pregnancy. Degree of lag depends on the severity of IUGR - 31% of normal in FGR III degree. When PI there is a decrease in vascular resistance in the thyroid arteries - by 22.2% from the norm in FGR III degree. In FGR II-III degree decreases the concentration of thyroid hormones and increased TSH: T3 total was reduced by 22%, total T4 - 66.7%, TSH increased - by 42%. In FGR II-III degree a marked lag growth adrenal compared with uncomplicated pregnancy - 25.3% width, 26% height and 15.5% in thickness. In FGR I degree the growth of vascular resistance occurs in the adrenal arteries - 16% of normal. In FGR III degree - reducing vascular resistance - 27% of normal.
Conclusions: In FGR II-III degree decreases the concentration of cortisol in the blood of a newborn - below 105 nmol/L, indicating that violations of adaptation followed by the appearance of severe complications in the early neonatal and postnatal periods.

Keywords: Placental insufficiency, intrauterine growth restriction, metabolic programming

Presenter: I.V. Ignatko

ID 788

PLASMINOGEN ACTIVATOR INHIBITOR - 1 IN MATERNAL PLASMA OF OVERWEIGHT AND NONOVERWEIGHT PREGNANT WOMEN AND ITS RELATIONSHIP TO LABOR COMPLICATIONS

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Brief Introduction: Overweight is a rising problem worldwide. It increases the risk of various pathologies, including those related to pregnancy and childbirth. Plasminogen activator inhibitor-1 (PAI-1) plays an important role in a pathogenesis of variety of overweight-related diseases.

The aim of this study is to determine PAI-1 concentration in maternal plasma of pregnant women with and without overweight, follow the changes during the first and second trimesters, and to find out whether there is a correlation between PAI-1 and childbirth complications.
Materials & Methods: It is a prospective study involving pregnant women at their first antenatal visit. According to body mass index (BMI), the patients were divided into groups with ($n = 20$) and without overweight ($n = 41$). At 9th-12th, 15th-18th and 24th-28th weeks of pregnancy plasma samples were collected and PAI-1 concentrations was established using Luminex-200 system. From the medical records clinical data on parturition were collected. Statistical analysis was carried out by programms R and LibreOffice Calc.
Clinical Cases or Summary Results: During pregnancy PAI-1 plasma concentration is gradually rising, and it is higher in women with excessive weight. Statistically significant differences were found between PAI-1 levels in patients with normal BMI and with or without fetal macrosomia - maternal cytokine concentration was higher when fetal macrosomia was found, and this relationship persists in both first and second trimester.
Conclusions: PAI-1 concentrations in the plasma of pregnant women starts to increase from the first trimester and is significantly higher in women with excessive weight. In addition, PAI-1 concentrations statistically significantly correlate with risk of fetal macrosomia in normal BMI patients.

Keywords: PAI-1, overweight, pregnacy, labor complications

Presenter: Karlina Elksne

ID 588

PERINATAL EPIGENETICS: LOW BIRTH WEIGHT AND ADULT ONSET SYSTEMIC SCLEROSIS

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Brief Introduction: Many lines of research require further development for the purpose of better understanding the mechanisms of fetal programming, clarifying the role of maternal nutrition, and more importantly, determining whether impaired fetal growth could have potentially adverse effects on chronic disease at an adult age. Low birth-weight, due to preterm birth or foetal growth restriction, is a marker of a lack of nutrients during ''critical'' periods of rapid cell division, that could programme the immune system. According to this new epigenetic developmental model, the aim of our study was to evaluate whether a significant correlation exists between low birth weight, other perinatal variables and the subsequent development of systemic sclerosis (SSc), a chronic systemic autoimmune disease.
Materials & Methods: During the period from June 2012 to November 2013, a multicenter case-control study was conducted with 332 consecutive prevalent cases of SSc and 243 controls, asking them to fill out a questionnaire regarding their demographic and perinatal information: gender, age, birth-weight, gestational age at birth, their mother's age at birth, breastfeeding, mother's smoke habit. A sub-sample of 40 cases and 40 controls was re-questioned later to verify the accuracy of the first response. Birth-weight was always treated as a dichotomous variable (<2500 g vs. ≥2500 g), low birth-weight was defined as weight less than 2500 g and small for gestational age was defined as a weight <2500 g in patients born at term. The association between both low birth-weight and small for gestational age with systemic sclerosis was expressed with the crude (univariate analysis) and adjusted (multivariate analysis) odds ratio.
Clinical Cases or Summary Results: Low birth weight infants (<2500 gr) had an increased risk of SSc when compared with higher birth weight infants (≥2500) (OR 2.59, 95% CI 1.39 to 5.05). When the analysis was limited to female subjects only, this value increased to an OR of 2.90. A similar result was found when we considered the exposure to small for gestational age (SGA) status (OR 2.60, 95% CI 1.34 to 5.32, that increases to 2.73, 95% CI 1.38 to 5.73 in female subjects).

In the multivariate analysis, the odds of disease in the low birth-weight female group was approximately 4 fold higher than in the normal birth-weight group.
Conclusions: Our study documents that the risk of developing SSc is higher when the patient was a LBW newborn. The precise cellular and molecular mechanisms which could explain the correlation between SSc and weight at birth, remain poorly understood. Environmental agents including, maternal diet or illness, endocrine disruptors or exposure to toxic levels of chemical agents, could act epigenetic mechanisms and cause modifications to the fetal immune system with the result of altering the phenotype in the off spring.