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.

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