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# Collagen IV (PT0131R) PT<sup>™</sup> Rabbit mAb

CatalogNo: YM8073 Recombinant R

## Key Features

Host Species

Rabbit

MW • 160kD (Calculated) 200kD (Observed) Reactivity

Human

IsotypeIgG,Kappa

Applications
• WB,IHC,IF,ELISA

### **Recommended Dilution Ratios**

IHC 1:200-1000 WB 1:1000-5000 IF 1:200-1000 ELISA 1:5000-20000

#### **Storage**

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

Formulation PBS, 50% glycerol, 0.05% Proclin 300, 0.05% BSA

#### **Basic Information**

Clonality	Monoclonal
Clone Number	PT0131R

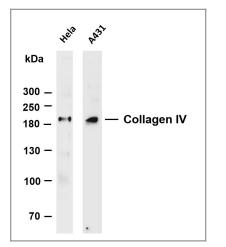
#### Immunogen Information

Specificity Endogenous

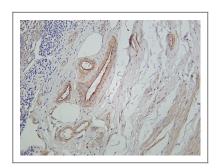
# Target Information

I larget mit	ination		
Gene name	COL4A1		
Protein Name	Collagen Type IV <b>Organism</b>	Gene ID	UniProt ID
	Human	<u>1282;</u>	<u>P02462;</u>
Cellular Localization	Cytoplasmic		
Tissue specificity	Highly expressed in placenta.		
Function	Highly expressed in placenta. Disease:Defects in COL4A1 are a cause of brain small vessel disease with hemorrhage [MIM:607595]. Brain small vessel diseases underlie 20 to 30 percent of ischemic strokes and a larger proportion of intracerebral hemorrhages. Inheritance is autosomal dominant.,Disease:Defects in COL4A1 are a cause of porencephaly type 1 [MIM:175780]; also known as encephaloclastic porencephaly. Porencephaly is a term used for any cavitation or cerebrospinal fluid-filled cyst in the brain. Porencephaly type 1 is usually unilateral and results from focal destructive lesions such as fetal vascular occlusion or birth trauma. Inheritance is autosomal dominant.,Disease:Defects in COL4A1 are the cause of hereditary angiopathy with nephropathy, aneurysms, and muscle cramps (HANAC) [MIM:611773]. The clinical renal manifestations include hematuria and bilateral large cysts. Histologic analysis revealed complex basement membrane defects in kidney and skin. The systemic angiopathy appears to affect both small vessels and large arteries.,Domain:Alpha chains of type IV collagen have a non-collagenous domain (NC1) at their C-terminus, frequent interruptions of the G-X-Y repeats in the long central triple-helical domain (which may cause flexibility in the triple helix), and a short N-terminal triple-helical domain (which may cause flexibility in the triple helix), and a short N-terminal triple-helical surface proteoglycans and entactin/nidogen. Potently inhibits endothelial cells.PTM:Lysines at the third position of the tripeptide repeating unit (G-X-Y) are hydroxylated in all cases and bind carbohydrates.,PTM:Prolines at the third position of the tripeptide repeating unit (G-X-Y) are hydroxylated in some or all of the chains.,PTM:The trimeric structure of the NC1 domains may be stabilized by covalent bonds between Lys and Met residues.,PTM:Lysines at the third position of the tripeptide repeating unit (G-X-Y) are hydroxylated in all cases and bind carbohydrates.,PTM:Prolines at the third po		

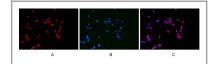
## Validation Data



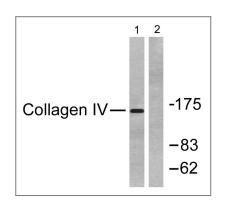
Various whole cell lysates were separated by 4-20% SDS-PAGE, and the membrane was blotted with anti-Collagen IV antibody. The HRP-conjugated Goat anti-Rabbit IgG(H + L) antibody was used to detect the antibody. Lane 1: Hela Lane 2: A431 Predicted band size: 160kDa Observed band size: 200kDa



Human appendix was stained with Anti-Collagen IV rabbit antibody



Immunofluorescence analysis of HEK293. Picture A: Collagen IV antibody (red). Picture B: DAPI (blue). Picture C: Merge of A+B



Western blot analysis of lysates from HeLa cells, using Collagen IV Antibody. The lane on the right is blocked with the synthesized peptide.

#### **Contact information**

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Please scan the QR code to access additional product information: **Collagen IV** (PT0131R) PT<sup>™</sup> Rabbit mAb