

“Modulation of protein content of LC3-II in offspring of animals submitted to the high fat diet during intrauterine life and lactation”.

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Abstract

Maternal obesity induced by high fat diet (HFD) during pregnancy and lactation can negatively interfere in the development of offspring. In adulthood, obesity and lipid overload represent factors that compromise autophagy, a process of lysosomal degradation that is essential for the maintenance of cellular homeostasis. Based in this context, the aim of this study was to evaluate the protein content of LC3-II, a principal marker of cellular autophagy, in the hypothalamus and liver of offspring at birth, after weaning and in adulthood. At birth, the offspring showed a decrease of LC3-II protein content only in the liver of offspring from obese dams (HFD-O) when compared to offspring control (SC-O). At weaning LC3-II decreased in both hypothalamic and hepatic tissue. However, during adulthood there were no differences between the groups. Overall, we believe that this study demonstrates that offspring's LC3-II, as well as in adulthood, is also highly associated with exposure to lipids and obesity.

Key words: Autophagy, liver and hypothalamus.

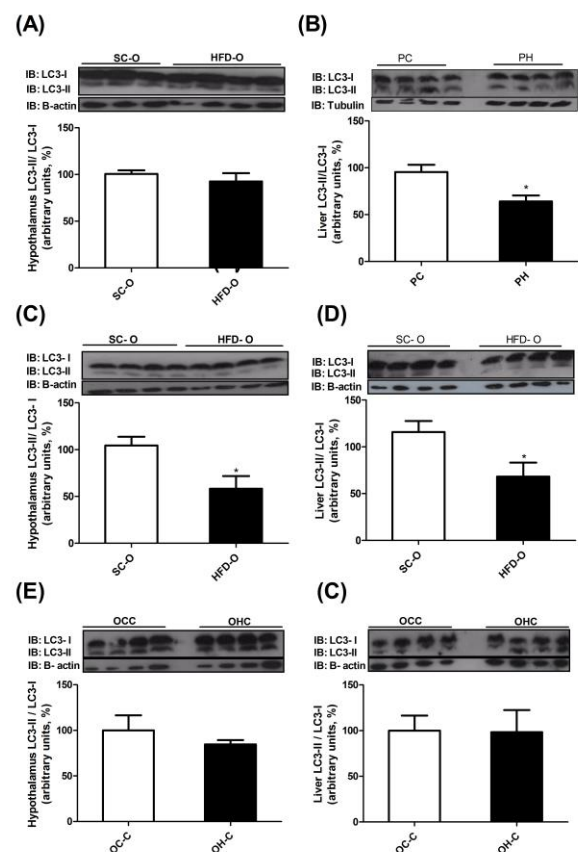
Introduction

Autophagy is a catabolic mechanism in which cellular organelles, proteins, and invading microorganisms are degraded by lysosomes; this maintains a balance between the synthesis, degradation, and subsequent recycling of cellular components, providing homeostasis and cell survival¹.

The liver and the hypothalamus are two important tissues that autophagy plays a fundamental role, such as the action in hepatic gluconeogenesis and in the control of hypothalamic food intake and energy expenditure¹. Importantly, LC3-II is a protein involved in the elongation of autophagosome membrane, essential to autophagy structure. Based in this context, the aim of the study was to evaluate the levels of LC3-II in the liver and the hypothalamus in the offspring of dams treated with HFD 45% during pregnancy and lactation, at birth (d0), at weaning (d18) and in adulthood (d82).

Results and Discussion

Results from this study demonstrate that offspring from obese dams have impairment of liver autophagy protein at birth and after lactation in both tissues (hypothalamus and liver). Surprisingly, the offspring from obese dams receiving control diet after weaning until 82 days have no impairment of autophagy's protein in both tissues analyzed (**Figure 01**). Thus, indirect exposure to lipids in the offspring from obese dams (HFD-O) is likely to be an essential condition for enhancing modulation of LC3-II of our experimental model



Conclusions

Maternal DIO downregulates an autophagy marker

Acknowledgement

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