

Collagen type XII degradation by MMP12

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Introduction

Reactome is open-source, open access, manually curated and peer-reviewed pathway database. Pathway annotations are authored by expert biologists, in collaboration with Reactome editorial staff and cross-referenced to many bioinformatics databases. A system of evidence tracking ensures that all assertions are backed up by the primary literature. Reactome is used by clinicians, geneticists, genomics researchers, and molecular biologists to interpret the results of high-throughput experimental studies, by bioinformaticians seeking to develop novel algorithms for mining knowledge from genomic studies, and by systems biologists building predictive models of normal and disease variant pathways.

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Literature references

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Reactome database release: 88

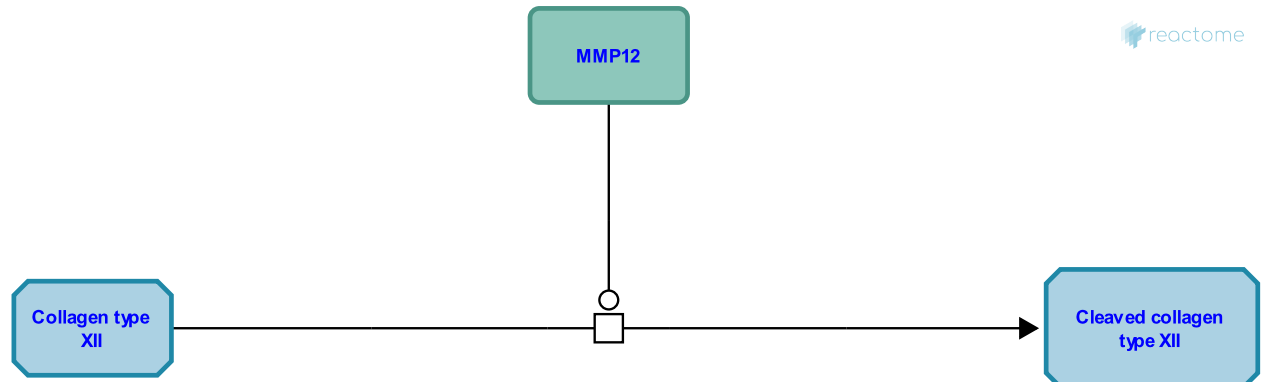
This document contains 1 reaction ([see Table of Contents](#))

Collagen type XII degradation by MMP12 [↗](#)

Stable identifier: R-HSA-2168046

Type: transition

Compartments: extracellular region



Collagen type XII is a member of the fibril-associated collagens with interrupted triple helices (FACIT) group, thought to be bound to the surface of interstitial collagen fibrils (Keene et al. 1991). It has only one alpha chain type, with two collagenous (Col1 and Col2) and three noncollagenous domains (NC1-NC3). Whereas the collagenous and the NC1 and NC2 regions are short, the NHE-terminal NC3 is a huge trimeric domain (Yamagata et al. 1991, Wälchli et al. 1993). Collagen XII may enhance the stability of connective tissues by bridging collagen fibrils (Nishiyama et al. 1994, Bader et al. 2009). It may be a stress response molecule, directly influenced by stretch and shear stress. Expression of COL12A1 is directly stimulated by mechanical forces (Flück et al. 2003, Jin et al. 2003, Arai et al. 2008). Expression is predominantly in bone, suggesting involvement of type XII collagen in the regulation of osteoblasts and cell interactions. Transgenic type XII collagen-null mice have skeletal abnormalities. They have decreased bone matrix deposition and delayed maturation. Compared with controls, Col12a knockout osteoblasts are disorganized, being less polarized with disrupted cell-cell interactions, decreased connexin43 expression and impaired gap junction function (Izu et al. 2011).

MMP12 can cleave collagen XII (Didangelos et al. 2011).

Literature references

Mandal, K., Xu, Q., Mayr, M., Jahangiri, M., Yin, X., Smith, A. et al. (2011). Extracellular matrix composition and remodeling in human abdominal aortic aneurysms: a proteomics approach. *Mol Cell Proteomics*, 10, M111.008128. [↗](#)

Editions

2011-07-12	Authored	Jupe, S.
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