

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

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**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 was also evaluated with <sup>1</sup>H Magnetic Resonance Spectroscopy (MRS). <sup>1</sup>H Sub-cutaneous and visceral adipose tissues were measured with <sup>1</sup>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 <sup>31</sup>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 ( $p < 0.01$ ); <sup>1</sup>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.