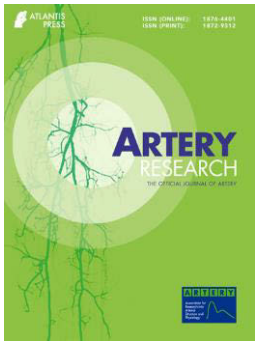

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ABSTRACT OF ARTERY CAREER PROGRESSION LECTURE 1

FUNCTIONAL GENOMICS AND ARTERIAL STIFFNESS

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Arterial stiffness, which has an independent predictive value for cardiovascular events, is influenced by genetic factors. The molecules underlying the process of arterial stiffening and, specifically, the identification of molecules that can contribute quantitatively to arterial stiffness are still largely unknown. The present report proposes an integrated view of the molecular determinants of arterial stiffness, based on a candidate gene approach. Indeed, studies on gene expression profile of human and animal aortas during various diseases (hypertension, chronic renal failure, impact of maternal diabetes…) allow us to understand the influence of abnormal, genetically determined, wall components on arterial stiffness.

During the whole life, large arteries are submitted to cardiovascular risk factors, inducing changes in structural and functional proteins of the arterial wall, then decreasing its elastic properties. Hypertension and chronic renal failure are considered models of accelerated arterial stiffness. Using a microarray approach, we have shown that it is possible to identify genes differentially expressed between stiff and distensible aortas. The proteins encoded by these genes mostly belong to the ''signaling'' and ''structure/motility'' categories and could play a role in arterial stiffening through either cell-matrix interactions or enhanced contractile signaling pathways.

Fetal environment influences the development of cardiovascular diseases in adulthood. To better understand the mechanisms involved, we studied a model of rats exposed *in utero* to maternal diabetes. These rats develop hypertension as early as 6 month of age. At the pre-hypertensive stage (3 months), we found abnormalities of gene expression in favour of vasoconstriction and increased arterial stiffness in the offspring of diabetic mothers. These results suggest that the vascular fetal programming was modified by the *in utero* exposure to maternal hyperglycemia. Modified gene expression could explain, at least in part, the arterial disorders observed later during adult life.

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