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RESEARCH AREA

My scientific interest is primarily focused on the molecular regulation of cardiomyocyte contractile function. In a unique cellular cardiac physiology laboratory in Hungary, I investigate the mechanical properties of individual cardiomyocytes obtained from human heart biopsies and experimental animal models. Our highly sensitive mechanical measurement system enables the analysis of contractile parameters in isolated cells, even from frozen or freshly obtained human tissue samples.

The extremely small size of the preparation (a single isolated cardiomyocyte) provides the opportunity to specifically modify the composition of contractile proteins – for example through post-translational mechanisms such as phosphorylation or degradation – and to model various pathophysiological states. In this way, cellular mechanical measurements can be complemented with detailed biochemical analyses, allowing the mapping of cellular and subcellular changes in myocardial contractility under different human and experimental disease conditions.

A key focus of my research is the titin protein, which, due to its enormous size and structural complexity, poses a major challenge worldwide. Using molecular and protein biochemical approaches, I study the role of titin in determining the mechanical properties of the myocardium and also aim to develop novel methodological approaches.

In collaboration with national and international partners, my goal is to gain deeper insights into the pathomechanisms of cardiovascular diseases and to identify novel therapeutic strategies, using both human tissue samples and in vivo animal models.

TECHNIQUES AVAILABLE IN THE LAB

The methods that can be acquired in the laboratory include the isolation of individual cardiomyocytes from human heart biopsies and experimental animal models, as well as the analysis of their contractile properties using a highly sensitive mechanical measurement system. It is possible to determine active and passive force as well as calcium sensitivity, along with the targeted enzymatic modification of contractile proteins (such as phosphorylation or degradation) and the induction of various model conditions. Cellular measurements can be complemented by biochemical analyses (e.g., Western blot), which allow the detailed investigation of post-translational modifications of proteins, with special emphasis on titin and other key sarcomeric proteins (troponin I, MyBP-C). The laboratory also provides the opportunity to acquire expertise in the proper handling and preparation of human and animal myocardial tissue samples, as well as in the design and execution of experiments involving pharmacological agents. The skills gained extend to data processing and evaluation, as well as to the practical application of a translational research perspective.

SELECTED PUBLICATIONS

Bódi, B., Vágó, R. R., Nagy, L., Ráduly, A. P., Gulyás, A., Kupecz, K., Azar, L., Márványkövi, F. M., Szűcs, G., Siska, A., Cserni, G., Földesi, I., Papp, Z., & Sárközy, M. (2025). Differential Myocardial Responses in Male and Female Rats with Uremic Cardiomyopathy. **Int J Mol Sci 26(5)**: 2259.

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Bódi, B., Kovács, Á., Gulyás, H., Mártha, L., Tóth, A., Mátyás, C., Barta, B., Oláh, A., Merkely, B., Radovits, T., Papp (2021). Z. Long-Term PDE-5A Inhibition Improves Myofilament Function in Left and Right Ventricular Cardiomyocytes through Partially Different Mechanisms in Diabetic Rat Hearts. **Antioxidants 10:** 1-13.

Bódi, B., Pilz, P., Mártha, L., Lang, M., Hamza, O., Fagyas, M., Szabó, P., Abraham, D., Tóth, A., Podesser, B., Kiss, A., Papp, Z. (2021). Alterations in ACE and ACE2 Activities and Cardiomyocyte Signaling Underlie Improved Myocardial Function in a Rat Model of Repeated Remote Ischemic Conditioning. **Int J Mol Sci 22:** 1-17.

Bódi, B., Pásztorné Tóth, E., Nagy, L., Tóth, A., Mártha, L., Kovács, Á., Balla, G., Kovács, T., Papp, Z. (2017). Titin isoforms are increasingly protected against oxidative modifications in developing rat cardiomyocytes. **Free Radic Biol Med 113:** 224-235.