Results: 180 multiple gestations were analyzed, and 362 newborns were studied. Among them 9% were monochorionic monoamniotic, 37% were monochorionic diamniotic, and 54% were dichorionic diamniotic. Multiple pregnancies presented more risk of prematurity, lower birth weight and higher mortality than single pregnancies. The relative risk of developing a gestational hypertensive disease was 1.47 (1.01-2.19). Although the monochorionic monoamniotic group reaches the highest percentage of term birth, it has an increased risk of developing a gestational hypertensive disease, fetal growth restriction, and mortality. Regarding uric acid levels, it was observed that the behavior of the uricemia ratio was similar in normotensive women with simple and multiple pregnancies (1.20±0.29 vs 1.30±0.07) and in simple and multiple pregnancies associated to a hypertensive disorder such as preeclampsia (1.57±0.13 vs 1.73±0.13) or gestational hypertension (1.42±0.24 vs 1.3±0.09).
Conclusion: Our results showed no difference in the severity of the presentation of the hypertensive disorder between multiple and single pregnancies, revealing a similar maternal systemic dysfunction between both groups.

P2.117.
COMPARISON OF *IN VITRO* PRE-ECLAMPSIA (PE) CELL MODELS WITH PE PLACENTAL TISSUE

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Objectives: We induced hypoxia and oxidative stress (OS) in cytotrophoblasts (CTB) from control placentae to mimic the characteristics of preeclampsia (PE). For validation we compared the gene and protein expression of established PE biomarkers between the cell models and PE placentae.
Methods: CTB isolated from healthy term placentae (n=2) were cultured either under normoxic (5% pCO_2;21% pO_2;24h), hypoxic (5% pCO_2;1.5% pO_2;24h) or OS (consecutive 6h-intervals of normoxia and hypoxia until 24h) conditions. mRNA abundances of PE biomarkers (Table1) were analysed by qRT-PCR in the cell models and PE placentae (n=12). Additionally, the secretion of sFlt1 and PlGF was determined by ELISA in cell culture supernatants. Statistical analysis was performed using unpaired t-test.
Results: The cell models recapitulated important pathophysiological changes occurring in PE (Table1) including the upregulation of sFlt1 which was also observed in PE placentae. Interestingly, in the cell models the sFlt1/PlGF ratio was above the clinically applied cutoff value of 38 for hypoxic (77.3±35.2) and OS (124±88.3), but <38 for normoxic (19.4±16) conditions.
nm-not measured
Conclusion: The established cell models are mimicking important characteristics of PE. The sFlt1/PlGF ratio obtained in cell models is in agreement with reports proposing sFlt1/PlGF as predictive diagnostic marker for PE. Thus, hypoxia and OS induced in primary CTB can serve as suitable models to study the detailed mechanisms of PE.

P2.118.
TISSUE FACTOR AND THROMBOMODULIN: DETERMINATION OF MRNA LEVELS IN EXOSOMES DERIVED FROM HUMAN PLACENTAL EXPLANT CULTURES.

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Objectives: To compare the expression of tissue factor and thrombomodulin mRNA content in exosomes isolated from placental explant from normal and complicated pregnancies with preeclampsia.
Methods: We used twenty-one placental explant cultures of pregnant women without complications with healthy newborns (n=7), pregnant women diagnosed with early onset preeclampsia (n=7) and late onset preeclampsia (n=7). The exosome isolation was performed by ultracentrifugation. Quantification of exosomes was performed by ELISA (ExoQuant™ Overall Exosome Capture and Quantification Assay Kit). The exosome RNA extraction was done with the Total Exosome RNA and Protein Isolation Kit. Measurement of mRNA expression of tissue factor (TF) and thrombomodulin (TM) was performed by real-time PCR
Results: We found that the pregnant women with late onset PE have more concentration of exosomes isolated from placental explants compare to pregnant women with early onset PE and controls (P=0.0115). We found TF mRNA within exosomes in 57% of samples in control group, 43% in early PE and 86% in late PE groups. We found TM mRNA within exosomes in 43% of samples from control and early onset PE groups, and 57% of samples from late onset PE group, without statistically significant differences. There were no statistically significant differences in the amount of TM mRNA of exosomes between the groups. There were statistically significant differences in mRNA quantity of TF contained in isolated exosomes between early and late onset PE groups (P=0.0476), however there were no differences when compared to control group.
Conclusion: This is the first time that the presence of TF and TM mRNA contained in isolated exosomes from human placental explant cultures has been described. The increase of TF mRNA in exosomes from pregnant women with late onset PE may be involved in pathophysiology of the preeclampsia in this presentation of the disease.

P2.119.
PREECLAMPSIA AND OX-LDL MODIFY THE EXPRESSION OF AUTOPHAGY MARKERS IN PLACENTA AND FIRST TRIMESTER TROPHOBLAST CELL LINE IMPAIRING TROPHOBLAST INVASION AND MIGRATION.

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Placentation requires that the trophoblastic cells invade the maternal arteries and differentiate towards an endothelial phenotype, which is altered in preeclampsia (PE).
PE associates with increased concentration oxidized LDL (ox-LDL) in the maternal circulation. Ox-LDL leads to impaired trophoblast invasion in vitro and changes in basal autophagy in different cellular models (higher or lower depending on the model considered). Even if studies indicate autophagy is dysregulated in PE, if and how ox-LDL affect this cellular process is unknown.
Objetives: To determine autophagy levels, by using different markers, in trophoblast and endothelial cells from normal and PE placenta. To determine the effect of ox-LDL in the process of autophagy and in the capacity of invasion and migration of trophoblast cells.