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University Hospital, Freiburg, Germany

STRETCH-ACTIVATED CHANNELS: COMMON GATES FOR TISSUE REMODELLING

15.OCTOBER 2024 – 11 AM – LECTURE HALL

Fibrosis is the common denominator of many diseases. In the heart in particular, excessive accumulation of extracellular matrix proteins represents a major threat, as it alters both electrical and mechanical properties of the tissue, generating a proarrhythmogenic substrate. A hallmark of fibrosis is the stiffening of the tissue which alters cellular microenvironment and cell functions fuelling disease progression. Molecular mechanisms underlying this vicious loop are not well understood.

Stretch-activated channels (SAC) transduce mechanical cues into biophysical or biochemical signals that can be interpreted by the cell. SAC are widely expressed in various organs and cell types, including muscle, endothelial and interstitial cells. We will present how the SAC Piezo1 and TREK-1 can alter cardiac fibroblast fate and functions, including substrate stiffness sensing and collagen production, to contribute to cardiac fibrosis. We will also highlight that the influence of SAC is not limited to the cells in which they are expressed but expands via homo- and hetero-cellular paracrine communication. Altogether, we hope these insights will help to re-think the importance of the mechanical environment and mechanosensors in driving cellular functions.

Invited by : Eva Faurobert

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Allée des Alpes, 38700 La Tronche (tram line B, stop : Grand Sablon)
The seminar is followed by discussions and exchanges with the speaker and a sandwich buffet is offered

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