Consortium

DisCo-I is a new Consortium with young researchers who will combat major chronic diseases with **fibrotic** component. It offers fresh talent **comprehensive and inter-sectoral** training with the aim to prove that the **failure** of extracellular matrix **degradation** is the driving force of fibrosis.

The consortium will focus on delivering currently **missing fundamental knowledge** on the molecular pathophysiology of **type I collagen (COL1) degradation.** DisCo-I will also develop novel and non-invasive **diagnostic** biomarkers for fibrosis and set the stage for novel anti-fibrotic **therapies**.

Beneficiaries

HELLENIC REPUBLIC
National and Kapodistrian University of Athens
EST. 1837

ACADEMY OF ATHENS

BRFAA
BIOMEDICAL RESEARCH FOUNDATION ACADEMY OF ATHENS

RD Néphrologie

UNIVERSITÉ TOULOUSE III PAUL SABATIER

Inserm
La science pour la santé
From science to health

Funded by the European Union

DisCoI

mosaiques diagnostics

NORDIC BIOSCIENCE

UNIKLINIK RWTH AACHEN

UNIVERSITY OF COPENHAGEN

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DisCoI

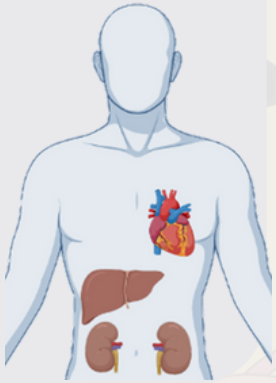
"Discovering Collagen I degradation process in chronic diseases with fibrotic component"

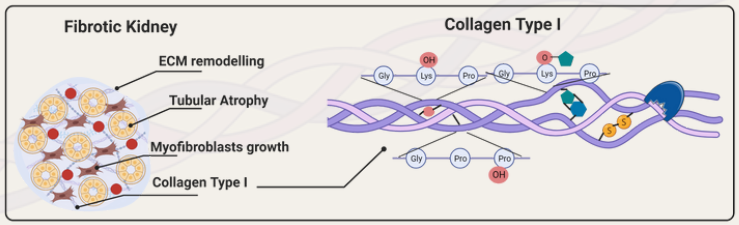
Funded by the European Union

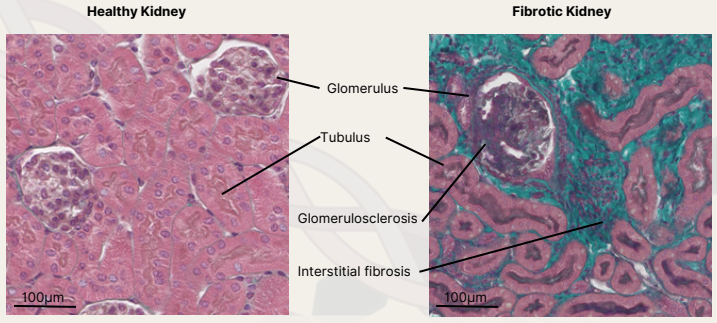
Fibrosis

Fibrosis refers to the scarring and stiffness of tissues caused by the accumulation of extracellular matrix (ECM) proteins, especially **type I collagen (COL1)**.

Fibrosis can harm patients by causing **organ dysfunction** and reducing their quality of life, as well as increasing morbidity and mortality rates.







COL1 is a fibrous protein and maintains the structural integrity of tissues through its unique triple helix structure. However, excessive deposition of COL1 can disrupt tissue architecture and function, which can lead to the development of fibrosis in the kidney. Analogue processes may occur in the liver and the heart.

Objectives

- Investigating the **molecular mechanism** associated with fibrosis and COL1 degradation and its clinical implications
- Identifying non-invasive biomarkers of COL1 degradation

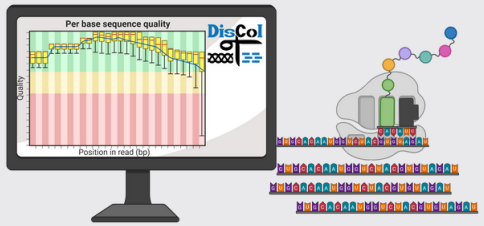
Early diagnosis of fibrosis is challenging and treatment options are limited

Molecular Characterization

COL1 turnover will be quantified in kidney, heart and liver fibrosis using **existing** and **newly-developed tests.**

We will identify **differentially** expressed mRNA, proteins and peptides using human urine, blood and tissue samples. With this information, **new diagnostic** methods will be developed and **validated**.

Bioinformatics



Our molecular characterisation of fibrosis on the **mRNA**, **protein** and **peptide** level will be used to generate a **computational model**, identifying also differentially regulated **proteases** to be **validated** *in vivo*. The products generated by proteases (peptides) can be used to develop **new diagnostic tests**.

Model Validation

We will investigate fibrosis-associated **COL1 degradation** *in vivo*. Through animal models, we can explore the impact of fibrosis on **organ function** and the correlation between **urinary peptides and ECM** as well as **protease activity**.