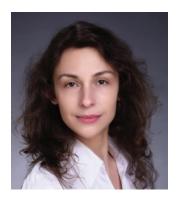
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RESEARCH AREA

Antibiotics and non-steroidal anti-inflammatory drugs (NSAIDs) have a potential influence on eukaryotic cells through affecting mitochondrial functions, the oxidative phosphorylation and mitochondrial reactive oxygen free radicals formation. Both drug classes are commonly used in acute and chronic inflammatory and non-inflammatory diseases of the gastrointestinal (GI) tract, where mitochondrial dysfunction can also occur. Our research group investigates the potential role of mitochondrial dysfunction in the inflamed and non-inflamed states of the digestive tract in experimental and clinical settings. We also aim at investigating the effect of drugs being commonly administered in these diseases. We perform a comprehensive analysis of in vitro doseresponse effect of antibiotics and NSAIDs using high resolution respirometry (HRR) in clinical and experimental tissue samples. In parallel, simultaneous manifestations of intramitochondrial and microcirculatory dysfunctions are monitored in a colitis model with particular interest in mucosal barrier functions and composition of the microbiome; these are examined in the presence and absence of various treatment combinations (antibiotics, antibiotics and NSAIDs, respectively).

TECHNIQUES AVAILABLE IN THE LAB

Processing of human samples and samples from in vivo animal models (mitochondria, epithelial cell and platelet isolation, tissue homogenates from punch biopsies). Basic laboratory techniques (measurements of enzyme activities, spectrophotometry, ELISA). Measurement of mitochondrial respiration, hydrogen-peroxyde production and membrane potential using high-resolution respirometry and its fluorescent moduls (Oxygraph2k). Microcirculatory measurements with orthogonal polarization spectral imaging and laser-Doppler.

SELECTED PUBLICATIONS

Benke, K., Jász, D.K., Szilágyi, Á.L., Baráth, B., Tuboly, E., Márton, A.R., Varga, P., Mohácsi, Á., Szabó, A., Széll, Z., Ruppert, M., Radovits, T., Szabó, G., Merkely, B., Hartmann, P., Boros M. (2021) Methane supplementation improves graft function in experimental heart transplantation. J Heart Lung Transplant 40: 183-192.

Jász, D.K., Szilágyi, Á.L., Tuboly, E., Baráth, B., Márton, A.R., Varga, P., Varga, G., Érces, D., Mohácsi, Á., Szabó, A., Bozó, R., Gömöri, K., Görbe, A., Boros, M., **Hartmann, P.** (2021) Reduction in hypoxia-reoxygenation-induced myocardial mitochondrial damage with exogenous methane. J Cell Mol Med 25: 5113-5123.

Horváth, T., Jász, D.K., Baráth, B., Poles, M.Z., Boros, M., Hartmann, P. (2021) Mitochondrial Consequences of Organ Preservation Techniques During Liver Transplantation. Int J Mol Sci 22: 2816.

Strifler, G., Tuboly, E., Görbe, A., Boros, M., Pécz, D., **Hartmann**, **P.** (2016) Inhaled Methane limits the mitochondrial electron transport chain dysfunction during experimental liver ischemia-reperfusion injury. **PLoS One 11:** e0146363.

Strifler, G., Tuboly, E., Szél, E., Kaszonyi, E., Cao, C., Kaszaki, J., Mészáros, A., Boros, M., **Hartmann, P.** (2016) Targeting mitochondrial dysfunction with L-alpha glycerylphosphorylcholine. **PLoS One 11:** e0166682.