10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

Collagen I Recombinant Rabbit mAb

Catalog # AP96688

Product Information

Application	WB, IHC-P, IHC-F, IF
Primary Accession	P02452
Other Accession	P02452
Reactivity	Rat, Human, Mouse
Host	Rabbit
Clonality	Recombinant
Calculated MW	138911
Physical State	Liquid
Immunogen	KLH conjugated synthetic peptide derived from human Collagen I
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SIMILARITY	Belongs to the fibrillar collagen family. Contains 1 fibrillar collagen NC1 domain. Contains 1 VWFC domain.
SUBUNIT	Trimers of one alpha 2(I) and two alpha 1(I) chains. Interacts with MRC2. Interacts with TRAM2. Subcellular Location : Secreted, extracellular space, extracellular matrix.
Post-translational modifications	Proline residues at the third position of the tripeptide repeating unit (G-X-P) are hydroxylated in some or all of the chains. Proline residues at the second position of the tripeptide repeating unit (G-P-X) 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.
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 2 (OI2) [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.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	Collagens are highly conserved throughout evolution and are characterised by an uninterrupted "Glycine X Y" triplet repeat that is a necessary part of the triple helical structure. Type I collagen (95 kDa) is found in bone, cornea, skin and tendon. Mutations in the encoding gene are associated with osteogenesis imperfecta, Ehlers Danlos syndrome, and idiopathic osteoporosis. Reciprocal translocations between chromosomes 17 and 22, where this gene and the gene for Platelet-derived growth factor beta are located, are associated with a particular type of skin tumor called dermatofibrosarcoma protuberans, resulting from unregulated expression of the growth factor.

Additional Information

Gene ID	1277
Other Names	CAFYD; EDSARTH1; EDSC; OI1; OI2; OI3; OI4; Col1a-1; Cola-1; Cola1; Mov-13; Mov13; CO1A1_BOVIN; COL1A1; Alpha-1 type I collagen; CO1A2_CANLF; COL1A2; Alpha-2 type I collagen; collagen type I alpha 1 chain; collagen, type I, alpha 1
Dilution	WB=1:500-2000,IHC-P=1:100-500,IHC-F=1:100-500,IF=1:100-500,ICC/IF=1:50-200,Flow-Cyt=1:50-100
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

Protein Information

Name	COL1A1
Function	Type I collagen is a member of group I collagen (fibrillar forming collagen).
Cellular Location	Secreted, extracellular space, extracellular matrix {ECO:0000255|PROSITE-ProRule:PRU00793}
Tissue Location	Forms the fibrils of tendon, ligaments and bones. In bones the fibrils are

mineralized with calcium hydroxyapatite

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.