

Letter to the Editor

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Role of Hyperleptinemia in the Regulation of Blood Pressure and Cardiac Function

To the Editor:

We read with great interest the article by Dr Samuelsson et al¹ dealing with the relationships between hyperleptinemia and blood pressure or myocardial function. The results of their study demonstrated that exposure to hyperleptinemia in neonatal rats might lead to early onset of hypertension, which might be a result of the heightened sympathetic tone. Echocardiographic investigation also revealed altered left ventricular structure and systolic function in the leptin-treated neonatal rats. The authors indicated that neonatal hyperleptinemia might play an important role in the developmental origin of hypertension and cardiac dysfunction, suggesting that high leptin concentrations, secondary to maternal obesity would constitute a cardiovascular risk for the offspring.

Evidence indicates that acute administration of leptin increased plasma nitric oxide (NO)-metabolite concentration in a dose-dependent manner in the adult rats.^{2,3} It was also demonstrated that leptin-infusion increased blood pressure in rats under NO synthase inhibition and decreased blood pressure in rats with pharmacologically induced ganglionic blockade.² It is strongly suggested that leptin might have a balanced effect on blood pressure with a pressor response attributable to sympathetic activation and a depressor response attributable to NO release. In this issue, Nickola et al⁴ demonstrated that leptin attenuated cardiac contraction in rat ventricular myocytes, possibly through an increased NO production. We showed that relaxing effect of leptin on blood vessels was partially mediated by the NO-dependent mechanism.⁵ In this context, we speculate that the leptin-induced NO might participate actively in the homeostasis of not only blood pressure but also cardiac function and vascular tone. Therefore, we would like to know whether endothelial function might be altered in

the leptin-treated neonatal rats, and whether leptin-induced NO might contribute, at least in part, to the cardiac dysfunction in the models of Samuelsson et al.¹ It would be important to assess more precisely the links between leptin and NO production and their role in the early onset of hypertension and heart failure.

Disclosures

None.

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