

Product datasheet

Native Human Collagen I protein ab7533

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Description

Product name	Native Human Collagen I protein
Purity	> 95 % n/a. This product has been prepared from human placenta by pepsin digestion and is chromatographically and immunologically pure.
Expression system	Native
Accession	P02452
Protein length	Full length protein
Animal free	No
Nature	Native
Species	Human
Predicted molecular weight	95 kDa

Specifications

Our [Abpromise guarantee](#) covers the use of **ab7533** in the following tested applications.

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

Applications	Western blot Immunoprecipitation ELISA
Form	Liquid
Additional notes	This product is free from other collagens, human serum proteins and non-collagen extracellular matrix proteins. This product reacts with anti-Collagen Type I. Reaction with anti-Collagen II, III, IV, V or VI is negligible (typically less than 1% cross reactivity was detected by ELISA).

Preparation and Storage

Stability and Storage	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. Preservative: 0.01% Sodium azide Constituent: 4.1% Sodium acetate
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General Info

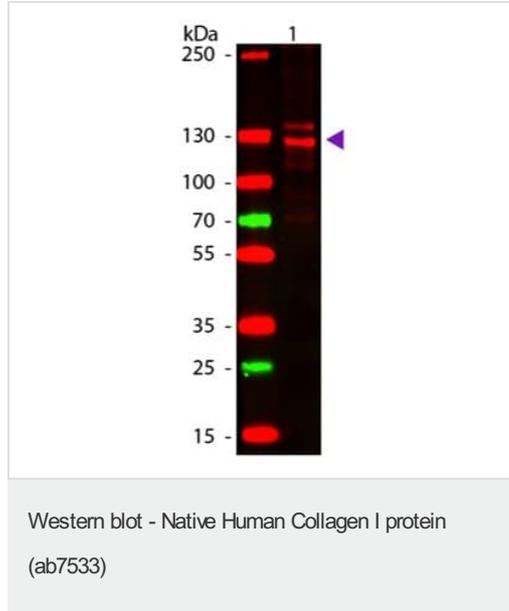
Function	Type I collagen is a member of group I collagen (fibrillar forming collagen).
Tissue specificity	Forms the fibrils of tendon, ligaments and bones. In bones the fibrils are mineralized with calcium hydroxyapatite.
Involvement in disease	<p>Defects in COL1A1 are the cause of Caffey disease (CAFFD) [MIM:114000]; also known as infantile cortical hyperostosis. Caffey disease is characterized by an infantile episode of massive subperiosteal new bone formation that typically involves the diaphyses of the long bones, mandible, and clavicles. The involved bones may also appear inflamed, with painful swelling and systemic fever often accompanying the illness. The bone changes usually begin before 5 months of age and resolve before 2 years of age.</p> <p>Defects in COL1A1 are a cause of Ehlers-Danlos syndrome type 1 (EDS1) [MIM:130000]; also known as Ehlers-Danlos syndrome gravis. EDS is a connective tissue disorder characterized by hyperextensible skin, atrophic cutaneous scars due to tissue fragility and joint hyperlaxity. EDS1 is the severe form of classic Ehlers-Danlos syndrome.</p> <p>Defects in COL1A1 are the cause of Ehlers-Danlos syndrome type 7A (EDS7A) [MIM:130060]; also known as autosomal dominant Ehlers-Danlos syndrome type VII. EDS is a connective tissue disorder characterized by hyperextensible skin, atrophic cutaneous scars due to tissue fragility and joint hyperlaxity. EDS7A is marked by bilateral congenital hip dislocation, hyperlaxity of the joints, and recurrent partial dislocations.</p> <p>Defects in COL1A1 are a cause of osteogenesis imperfecta type 1 (OI1) [MIM:166200]. A dominantly inherited connective tissue disorder characterized by bone fragility and blue sclerae. Osteogenesis imperfecta type 1 is non-deforming with normal height or mild short stature, and no dentinogenesis imperfecta.</p> <p>Defects in COL1A1 are a cause of osteogenesis imperfecta type 2A (OI2A) [MIM:166210]; also known as osteogenesis imperfecta congenita. A connective tissue disorder characterized by bone fragility, with many perinatal fractures, severe bowing of long bones, undermineralization, and death in the perinatal period due to respiratory insufficiency.</p> <p>Defects in COL1A1 are a cause of osteogenesis imperfecta type 3 (OI3) [MIM:259420]. A connective tissue disorder characterized by progressively deforming bones, very short stature, a triangular face, severe scoliosis, grayish sclera, and dentinogenesis imperfecta.</p> <p>Defects in COL1A1 are a cause of osteogenesis imperfecta type 4 (OI4) [MIM:166220]; also known as osteogenesis imperfecta with normal sclerae. A connective tissue disorder characterized by moderately short stature, mild to moderate scoliosis, grayish or white sclera and dentinogenesis imperfecta.</p> <p>Genetic variations in COL1A1 are a cause of susceptibility to osteoporosis (OSTEOP) [MIM:166710]; also known as involutional or senile osteoporosis or postmenopausal osteoporosis. Osteoporosis is characterized by reduced bone mass, disruption of bone microarchitecture without alteration in the composition of bone. Osteoporotic bones are more at risk of fracture.</p> <p>Note=A chromosomal aberration involving COL1A1 is found in dermatofibrosarcoma protuberans. Translocation t(17;22)(q22;q13) with PDGF.</p>
Sequence similarities	<p>Belongs to the fibrillar collagen family.</p> <p>Contains 1 fibrillar collagen NC1 domain.</p> <p>Contains 1 VWFC domain.</p>
Post-translational modifications	Proline residues at the third position of the tripeptide repeating unit (G-X-Y) are hydroxylated in some or all of the chains. Proline residues at the second position of the tripeptide repeating unit (G-X-Y) are hydroxylated in some of the chains.

O-linked glycan consists of a Glc-Gal disaccharide bound to the oxygen atom of a post-translationally added hydroxyl group.

Cellular localization

Secreted > extracellular space > extracellular matrix.

Images



Native Human Collagen I protein (ab7533) at 1/1000 dilution + Human Collagen at 0.05 µg

Secondary

DyLight™ 649 Rabbit Secondary Antibody at 1/20000 dilution

Observed band size: 130 kDa

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