Differential Expression of mTOR Related Molecules in the Placenta of Patients with Gestational Diabetes Mellitus, Intrauterine Growth Restriction, or Preeclampsia

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Differential Expression of mTOR Related Molecules in the Placenta of Patients with Gestational Diabetes Mellitus, Intrauterine Growth Restriction, or Preeclampsia

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Introduction

The mechanistic target of rapamycin (mTOR) pathway is involved in placental growth and function during pregnancy by responding to nutrient availability and growth factors regulating protein transcription and cell growth. mTOR pathway disruptions are associated with the development of obstetric complications, which may result in adverse health outcomes for the mother and/or fetus. The purpose of this study was to identify the differing placental expression of various mTOR-associated proteins during normal gestation (Control), gestational diabetes mellitus (GDM), intrauterine growth restriction (IUGR), and preeclampsia (PE).

Methods

Placenta Samples
Placental biopsies and slides from normal pregnancy (Control), Gestational Diabetes (GDM), Intrauterine Growth Restriction (IUGR), and Preeclampsia (PE) were purchased from the Research Center for Women’s and Infant’s Health BiBank, Ontario, Canada.

Immunofluorescence (IF)
IF was performed on paraffin embedded placental sections. Slides were incubated with rabbit polyclonal primary antibody against cytokeratin 7 (trophoblast localization), phospho (p)AKT, (p)ERK, (p)mTOR, (p)p70, or (p)4EBP1. Donkey anti-rabbit Texas Red (TR) or FITC secondary antibodies were used. DAPI specified nuclear staining and resulting immunofluorescence was detected on a BXS1 microscope.

mTOR PCR Array
A real-time PCR array was completed to assess differing placental expression of additional mTOR-associated genes during these conditions.

Statistical Analysis
Data are shown as Mean ± SE and P<0.05 was considered significant.

Results

Figure 1. Placental active AKT. Increased (p)AKT was detected during GDM but decreased in the IUGR placenta.

Figure 2. Placental ERK activation. Increased (p)ERK expression was only detected in the IUGR placenta.

Figure 3. Placental phospho (p) mTOR. Increased (p)mTOR expression was detected during GDM, but decreased in the IUGR and PE placentas.

Figure 4. Placental phospho (p)p70. Increased (p)p70 expression was detected during IUGR, but decreased in the GDM and PE placentas.

Figure 5. Placental phospho (p) 4EBP1. Increased (p)4EBP1 expression was detected during GDM, IUGR and PE placenta as compared to controls.

Results Summary

Compared to control samples we observed:
1) increased (p)AKT during GDM while decreased during IUGR.
2) increased (p)ERK during IUGR.
3) increased (p)mTOR during GDM, and decreased (p)mTOR during IUGR and PE.
4) increased (p)p70 during IUGR and decreased (p)p70 during GDM and PE.
5) increased (p)4EBP1 during GDM, IUGR, and PE.
6) differential placental expression of mTOR pathway associated genes.

Conclusions

We conclude that diverse regulation of the mTOR pathway is uniquely involved in the development of the obstetric complications studied. These results may provide insights into the physiological relevance of these pathways, and if so, their modification during gestation may help alleviate these diseases.