

Product datasheet

Human Collagen III peptide ab101725

Overview

Product name Human Collagen III peptide

Description

Nature Synthetic

Amino Acid Sequence

Species Human

Specifications

Our [Abpromise guarantee](#) covers the use of **ab101725** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Applications Blocking - Blocking peptide for Anti-Collagen III antibody ([ab83829](#))

Purity 70 - 90% by HPLC.

Form Liquid

Additional notes

- First try to dissolve a small amount of peptide in either water or buffer. The more charged residues on a peptide, the more soluble it is in aqueous solutions.
- If the peptide doesn't dissolve try an organic solvent e.g. DMSO, then dilute using water or buffer.
- Consider that any solvent used must be compatible with your assay. If a peptide does not dissolve and you need to recover it, lyophilise to remove the solvent.
- Gentle warming and sonication can effectively aid peptide solubilisation. If the solution is cloudy or has gelled the peptide may be in suspension rather than solubilised.
- Peptides containing cysteine are easily oxidised, so should be prepared in solution just prior to use.

Preparation and Storage

Stability and Storage Shipped at 4°C. Upon delivery aliquot and store at -20°C or -80°C. Avoid repeated freeze / thaw cycles.

Information available upon request.

General Info

Function	Collagen type III occurs in most soft connective tissues along with type I collagen.
Involvement in disease	<p>Defects in COL3A1 are a cause of Ehlers-Danlos syndrome type 3 (EDS3) [MIM:130020]; also known as benign hypermobility syndrome. EDS is a connective tissue disorder characterized by hyperextensible skin, atrophic cutaneous scars due to tissue fragility and joint hyperlaxity. EDS3 is a form of Ehlers-Danlos syndrome characterized by marked joint hyperextensibility without skeletal deformity.</p> <p>Defects in COL3A1 are the cause of Ehlers-Danlos syndrome type 4 (EDS4) [MIM:130050]. EDS is a connective tissue disorder characterized by hyperextensible skin, atrophic cutaneous scars due to tissue fragility and joint hyperlaxity. EDS4 is the most severe form of the disease. It is characterized by the joint and dermal manifestations as in other forms of the syndrome, characteristic facial features (acrogeria) in most patients, and by proneness to spontaneous rupture of bowel and large arteries. The vascular complications may affect all anatomical areas. Defects in COL3A1 are a cause of susceptibility to aortic aneurysm abdominal (AAA) [MIM:100070]. AAA is a common multifactorial disorder characterized by permanent dilation of the abdominal aorta, usually due to degenerative changes in the aortic wall. Histologically, AAA is characterized by signs of chronic inflammation, destructive remodeling of the extracellular matrix, and depletion of vascular smooth muscle cells.</p>
Sequence similarities	<p>Belongs to the fibrillar collagen family.</p> <p>Contains 1 fibrillar collagen NC1 domain.</p> <p>Contains 1 VWFC domain.</p>
Post-translational modifications	<p>Proline residues at the third position of the tripeptide repeating unit (G-X-Y) are hydroxylated in some or all of the chains.</p> <p>O-linked glycan consists of a Glc-Gal disaccharide bound to the oxygen atom of a post-translationally added hydroxyl group.</p>
Cellular localization	Secreted > extracellular space > extracellular matrix.

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