

Collagen I A1(PT0817R) PT® Rabbit mAb

CatalogNo: YM8576 **Recombinant** 

Key Features

Host Species

- Rabbit

Reactivity

- Human, Mouse, Rat

Applications

- WB, IHC, IF, IP, ELISA

MW

- 139kD (Calculated)
220kD (Observed)

Isotype

- IgG, Kappa

Recommended Dilution Ratios

IHC 1:200-1:1000

WB 1:2000-1:10000

IF 1:200-1:1000

ELISA 1:5000-1:20000

IP 1:50-1:200

Storage

Storage* -15°C to -25°C/1 year (Do not lower than -25°C)

Basic Information

Clonality Monoclonal

Clone Number PT0817R

Immunogen Information

Specificity Endogenous

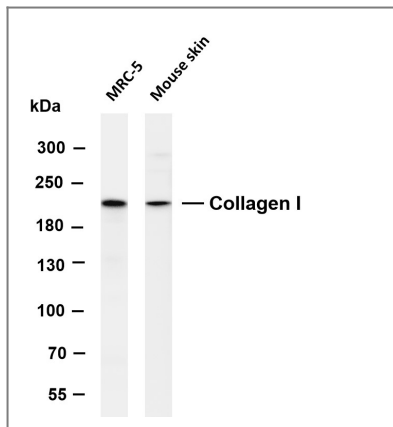
Target Information

Gene name	COL1A1		
Protein Name	Collagen I α 1 (Cleaved-Ala1218)		
	Organism	Gene ID	UniProt ID
	Human	1277 ;	P02452 ;
	Mouse	12842 ;	P11087 ;
	Rat	29393 ;	P02454 ;
Cellular Localization	Secreted, extracellular space, extracellular matrix .		
Tissue specificity	Forms the fibrils of tendon, ligaments and bones. In bones the fibrils are mineralized with calcium hydroxyapatite.		

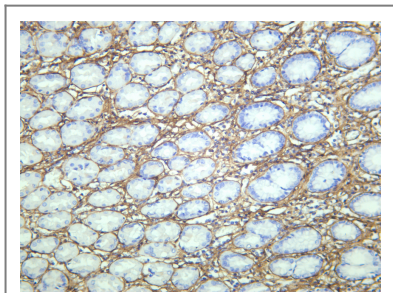
Function

Disease: A chromosomal aberration involving COL1A1 is a cause of dermatofibrosarcoma protuberans (DFSP) [MIM:607907]. Translocation t(17;22)(q22;q13) with PDGF. DFSP is an uncommon, locally aggressive, but rarely metastasizing tumor of the deep dermis and subcutaneous tissue. It typically occurs during early or middle adult life and is most frequently located on the trunk and proximal extremities. Disease: 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. Disease: Defects in COL1A1 are a cause of osteogenesis imperfecta type I (OI-I) [MIM:166200]. OI-I is a dominantly inherited serious newborn disease characterized by bone fragility, normal stature, little or no deformity, blue sclerae and hearing loss in 50% of families. Dentinogenesis imperfecta is rare and may distinguish a subset of OI type I (formation of dentine). Disease: Defects in COL1A1 are a cause of osteogenesis imperfecta type II (OI-II) [MIM:166210]; also known as osteogenesis imperfecta congenita. OI-II is lethal in the perinatal period and is characterized by calvarial mineralization, beaded ribs, compressed femurs, marked long bone deformity and platyspondyly (congenital flattening of the vertebral bodies). Disease: Defects in COL1A1 are a cause of osteogenesis imperfecta type III (OI-III) [MIM:259420]; also called progressively deforming osteogenesis imperfecta with normal sclerae. OI-III is characterized by progressively deforming bones, usually with moderate deformity at birth, sclerae is variable in color, dentinogenesis imperfecta and hearing loss are common. The stature is very short. Disease: Defects in COL1A1 are a cause of osteogenesis imperfecta type IV (OI-IV) [MIM:166220]. OI-IV is characterized by normal sclerae, moderate to mild deformity and variable short stature. Dentinogenesis imperfecta is common and hearing loss occurs in some patients. Disease: Defects in COL1A1 are the cause of Caffey disease [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. Disease: 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. Disease: Genetic variations in COL1A1 are associated with susceptibility to involutional osteoporosis [MIM:166710]; also known as senile osteoporosis or postmenopausal osteoporosis. Osteoporosis is characterized by reduced bone mineral density, disruption of bone microarchitecture, and the alteration of the amount and variety of non-collagenous proteins in bone. Osteoporotic bones are more at risk of fracture. Function: Type I collagen is a member of group I collagen (fibrillar forming collagen). online information: Collagen type I alpha-1 chain mutations, online information: Type-I collagen entry, PTM: O-linked glycan consists of a Glc-Gal disaccharide bound to the oxygen atom of a post-translationally added hydroxyl group. PTM: Proline residues at the third position of the tripeptide repeating unit (G-X-Y) are hydroxylated in some or all of the chains. similarity: Belongs to the fibrillar collagen family. similarity: Contains 1 VWFC domain. subunit: Trimers of one alpha 2(I) and two alpha 1(I) chains. Interacts with MRC2. tissue specificity: Forms the fibrils of tendon, ligaments and bones. In bones the fibrils are mineralized with calcium hydroxyapatite.

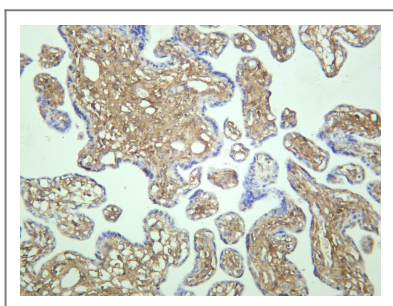
Validation Data



Various whole cell lysates were separated by 4-8% SDS-PAGE, and the membrane was blotted with anti-Collagen I antibody. The HRP-conjugated Goat anti-Rabbit IgG(H + L) antibody was used to detect the antibody. Lane 1: MRC-5 Lane 2: Mouse skin Predicted band size: 139kDa Observed band size: 220kDa



Human stomach was stained with anti-Collagen I rabbit antibody



Human placenta was stained with anti-Collagen I rabbit antibody

Contact information

Orders: order@immunoway.com
 Support: tech@immunoway.com
 Telephone: 877-594-3616 (Toll Free), 408-747-0185
 Website: <http://www.immunoway.com>
 Address: 2200 Ringwood Ave San Jose, CA 95131 USA



Please scan the QR code to access additional product information:

Collagen I
A1(PT0817R) PT®
Rabbit mAb

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