

Modification of cardiac morphology was associated with impaired myocardial sensitivity to ischemia-reperfusion injury in a diet-induced metabolic syndrome model

Natacha Fourny, Carole Lan, Frank Kober, Monique Bernard, Martine Desrois

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Natacha Fourny, Carole Lan, Frank Kober, Monique Bernard, Martine Desrois. Modification of cardiac morphology was associated with impaired myocardial sensitivity to ischemia-reperfusion injury in a diet-induced metabolic syndrome model. 53rd Annual Meeting of the European Association for the Study of Diabetes (EASD), Sep 2017, Lisbon/PT, France. pp.S547–S547, 10.1007/s00125-017-4350-z. hal-01784727

HAL Id: hal-01784727 https://amu.hal.science/hal-01784727

Submitted on 7 May 2018

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Fourny N, Lan C, Kober F, Bernard M, Desrois M.

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In: Diabetologia. Vol. 60 Supplement 1. 53rd Annual Meeting of the European Association for the Study of Diabetes (EASD), Lisbon/PT, September 11-15.; 2017. pp. S547–S547. doi: 10.1007/s00125-017-4350-z.

Background and aims: Metabolic Syndrome (MetS) is defined by multiple risk factors that predict type 2 diabetes and cardiovascular complications, such as myocardial infarction, especially in women. Consequently the aim of this study was to investigate in vivo and ex vivo the effects of a high-fat-high-sucrose diet (HFHSD) on the development of metabolic syndrome (MetS), cardiac morphology and sensitivity to ischemia-reperfusion injury of female Wistar rat.

Materials and methods: Female Wistar rats, subjected to HFHSD (FHFD) or Normal Diet (FND) during 5 months, were explored in vivo every month with multimodal cardiovascular magnetic resonance (CMR). Cine-MRI (Magnetic Resonance Imaging) and arterial spin labeling (ASL-FAIR) techniques were used to determine cardiac morphology, function and perfusion. Triglyceride (TG) content in heart and liver 1 was also evaluated with H Magnetic Resonance Spectroscopy (MRS). 1Sub-cutaneous and visceral adipose tissues were measured with H MRI. Then, rats underwent an intraperitoneal glucose tolerance test (IPGTT) to determine glycemic status. Finally, isolated heart were perfused with a physiological buffer containing 0.4 mM palmitate for 24 minutes before switching to 1.2 mM palmitate during 32 minutes low-flow (0.5 mL/min/g wet wt) ischemia. Next, flow was restored with 0.4 mM palmitate buffer for 32 minutes. High-energy phosphates and intracellular pH were measured during the experimental course by P magnetic resonance spectroscopy with simultaneous measurement of contractile function. Coronary flow was measured before and after ischemia. At the end of experiments, hearts were freeze-clamped for biochemical assays.

Results: In FHFD vs. FND, CMR showed an increase of systolic wall thickness over time (p<0.05) and diastolic wall thickness at 3 and 5 months 1(p<0.01); H MRS showed that hepatic TG content was increased (p<0.01) at 5 month but myocardial TG content was not different. IPGTT showed a significant glucose intolerance (p<0.001) and plasma free fatty acids were increased (p<0.05) in FHFD vs. FND. At 5 months, weight was not different between groups but FHFD exhibited an abdominal obesity with increased visceral adipose tissue (p<0.05), % fat (p<0.05) and % visceral fat (p<0.05) compared with FND. Ex vivo myocardial function was impaired in FHFD vs. FND before (p<0.01) and after ischemia (p<0.05).

Conclusion: HFHSD-induced MetS was characterized by glucose intolerance, abdominal obesity, hepatic fat deposit which were associated with modification

of cardiac morphology and higher myocardial sensitivity to ischemia-reperfusion injury. These results may be related to higher risk of cardiovascular complications among type 2 diabetic obese women. Supported by: Aix-Marseille Univ, CNRS, France Life Imaging Disclosure: N. Fourny: None.