Introduction

Puberty is a time of hormonal changes that are associated with insulin resistance. Although insulin sensitivity is restored at the end of puberty in healthy youth, it does not resolve in obese adolescents leading to an increase of cardio metabolic diseases such as type 2 diabetes.

In response to an increase in insulin demand, as during pregnancy or obesity-induced insulin resistance, β-cells increase their functional mass to maintain glucose homeostasis. However, the mechanism of pancreatic β-cell compensation in the face of pubertal insulin resistance has not been established. Hormonal changes during puberty could be linked to this β-cell adaptation.

Objective

To characterize pancreatic β-cell adaptation to pubertal insulin resistance.

Methods

- Wistar rats were subjected to metabolic and hormonal test every 5 days from weaning to adulthood.
- β-cell proliferation was assessed by immunostaining of pancreatic cryosections for Ki67 and insulin.

Results

Analysis of metabolic parameters in Wistar rats from weaning to adulthood. (A) Fasted blood glucose and insulin levels. Males n=4 (blue), females n=4 (red). (B) Glucose tolerance test (1g/kg glucose IP). (C) AUC for glycemia and insulinemia from IPGTT at 27, 42 and 57 days of life. Males n=4, females n=4. *p<0.05, **p<0.01.

Conclusions and perspective

- Insulin resistance and β-cell proliferation increase during puberty in rats. The parallel increase in IGF1 levels and β-cell proliferation point to a possible role of growth hormone in compensatory β-cell expansion.
- In future studies we will assess whether β-cell adaptation is compromised in a pathological model of metabolic stress during puberty.

Disclosure statement
None of the authors have a conflict of interest to declare.