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RESEARCH AREA

The cellular myocardial physiology laboratory, established in 2001 and unique in Hungary, provides the opportunity to study the mechanical properties of single myocardial cells obtained from human or experimental animal models of cardiovascular diseases. With the help of our sensitive mechanical measuring system, we can determine the contractile parameters of individual myocardial cells isolated from deep-frozen or even biopsy-derived myocardial tissues. Thus, the Ca2+-dependent isometric force generation of contractile proteins can be measured directly at the cellular level, and direct conclusions can be drawn about the kinetic characteristics of the actin-myosin cycle. In addition to cellular studies, the small size of the preparation (single isolated myocardial cell) allows the characterization of the composition of contractile proteins under control conditions and following enzymatic modifications (e.g., phosphorylation, degradation) or the induction of different model conditions. Thus, our experimental system is suitable for mapping cellular and subcellular changes during altered myocardial contractility in various human and experimental disease states.

TECHNIQUES AVAILABLE IN THE LAB

Preparation of laboratory solutions, myocardial cell isolation, performance of mechanical measurements on isolated myocardial cells, evaluation of measurement data, muscle biochemical methods.

SELECTED PUBLICATIONS

Bódi, B., Oláh, A., Mártha, L., Tóth, A., Radovits, T., Merkely, B., **Papp, Z.** (2021) Exercise-induced alterations of myocardial sarcomere dynamics are associated with hypophosphorylation of cardiac troponin I. R**eviews in Cardiovascular Medicine 22:** 1079-1085.

Bódi, B., Kovács, Á., Gulyás, H., Mártha, L., Tóth, A., Mátyás, C., Barta, B., Oláh, A., Merkely, B., Radovits, T., **Papp, Z.** (2021) 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.

Alvarado, G., Tóth, A., Csősz, É., Kalló, G., Dankó, K., Csernátony, Z., Smith, A., Gram, M., Akerström, B., Édes, I., Balla, G., **Papp, Z.**, Balla, J. (2020) Heme-Induced Oxidation of Cysteine Groups of Myofilament Proteins Leads to Contractile Dysfunction of Permeabilized Human Skeletal Muscle Fibres. **Int J Mol Sci 21:** 1-17.

Ruppert, M., Bódi, B., Korkmaz-Icöz, S., Loganathan, S., Jiang, W., Lehmann, L., Oláh, A., Barta, B., Sayour, A., Merkely, B., Karck, M., **Papp, Z.**, Szabó, G., Radovits, T. (2019) Myofilament Ca2+ sensitivity correlates with left ventricular contractility during the progression of pressure overload-induced left ventricular myocardial hypertrophy in rats. **J Mol Cell Cardio 129:** 208-218.