Endothelial and vascular function in mice overexpressing human soluble endoglin

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Objectives: A soluble form of endoglin (sEng) circulating in plasma and its increased levels has been detected in various pathological conditions related to cardiovascular system where endothelial dysfunction plays an important role. High concentration of sEng was also proposed to contribute to the development of endothelial dysfunction, however there is no evidence that this happens in atherosclerotic prone vessels. Therefore, in the present study we analyzed whether high sEng levels induce endothelial dysfunction in mouse aorta.

Methods: Four to 6-month-old transgenic mice with high expression of human sEng (Sol-Eng+) and age-matched transgenic littermates that do not develop high levels of human sEng (control animals) on chow diet were used. Analysis of vascular function in isolated aorta, Western Blot analysis and ELISA were performed.

Results: As expected, Sol-Eng+ transgenic mice showed higher levels of plasma concentrations of human sEng as well as increased blood arterial pressure, as compared to control animals. Functional analysis either in vivo or ex vivo in isolated aorta demonstrated that the endothelium-dependent vascular function was similar in Sol-Eng+ and control mice. In addition, Western blot analysis showed no differences between Sol-Eng+ and control mice in the protein expression levels of endoglin, eNOS and pro-inflammatory ICAM-1 and VCAM-1.

Conclusions: Our results demonstrate that high levels of sEng alone do not induce endothelial dysfunction in Sol-Eng+ mice. However, these data do not rule out the possibility that sEng might contribute to alteration of endothelial function in combination with other risk factors related to cardiovascular disorders.

Acknowledgments. This work was supported by grants from Czech Science foundation GACR number 15-24015S, the Grant Agency of Charles University in Prague (1284214/C and 1158413/C), Charles University in Prague (SVV/2014/260064), European Regional Development Fund under the Innovative Economy Program of the European Union (grant coordinated by JCET-UJ, No POIG.01.01.02-00-069/09), Ministerio de Economia y Competitividad of Spain (SAF2010-19222 and SAF2013-43421-R and SAF2010-1588), Junta de Castilla y Leon (GR100), Centro de Investigacio´n Biome´dica en Red de Enfermedades Raras (CIBERER) and Red de Investigacion Cooperativa en Enfermedades Renales (RD12/0021/0032; REDINREN). CIBERER and REDINREN are initiatives of the Instituto de Salud Carlos III (ISCIII) of Spain supported by FEDER funds. The Cardiovascular Phenotyping Unit of the University of Salamanca, including the telemetry equipment, was acquired with the support of the European Regional Development Funds (FEDER). Ministerio de Economia y Competitividad (BES-2008-005550). This work has been co-financed by the European Social Fund and the state budget of the Czech Republic (Project No. CZ.1.07/2.3.00/30.0061).

11th International HHT Scientific Conference
June 11-14, 2015
Captiva, Florida USA