MIROSLAVA KVANDOVA

Centre of Experimental Medicine
Slovak Academy of Sciences

Project number
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BIOGRAPHY

Miroslava Kvandová is a postdoctoral researcher focused mainly on basic research linked to molecular cardiology. She obtained her doctoral degree in Animal Physiology from Comenius University in Bratislava, Slovakia. She has previously worked at Johannes Gutenberg University Mainz where she worked with the working group of Prof. Thomas Münzel to study environmental stressors as novel cardiovascular risk factors. Currently, obtaining a SASPRO 2 fellowship allowed her to start her own research group in the Department of Experimental Hypertension at the Institute of Normal and Pathological Physiology, Slovak Academy of Sciences where she focuses her research on cardiometabolic diseases. The enormous number of people suffering from cardiometabolic diseases drives and motivates her to enlighten and fully understand through basic research the underlying occurrence and progression of these diseases as well as improve their treatment and prevention.

PROJECT SUMMARY

Significance of endothelial α1AMPK for vascular dysfunction and metabolic senescence in rat model of metabolic syndrome/ diabetes mellitus type II

Endothelial dysfunction is an early common feature of many cardiovascular diseases, caused by decreased nitric oxide (NO) production and/or increased NO inactivation due to oxidative stress. This influences a patient's risk of future cardiovascular events. The overall goal is to improve primary and secondary prevention for cardiovascular diseases. Therefore, analyzing key factors that prevent or positively influence endothelial dysfunction is essential. Working group of prof. Münzel/Daiber (current affiliation) has been focused on the role of AMP-dependent protein kinase (AMPK) for several years. This ubiquitously expressed enzyme is the central energy sensor of cells in the cardiovascular system. The protective effect of AMPK has been already demonstrated, especially its protective properties on endothelial function, oxidative stress, cell aging, and inflammation. In addition, AMPK regulates many metabolic pathways that are disturbed in the context of diabetes mellitus, such as the activation of glucose transport in skeletal muscle or the inhibition of gluconeogenesis in the liver. These properties suggest that AMPK may improve diabetic metabolic control. It has been shown for diabetes mellitus that vascular changes are prognostically decisive. Despite enormous research, the molecular changes that lead to endothelial dysfunction and predisposition to cardiovascular diseases due to α1AMPK-related dysregulation are insufficiently known. Therefore, the following questions will be addressed:

1. How do α1AMPK influence endothelial function, formation of reactive oxygen species, and vascular inflammation in the rat model of the metabolic syndrome/diabetes mellitus II type?
2. Exploring the role of α1AMPK expression in endothelial cell death and the development of metabolic senescence in hyperglycemia and diabetes?
3. Are metabolic syndrome/diabetes mellitus II type mediated disorders of the endothelial function associated with the gender-specific regulation of α1AMPK?

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PUBLICATIONS

1. Jansen, T., M. Kvandova, A. Daiber, P. Stamm, K. Frenis, E. Schulz, T. Munzel and S. Kroller-Schon (2020). "The AMP-Activated Protein Kinase Plays a Role in Antioxidant Defense and Regulation of Vascular Inflammation." Antioxidants (Basel) 9(6).
DOI: 10.3390/antiox9060525

2. Jansen, T., M. Kvandova, I. Schmal, S. Kalinovic, P. Stamm, M. Kuntic, M. Foretz, B. Viollet, A. Daiber, M. Oelze, J. F. Keaney, Jr., T. Munzel, E. Schulz and S. Kroller-Schon (2021). "Lack of Endothelial alpha1AMPK Reverses the Vascular Protective Effects of Exercise by Causing eNOS Uncoupling." Antioxidants (Basel) 10(12).
DOI: 10.3390/antiox10121974

3. Kant, S., K. V. Tran, M. Kvandova, A. D. Caliz, H. J. Yoo, H. Learnard, A. C. Dolan, S. M. Craige, J. D. Hall, J. M. Jimenez, C. St Hilaire, E. Schulz, S. Kroller-Schon and J. F. Keaney, Jr. (2022). "PGC1alpha Regulates the Endothelial Response to Fluid Shear Stress via Telomerase Reverse Transcriptase Control of Heme Oxygenase-1." Arterioscler Thromb Vasc Biol 42(1): 19-34.
DOI: 10.1161/ATVBAHA.121.317066.

4. Kroller-Schon, S., T. Jansen, T. L. P. Tran, M. Kvandova, S. Kalinovic, M. Oelze, J. F. Keaney, Jr., M. Foretz, B. Viollet, A. Daiber, S. Kossmann, J. Lagrange, K. Frenis, P. Wenzel, T. Munzel and E. Schulz (2019). "Endothelial alpha1AMPK modulates angiotensin II-mediated vascular inflammation and dysfunction." Basic Res Cardiol 114(2): 8.
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5. Kuntic, M., M. Oelze, S. Steven, S. Kroller-Schon, P. Stamm, S. Kalinovic, K. Frenis, K. Vujacic-Mirski, M. T. Bayo Jimenez, M. Kvandova, K. Filippou, A. Al Zuabi, V. Bruckl, O. Hahad, S. Daub, F. Varveri, T. Gori, R. Huesmann, T. Hoffmann, F. P. Schmidt, J. F. Keaney, A. Daiber and T. Munzel (2020). "Short-term e-cigarette vapour exposure causes vascular oxidative stress and dysfunction: evidence for a close connection to brain damage and a key role of the phagocytic NADPH oxidase (NOX-2)." Eur Heart J 41(26): 2472-2483.
DOI: 10.1093/eurheartj/ehz772

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