Datasheet for ABIN6138841

anti-COL4A1 antibody (AA 1445-1669)

2 Images

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Overview

Quantity:	100 µL
Target:	COL4A1
Binding Specificity:	AA 1445-1669
Reactivity:	Human
Host:	Rabbit
Clonality:	Polyclonal
Conjugate:	This COL4A1 antibody is un-conjugated
Application:	Western Blotting (WB)

Product Details

Immunogen:	Recombinant fusion protein containing a sequence corresponding to amino acids 1445-1669 of human COL4A1 (NP_001836.2).
Sequence:	GFLVTRHSQT IDDPQCPSGT KILYHGYSLL YVQGNERAHG QDLGTAGSCL RKFSTMPFLF CNINNVCNFA SRNDYSYWLS TPEPMPMSMA PITGENIRPF ISRCAVCEAP AMVMAVHSQT IQIPPCPSGW SSLWIGYSFV MHTSAGAEGS GQALASPGSC LEEFRSAPFI ECHGRGTCNY YANAYSFWLA TIERSEMFKK PTPSTLKAGE LRTHVSRCQV CMRRT
Isotype:	IgG
Cross-Reactivity:	Human
Characteristics:	Polyclonal Antibodies
Purification:	Affinity purification

Target Details

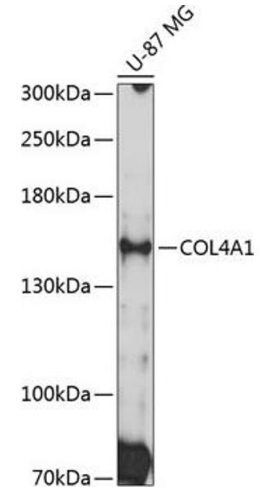
Target:	COL4A1
Alternative Name:	COL4A1 (COL4A1 Products)
Background:	This gene encodes a type IV collagen alpha protein. Type IV collagen proteins are integral components of basement membranes. This gene shares a bidirectional promoter with a paralogous gene on the opposite strand. The protein consists of an amino-terminal 7S domain, a triple-helix forming collagenous domain, and a carboxy-terminal non-collagenous domain. It functions as part of a heterotrimer and interacts with other extracellular matrix components such as perlecans, proteoglycans, and laminins. In addition, proteolytic cleavage of the non-collagenous carboxy-terminal domain results in a biologically active fragment known as arresten, which has anti-angiogenic and tumor suppressor properties. Mutations in this gene cause porencephaly, cerebrovascular disease, and renal and muscular defects. Alternative splicing results in multiple transcript variants.,COL4A1,BSVD,RATOR,Stem Cells,COL4A1
Molecular Weight:	127 kDa/160 kDa
Gene ID:	1282
UniProt:	P02462
Pathways:	Skeletal Muscle Fiber Development, Growth Factor Binding

Application Details

Application Notes:	WB,1:500 - 1:2000
Restrictions:	For Research Use only

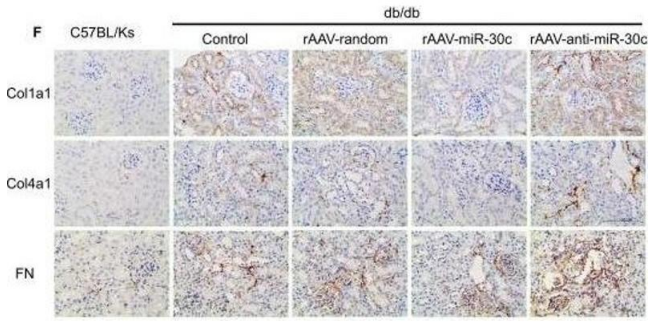
Handling

Format:	Liquid
Buffer:	PBS with 0.02 % sodium azide,50 % glycerol, pH 7.3.
Preservative:	Sodium azide
Precaution of Use:	This product contains Sodium azide: a POISONOUS AND HAZARDOUS SUBSTANCE which should be handled by trained staff only.
Storage:	-20 °C
Storage Comment:	Store at -20°C. Avoid freeze / thaw cycles.



Western Blotting

Image 1. Western blot analysis of extracts of U-87 MG cells, using COL4 Antibody (0710) at 1:3000 dilution._Secondary antibody: HRP Goat Anti-Rabbit IgG (H+L) (ABIN1684268 and ABIN3020597) at 1:10000 dilution._Lysates/proteins: 25 µg per lane._Blocking buffer: 3 % nonfat dry milk in TBST._Detection: ECL Enhanced Kit (RM00021)._Exposure time: 30s.



Immunohistochemistry

Image 2. MiR-30c reduced fibrosis in DN via reducing TGF-β1 secretion from TECs. (A) Expression levels of Snail1 and TGF-β1 in renal cortex detected by immunohistochemical staining (400x). Scale bar, 100 µm. (B) TGF-β1 protein levels in renal cortex lysates measured by ELISA and normalized to total protein concentration in homogenates. (C) Relative TGF-β1 mRNA level in renal cortex from mice measured by real-time PCR. (D) Representative images of immunofluorescence staining for EdU (green), α-SMA (red), and Hoechst (blue). Scale bar, 50 µm. (E) Relative col1a1, col4a1, and FN expression levels in renal cortex from mice measured by real-time PCR. (F) Relative col1a1, col4a1, and FN expression levels in renal cortex from mice measured by immunohistochemical staining (400x). Scale bar, 100 µm. (G) Relative col1a1, col4a1, and FN expression levels in renal cortex from mice measured by Western blot. Data are representative of three experiments. Data are expressed as mean ± SEM, n = 8, *P < 0.05 vs. C57BL/Ks. #P < 0.05 vs. db/db control. (H) Schematic representation of the association among miR-30c, EMT, and tubulointerstitial fibrosis in DN. In tubular epithelial cells (TECs) of DN, miR-30c was decreased due to hyperglycemia. The loss of miR-30c resulted in Snail1 activation, which drove the EMT program in TECs. Snail1-driven EMT promoted epitheliums

to dedifferentiate into fibroblasts. Moreover, TECs released TGF-β1 to the microenvironment which promoted both the transitional and resident fibroblasts proliferation and activation. Thus, plenty of myofibroblasts accumulated and produced dominant extracellular matrix (ECM) components, contributing to pathologic process of tubulointerstitial fibrosis in DN. - figure provided by CiteAb. Source: PMID28127848