Postnatal overfeeding causes early shifts in gene expression in the heart and long-term alterations in cardiometabolic and oxidative parameters

Submitted by Emmanuel Lemoine on Tue, 02/24/2015 - 15:49

Titre
Postnatal overfeeding causes early shifts in gene expression in the heart and long-term alterations in cardiometabolic and oxidative parameters

Type de publication
Article de revue

Auteur
Habbout, A. [1], Guenancia, C. [2], Lorin, J. [3], Rigal, E. [4], Fassot, Céline [5], Rochette, L. [6], Vergely, C. [7]

Editeur
Public Library of Science

Type
Article scientifique dans une revue à comité de lecture

Année
2013

Langue
Anglais

Date
2013

Numéro
2

Volume
8

Titre de la revue
PloS one

ISSN
1932-6203

Mots-clés
Animals [8], Blood Glucose [9], Body Composition [10], Body Weight [11], Disease Susceptibility [12], Female [13], Gene Expression [14], Gene Expression Profiling [15], Gene Expression Regulation [16], Heart/physiology/physiopathology [17], Mice [18], Myocardial Reperfusion Injury/genetics/metabolism [19], Myocardium/metabolism [20], Overnutrition [21], Oxidation-Reduction [22], Oxidative Stress [23], Reactive Nitrogen Species [24], Time Factors [25], Ventricular Remodeling [26]
BACKGROUND: Postnatal overfeeding (OF) in rodents induces a permanent moderate increase in body weight in adulthood. However, the repercussions of postnatal OF on cardiac gene expression, cardiac metabolism and nitro-oxidative stress are less well known. METHODOLOGY/PRINCIPAL FINDINGS: Immediately after birth, litters of C57BL/6 mice were either maintained at 10 (normal-fed group, NF), or reduced to 3 in order to induce OF. At weaning, mice of both groups received a standard diet. The cardiac gene expression profile was determined at weaning and cardiac metabolism and oxidative stress were assessed at 7 months. The cardiac expression of several genes, including members of the extracellular matrix and apelin pathway, was modified in juvenile OF mice. In adult mice, OF led to an increase in body weight (+30%) and to significant increases in plasma cholesterol, insulin and leptin levels. Myocardial oxidative stress, SOD and catalase activity and mRNA expression were increased in OF mice. In vivo, diastolic and systolic blood pressures were significantly higher and LV shortening and ejection fraction were decreased in OF mice. Ex vivo, after 30 min of ischemia, hearts isolated from OF mice showed lower functional recovery and larger infarct size (31% vs. 54%, p<0.05). Increases in collagen deposition and expression/activity of matrix-metalloproteinase-2 were observed in adult OF mouse hearts. Moreover, an increase in the expression of SOCS-3 and a decrease in STAT-3 phosphorylation were observed in ventricular tissues from OF mice. CONCLUSIONS/SIGNIFICANCE: Our study emphasizes that over-nutrition during the immediate postnatal period in mice leads to early changes in cardiac gene expression, which may permanently modify the heart’s structural organization and metabolism and could contribute to a greater susceptibility to myocardial ischemia-reperfusion injury.
Publié sur Okina (http://okina.univ-angers.fr)