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RESEARCH AREA

Despite improving therapeutic options, cardiovascular diseases including myocardial infarction remain the leading cause of death. Research aiming to reduce heart damage is therefore of great importance. The heart has its own adaptive response to the cardiac injury. Various procedures can enhance this response and result in cardioprotection by reducing the injury. In addition to classical pre- and postconditioning techniques, our research group investigates pharmacological and non-pharmacological cardioprotective approaches and molecular mechanisms to alleviate cardiac injury. Certain metabolic diseases (e.g. hypercholesterolemia, diabetes) increase the risk of myocardial infarction, directly impair cardiac function and interfere with the heart's adaptive response to myocardial infarction. Our research focuses on the adverse effects of metabolic diseases as cardiovascular risk factors on cardiac function and adaptive response. We comprehensively analyse molecular changes with genomic and proteomic approaches, then we elucidate them in detail by focused studies. We also conduct experiments regarding pharmacological and non-pharmacological interventions to affect these undesirable molecular changes and to mitigate adverse cardiac effects.

TECHNIQUES AVAILABLE IN THE LAB

Induction, maintenance and treatment of cell cultures, simulated ischemia/reperfusion protocol and determination of cell death. Isolated heart preparation, heart perfusion according to Langendorff and Neely, induction of global and regional ischemia, ex vivo heart function measurement, determination of myocardial infarction with biochemical assays and tissue staining techniques. In vivo echocardiography, blood pressure measurement, cardiac catheterization. Induction and pharmacological treatment of diabetes and hypercholesterolemia in experimental rats and mice. General biochemical methods to determine metabolites, proteins and nucleic acids (colorimetry, Western blot, ELISA, PCR, immunocytochemistry, etc.).

SELECTED PUBLICATIONS

Pipicz, M., Kocsis, G.F., Sarvary-Arantes, L., Bencsik, P., Varga, Z.V., Ferdinandy, P., Csont, T. (2017) Low-dose endotoxin induces late preconditioning, increases peroxynitrite formation, and activates STAT3 in the rat heart. **Molecules 22:** 433.

Varga, Z.V., **Pipicz, M.**, Baán, J.A., Baranyai, T., Koncsos, G., Leszek, P., Kuśmierczyk, M., Sánchez-Cabo, F., García-Pavía, P., Brenner, G.J., Giricz, Z., Csont, T., Mendler, L., Lara-Pezzi, E., Pacher, P., Ferdinandy, P. (2017) Alternative splicing of nox4 in the failing human heart. **Front Physiol 8:** 935.

Pipicz, M., Demján, V., Sárközy, M., Csont, T. (2018) Effects of cardiovascular risk factors on cardiac STAT3. **Int J Mol Sci 19:** 3572.

Szabó, M.R., Gáspár, R., **Pipicz, M.**, Zsindely, N., Diószegi, P., Sárközy, M., Bodai, L., Csont, T. (2020) Hypercholesterolemia Interferes with Induction of miR-125b-1-3p in Preconditioned Hearts. **Int J Mol Sci 21:** 3744

Demján, V., Sója, A., Kiss, T., Fejes, A., Gausz, F. D., Szűcs, G., Siska, A., Földesi, I., Tengölics, R., Darula, Z., Csupor, D., **Pipicz, M.**, & Csont, T. (2021) Stellaria media tea protects against diabetes-induced cardiac dysfunction in rats without affecting glucose tolerance. **J Tradit Complement Med 12:** 250-259.