IJAE
Vol. 117, n. 2 (Supplement): 28, 2012
ITALIAN JOURNAL OF ANATOMY AND EMBRYOLOGY

Expression of endothelial nitric oxide synthase in blood vessels and silicic acid consumption

Barbara Buffoli, Elisa Borsani, Davide Merigo, Marco Cocchi, Ottavia Filippi, Mauro Labanca and Luigi F. Rodella

Human Anatomy Division, Department of Biomedical Sciences and Biotechnologies, University of Brescia, Brescia, Italy

NO produces by endothelial nitric oxide synthase (eNOS) represents one of the most representative vasoactive molecules able to regulate vascular tone; it is released by endothelial cells and it diffuses to adjacent vascular smooth muscle cells causing vasorelaxation. In addition, endothelium-derived NO is know to be involved in multiple ways to prevent the progression of age-related vascular diseases. Senescent endothelial cells are characterized by a decreased production of endothelium-derived NO due to a decrease of eNOS activity that could be attributable to a reduction in eNOS protein expression as well as in eNOS phosphorylation (Matsushita et al., 2001).

Previous studies showed that silicon, mainly as silicic acid, plays an important role as protective factor against the development of age-related vascular diseases, maintaining integrity, stability and elastic properties of arterial walls (Schwarz et al., 1977). So, the aim of this study was to evaluate the relationship between the expression of eNOS and silicic acid consumption in a mouse model of early physiological aging. We evaluated qualitatively and quantitatively by immunohistochemical method, the expression of eNOS in the vessel wall of aorta and renal vessels in relation with the administration of silicic acid in drinking water. The results showed that loss of eNOS expression was prevented by regular consumption of silicic acid rich water, supporting the potential protective role of silicon against age related-vascular disorders.

References

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Keywords: Blood vessels, eNOS, silicic acid.