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RESEARCH AREA

The major interest of our research group is soft tissue calcification, a process in which bone-like matrix deposits in the soft tissues outside of the skeletal system. Soft tissue calcification impairs the normal function of tissues and organs. The two most common types of soft tissue calcification are the vascular and the valve calcifications. Cardiovascular calcification is strongly associated with increased incidence of major cardiovascular events, such as myocardial infarction, stroke and cardiac death.

Cardiovascular calcification mostly affects the elderly, but some endemic diseases such as chronic kidney disease and type 2 diabetes largely accelerates calcification, and reduce life expectancy of these patients.

Vascular and valve calcification is driven by an osteoblastic phenotype switch of vascular smooth muscle cells and valve interstitial cells. In the past decades numerous calcification inducers and inhibitors have been identified, but many details of the molecular mechanism and the possible therapeutic interventions are still under investigation.

TECHNIQUES AVAILABLE IN THE LAB

Cell culture and treatment of human aorta smooth muscle cells and valve interstitial cells. Detection of calcification with staining and other technics.

Measurement of protein expression with western blot and ELISA technics.

RNA isolation and determination of mRNA expression with quantitative real time PCR.

In vivo investigation of calcification using a mouse model of chronic kidney disease. Laboratory animal care, experimental planning. Mice dissection, and detection of calcification with imaging technics, histology and immunohistochemistry.

SELECTED PUBLICATIONS

Balogh, E., Tóth, A., Méhes, G., Trencsényi, G., Paragh, G., **Jeney, V.** (2019) Hypoxia Triggers Osteochondrogenic Differentiation of Vascular Smooth Muscle Cells in an HIF-1 (Hypoxia-Inducible Factor 1)-Dependent and Reactive Oxygen Species-Dependent Manner. **Arterioscler Thromb Vasc Biol** **39**: 1088-1099.

Tóth, A., Balogh, E., **Jeney, V.** (2020) Regulation of Vascular Calcification by Reactive Oxygen Species. **Antioxidants (Basel)** **8;9**: 963.

Balogh, E., Chowdhury, A., Ababneh, H., Csiki, D.,M., Tóth, A., **Jeney, V.** (2021) Heme-Mediated Activation of the Nrf2/HO-1 Axis Attenuates Calcification of Valve Interstitial Cells. **Biomedicines** **15;9**: 427.

Tóth, A., Csiki, D.,M., Nagy, B., Jr, Balogh, E., Lente, G., Ababneh, H., Szöör, Á., **Jeney, V.** (2022) Daprodustat Accelerates High Phosphate-Induced Calcification Through the Activation of HIF-1 Signaling. **Front Pharmacol** **13**: 798053.

Csiki, D.M., Ababneh, H., Tóth, A., Lente, G., Szöör, Á., Tóth, A., Fillér, C., Juhász, T., Nagy, B., Jr, Balogh, E., **Jeney, V.** (2023) Hypoxia-inducible factor activation promotes osteogenic transition of valve interstitial cells and accelerates aortic valve calcification in a mice model of chronic kidney disease. **Front Cardiovasc Med** **10**: 1168339.