

## Abstract

Anna Girsén, University of Oulu, Dept of Obstetrics and Gynecology  
P.O. Box 5000, FIN-90014 University of Oulu, Finland  
[anna.girsén@oulu.fi](mailto:anna.girsén@oulu.fi)

**Thesis: Preeclampsia and maternal type-1 diabetes: new insights into maternal and fetal pathophysiology**

Public defence: May 15th 2009

Abnormal placentation is associated with preeclampsia and placental insufficiency, both of which increase the risk for fetal growth restriction. So far the early recognition of the risk population for preeclampsia has been problematic. The first hypothesis of this study was that in preeclampsia, the maternal serum proteomic profile is different from that in uncomplicated pregnancies, and this difference is detectable already in early pregnancy. The findings of this study demonstrate that in clinical preeclampsia the maternal serum proteomic profile is different from that in uncomplicated pregnancies with increased levels of placental proteins and antiangiogenic factors in pregnancies with clinical preeclampsia. Furthermore, the early pregnancy maternal serum proteomic profile in women who later develop preeclampsia revealed a distinct and different pattern compared with the profile in clinical preeclampsia. In early pregnancy, the differentially expressed proteins belong to placental proteins, vascular and/or transport proteins and matrix and/or acute phase proteins, while angiogenic and antiangiogenic proteins were not significantly expressed in early pregnancy.

Preeclampsia, placental insufficiency, fetal growth restriction and type-1 diabetes may have an impact on fetal cardiovascular hemodynamics. The second hypothesis in this thesis was that in placental insufficiency, abnormalities in fetal cardiovascular status correlate with biochemical markers of cardiac dysfunction and chronic hypoxia. In placental insufficiency, increases in fetal N-terminal pro-atrial (NT-proANP) and pro-B-type natriuretic peptide (NT-proBNP) and in fetal erythropoietin concentrations were related to increased pulsatility in the fetal umbilical artery and descending aorta. In addition, these fetuses demonstrated increased pulsatility in their systemic venous blood velocity waveforms. Thus, in placental insufficiency, biochemical markers of cardiac dysfunction and chronic hypoxia are associated with signs of increased fetal cardiac afterload and systemic venous pressure. Increased NT-proANP and NT-proBNP levels were also detected in fetuses of type-1 diabetic mothers with normal umbilical artery velocimetry. In these pregnancies, NT-proANP and NT-proBNP levels were related to poor maternal glycemic control during early pregnancy.

Electronic version of the thesis:

<http://herkules.oulu.fi/isbn9789514291104/isbn9789514291104.pdf>