Recombinant Human Collagen XI alpha 2/COL11A2 protein
Expression system	Wheat germ
Protein length	Protein fragment
Animal free	No
Nature	Recombinant
Species	Human
Sequence	APPVDVLRALRFPSLPDGVRRAKGICPADVAYRVARP AQLSAPTRQLFPG GFPKDFSLLTVVRTRPGLQAPLLTLYSAQGVRQLGLEL GRPVRFLYEDQT
Amino acids	29 to 128
Tags	GST tag N-Terminus

Specifications

Our Abpromise guarantee covers the use of **ab158168** in the following tested applications.

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

Applications	ELISA Western blot
Form	Liquid
Additional notes	Protein concentration is above or equal to 0.05 mg/ml. This product was previously labelled as Collagen XI alpha 2

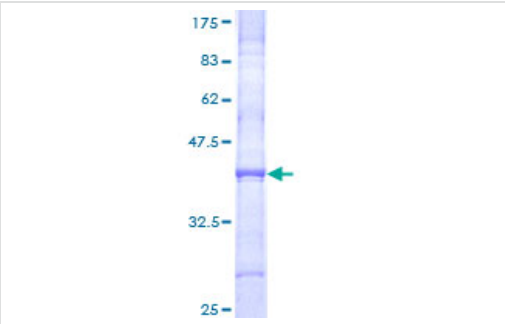
Preparation and Storage

Stability and Storage	Shipped on dry ice. Upon delivery aliquot and store at -80ºC. Avoid freeze / thaw cycles. pH: 8.00 Constituents: 0.31% Glutathione, 0.79% Tris HCl

General Info

Function	May play an important role in fibrillogenesis by controlling lateral growth of collagen II fibrils.
Involvement in disease	Defects in COL11A2 are the cause of Stickler syndrome type 3 (STL3) [MIM:184840]. STL3 is an autosomal dominant non-ocular form of Stickler syndrome. Classical Stickler syndrome associates ocular signs with more or less complete forms of Pierre Robin sequence, bone disorders and sensorineural deafness. Ocular symptoms are absent in STL3. Robin sequence includes an opening in the roof of the mouth (a cleft palate), a large tongue (macroglossia), and a small lower jaw (micrognathia). Bones are affected by slight platyspondylisis and large, often defective epiphyses. Juvenile joint laxity is followed by early signs of arthrosis. The degree of hearing loss varies among affected individuals and may become more severe over time. Syndrome expressivity is variable. Defects in COL11A2 are the cause of autosomal recessive otospondylomegaepiphyseal dysplasia (OSMED) [MIM:215150]. OSMED is a skeletal dysplasia accompanied by severe hearing loss. The phenotype overlaps that of autosomal dominant skeletal disorders (Stickler and Marshall syndromes) but can be distinguished by disproportionately short limbs and lack of ocular involvement. Defects in COL11A2 are the cause of Weissenbacher-Zweymueller syndrome (WZS) [MIM:277610]. WZS is an autosomal dominant disorder allelic with STL3 and OSMED. WZS is also referred to as heterozygous OSMED. Defects in COL11A2 are the cause of deafness autosomal dominant type 13 (DFNA13) [MIM:601868]. DFNA13 is a form of sensorineural hearing loss. Sensorineural deafness results from damage to the neural receptors of the inner ear, the nerve pathways to the brain, or the area of the brain that receives sound information. Defects in COL11A2 are the cause of deafness autosomal recessive type 53 (DFNB53) [MIM:609706].
Sequence similarities	Belongs to the fibrillar collagen family. Contains 1 fibrillar collagen NC1 domain. Contains 1 TSP N-terminal (TSPN) domain.
Post-translational modifications	Prolines at the third position of the tripeptide repeating unit (G-X-Y) are hydroxylated in some or all of the chains. A disulfide-bonded peptide called proline/arginine-rich protein or PARP is released from the N-terminus during extracellular processing and is subsequently retained in the cartilage matrix from which it can be isolated in significant amounts.
Cellular localization	Secreted > extracellular space > extracellular matrix.

Images



SDS-PAGE - Recombinant Human Collagen XI alpha 2/COL11A2 protein (ab158168)

ab158168 on a 12.5% SDS-PAGE stained with Coomassie Blue.

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