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▶ **To cite this version:**

N. Fourny, C. Lan, F. Kober, M. Bernard, M. Desrois. 128 - Intolerance to glucose and abdominal obesity in a diet-induced metabolic syndrome model were associated with modification of cardiac morphology and impaired myocardial function. Printemps de la Cardiologie : Recherche Fondamentale et Clinique, Apr 2017, Nantes, France. pp.189, 10.1016/S1878-6480(17)30469-X . hal-01784721

HAL Id: hal-01784721
https://hal-amu.archives-ouvertes.fr/hal-01784721

Submitted on 16 May 2018

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Final Version

128 Intolerance to glucose and abdominal obesity in a diet-induced meta- bolic syndrome model were associated with modification of cardiac morphology and impaired myocardial function.

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Introduction

Metabolic Syndrome (MetS) is defined by multiple risk fac- tors that predict type 2 diabetes and cardiovascular complications, such as myocardial infarction, especially in women. Consequently the aim of this pre- liminary 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 function of female Wistar rat.

Material and methods

Female Wistar rats, subjected to HFHSD (FHFD) or Normal Diet (FND) during 5 months, were explored every month with multi- modal cardiovascular magnetic resonance (CMR) to determine in vivo cardiac function, morphology and triglyceride (TG) content. Liver TG content was # evaluated with 1H Magnetic Resonance Spectroscopy (MRS). Then, rats underwent an IPGTT to determine glycemic status, and ex vivo experiments on isolated perfused heart were performed to study cardiac function and energy metabolism with 31P MRS under baseline conditions.

Results

In FHFD vs. FND, CMR showed an increase of systolic wall thick- ness over time ($p<0.05$) and diastolic wall thickness at 3 and 5 months ($p<0.01$); 1H MRS showed that hepatic TG content was increased ($p<0.01$) 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. Under baseline conditions, ex vivo myocardial function was impaired in FHFD vs. FND ($p<0.01$).

Conclusion

HFHSD-induced MetS was characterized by glucose into- lerance, abdominal obesity, hepatic fat deposit which were associated with modification of cardiac morphology and impaired myocardial function. These results may be related to higher risk of cardiovascular complications among type 2 diabetic obese female.

The author hereby declares no conflict of interest