

Product datasheet

Human Collagen Type I ELISA Kit ab285250

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Overview

Product name Human Collagen Type I ELISA Kit

Detection method Colorimetric

Precision

Intra-assay

Sample	n	Mean	SD	CV%
Overall				< 8%

Inter-assay

Sample	n	Mean	SD	CV%
Overall				< 10%

Sample type Serum, Plasma, Other biological fluids, Tissue Homogenate

Assay type Sandwich

Sensitivity < 0.188 ng/ml

Range 0.313 ng/ml - 20 ng/ml

Assay duration Multiple steps standard assay

Species reactivity **Reacts with:** Human

Product overview Human Collagen Type I ELISA kit (ab285250, E4617) is a sandwich ELISA assay for the quantitative measurement of COL1 in human serum, plasma and cell culture supernatants samples. The density of color is proportional to the amount of COL1 captured from the samples.

No significant cross-reactivity or interference between COL1 and analogues was observed.

Platform Microplate (12 x 8 well strips)

Properties

Storage instructions Store at +4°C. Please refer to protocols.

Components	96 tests	96 tests
25X Wash buffer	1 x 30ml	1 x 30ml
Antibody dilution buffer	1 x 10ml	1 x 10ml
Biotin/Detection antibody (Concentrated)	1 x 120µl	1 x 120µl
HRP-Streptavidin Conjugate (SABC)	1 x 120µl	1 x 120µl
Lyophilized Standard	2 vials	2 vials
Micro ELISA Plate	1 unit	1 unit
Plate Sealer	5 units	5 units
SABC dilution buffer	1 x 10ml	1 x 10ml
Sample / Standard Dilution Buffer	1 x 20ml	1 x 20ml
Stop Solution	1 x 10ml	1 x 10ml
TMB Substrate	1 x 10ml	1 x 10ml

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

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.

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.

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.

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.

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.

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. 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.

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.

Note=A chromosomal aberration involving COL1A1 is found in dermatofibrosarcoma protuberans. Translocation t(17;22)(q22;q13) with PDGF.

Sequence similarities

Belongs to the fibrillar collagen family.
Contains 1 fibrillar collagen NC1 domain.
Contains 1 VWFC domain.

Post-translational modifications

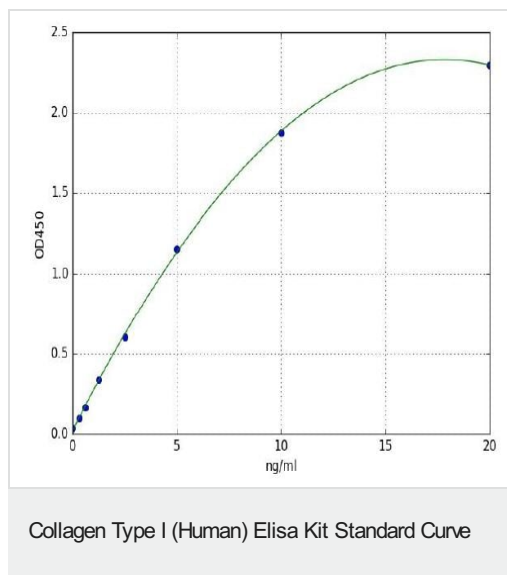
Proline residues at the third position of the tripeptide repeating unit (G-X-Y) are hydroxylated in some or all of the chains. Proline residues at the second position of the tripeptide repeating unit (G-X-Y) are hydroxylated in some of the chains.

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



Typical Standard Curve: These standard curves are for demonstration only. A standard curve must be run with each assay

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