Revision 1

Store at -20C

#30565

COL3A1 Antibody

Cell Signaling TECHNOLOGY®

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For Research Use Only. Not for Use in Diagnostic Procedures.

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, IP	H	Endogenous	200	Rabbit	#P02461	1281

Product Usage Information

Application	Dilution
Western Blotting	1:1000
Immunoprecipitation	1:50

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA and 50% glycerol. Store at –20°C. *Do not aliquot the antibody.*

Specificity/Sensitivity

COL3A1 Antibody recognizes endogenous levels of total COL3A1 protein.

Source / Purification

Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Pro1218 of human COL3A1 protein. Antibodies are purified by peptide affinity chromatography.

Background

The extracellular matrix (ECM) is a complex network of macromolecules that provides structural tissue support to cells in the basement membrane and interstitial matrix. It is composed of many molecules including proteins, glycoproteins, proteoglycans, and polysaccharides (1,2). One of the major proteins that comprises the ECM, and the human body, is collagen. Collagens are a large family of proteins. They are trimeric molecules composed of three alpha polypeptide chains that form a triple helix structure that is characteristic of all collagens (3). The large family of collagens is divided into three subgroups: fibrillar collagens, non-fibril forming collagens, and fibril-associated collagens. These subgroups differ in their structure and supramolecular assembly (3).

Collagen 3 alpha 1 (COL3A1) is a major fibrillar collagen composed of three identical alpha-1 chains. It is present in most soft tissues, along with COL1A1, and is particularly high in tissues exhibiting elastic properties, such as the cardiac arterial wall and skin (4). Heterozygous mutations in the *COL3A1* gene that cause missense mutation of a critical glycine residue in the triple helical domain of the alpha-1 chain result in vascular Ehlers-Danlos syndrome (vEDS). This mutation interferes with the ability of the alpha-1 chain to form collagen fibrils and thus disrupts macromolecular assembly of collagen fibers. vEDS is a severe and life-threatening disease as patients have a propensity for rupture of large arteries (4).

Increased amounts of type III COL3A1 are found in many fibrotic conditions, such as lung, liver, kidney fibrosis, and systemic sclerosis (5,6).

Background References

1. Barkan, D. et al. (2010) *Eur J Cancer* 46, 1181-8.
2. Hynes, R.O. (2009) *Science* 326, 1216-9.
3. Ricard-Blum, S. (2011) *Cold Spring Harb Perspect Biol* 3, a004978.
4. Kuivaniemi, H. and Tromp, G. (2019) *Gene* 707, 151-71.
5. Karsdal, M.A. et al. (2017) *Adv Drug Deliv Rev* 121, 43-56.
6. Ricard-Blum, S. et al. (2018) *Matrix Biol* 68-9, 122-49.

Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Western Blot Buffer

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

Applications Key

W: Western Blotting **IP:** Immunoprecipitation

Cross-Reactivity Key

H: Human

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