SANTA CRUZ BIOTECHNOLOGY, INC.

COL2A1 (N-19): sc-7764

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The extensive family of COL gene products (collagens) is composed of several chain types, including fibril-forming interstitial collagens (types I, II, III and V) and basement membrane collagens (type IV), each type containing multiple isoforms. Collagens are fibrous, extracellular matrix proteins with high tensile strength and are the major components of connective tissue, such as tendons and cartilage. All collagens contain a triple helix domain and frequently show lateral self-association in order to form complex connective tissues. Several collagens also play a role in cell adhesion, important for maintaining normal tissue architecture and function. In cartilage, Collagen Type II constitutes the bulk of the fibril. Sensitization with Collagen Type II induces an erosive poly-arthritis in rats, mice and higher primates which can resemble rheumatoid arthritis and relapsing polychrondritis.

CHROMOSOMAL LOCATION

Genetic locus: COL2A1 (human) mapping to 12q13.11; Col2a1 (mouse) mapping to 15 F1.

SOURCE

COL2A1 (N-19) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the N-terminus of the mature chain of Collagen α1 Type II of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-7764 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

COL2A1 (N-19) is recommended for detection of Collagen α1 Type II of mouse, rat, human and *Xenopus* origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for COL2A1 siRNA (h): sc-35081, COL2A1 siRNA (m): sc-35082, COL2A1 shRNA Plasmid (h): sc-35081-SH, COL2A1 shRNA Plasmid (m): sc-35082-SH, COL2A1 shRNA (h) Lentiviral Particles: sc-35081-V and COL2A1 shRNA (m) Lentiviral Particles: sc-35082-V.

Molecular Weight of Collagen Type II: 190 kDa.

Positive Controls: HISM cell lysate: sc-2229, CCD-1064Sk cell lysate: sc-2263 or ECV304 cell lysate: sc-2269.

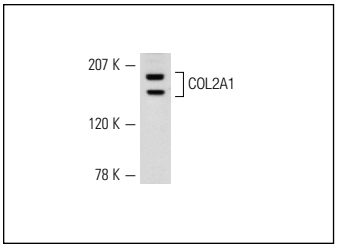
STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

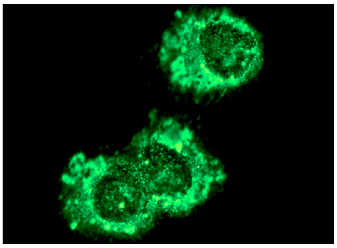
RESEARCH USE

For research use only, not for use in diagnostic procedures.

DATA



COL2A1 (N-19): sc-7764. Western blot analysis of COL2A1 expression in HISM whole cell lysate.



COL2A1 (N-19): sc-7764. Immunofluorescence staining of methanol-fixed ECV304 cells showing cytoplasmic localization.

SELECT PRODUCT CITATIONS

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2. Oldershaw, R.A., et al. 2008. Notch signaling through Jagged-1 is necessary to initiate chondrogenesis in human bone marrow stromal cells but must be switched off to complete chondrogenesis. Stem Cells 26: 666-674.
3. Neven, E., et al. 2009. Adequate phosphate binding with lanthanum carbonate attenuates arterial calcification in chronic renal failure rats. Nephrol. Dial. Transplant. 24: 1790-1799.
4. Wang, J., et al. 2009. Transcription factor Nfat1 deficiency causes osteoarthritis through dysfunction of adult articular chondrocytes. J. Pathol. 219: 163-172.
5. Quintana, L., et al. 2009. Early tissue patterning recreated by mouse embryonic fibroblasts in a three-dimensional environment. Tissue Eng. Part A 15: 45-54.
6. Neven, E., et al. 2010. Chondrocyte rather than osteoblast conversion of vascular cells underlies medial calcification in uremic rats. Arterioscler. Thromb. Vasc. Biol. 30: 1741-1750.
7. Jin, E.J., et al. 2010. TGF-β3 inhibits chondrogenesis by suppressing precartilage condensation through stimulation of N-cadherin shedding and reduction of cRREB-1 expression. Mol. Cells 29: 425-432.
8. Fujita, K., et al. 2012. Age-related expression of MCP-1 and MMP-3 in mouse intervertebral disc in relation to TWEAK and TNF-α stimulation. J. Orthop. Res. 30: 599-605.