

COL4A3 Human

Description: Human $\alpha 3$ chain of collagen IV; identical with the antigen called "glomerular basal membrane antigen" (GBM). Recombinant antigen for solid (ELISA) and fluid phase diagnostic assays. Binds IgG-type human auto-antibodies. Calculated Molecular weight: 43,591 Dalton. Calculated isoelectric point: pH 8.9. cDNA coding for a minicollagen version of the human collagen IV $\alpha 3$ chain fused to a hexa-histidine purification tag. The term minicollagen designates the removal of most of the epitope-less triplehelical collagenous region (situated between the N-terminal 7S domain and the C-terminal noncollagenous NC1 domain), which is a requirement for recombinant production of this antigen.

Catalog #: PRPS-394

For research use only.

Synonyms: Collagen alpha-3(IV) chain, Goodpasture antigen, COL4A3, Glomerular Basal Membrane, GBM.

Source: Sf9 insect cells.

Physical Appearance: Sterile Filtered clear solution.

Formulation:

The protein solution (0.6 mg/ml) contains 20mM Hepes, pH 8.0, and 4M Urea

Stability:

Recommendations for storage buffer: Ionic strength between 50 and 100 mM, neutral to slightly alkaline pH and 4 M urea as dissociating agent. Storage temperature is -70

Usage:

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Introduction:

Type IV collagen is a major structural component of basement membranes. It is a multimeric protein composed of 3 α subunits, which are encoded by 6 different genes, $\alpha 1$ through $\alpha 6$. Each of these α subunits can form a triple helix structure with 2 other subunits to form type IV collagen. The Goodpasture syndrome is a condition in which autoantibodies bind to the collagen molecules in the basement membranes of alveoli and glomeruli. The epitopes that elicit these autoantibodies are restricted basically to the non-collagenous C-terminal domain of the protein. There are numerous alternative transcripts that appear to be unique to the human COL4A3 and alternative splicing is limited to the six exons that encode this C-terminal domain. COL4A3 is also linked to an autosomal recessive form of Alport syndrome. The mutations contributing to the Alport syndrome are also situated within the exons that encode this C-terminal region. COL4A3 is organized in a head-to-head conformation with another type IV collagen gene so that each gene pair shares a common promoter. Several exons of COL4A3 are interspersed with exons of an uncharacterized gene which is on the opposite strand.

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