

follow up: Systolic blood pressure (equal to or more than 120) and BMI (equal or more than 25) in early pregnancy as well as family history of CVD. History of PE was not significantly related.

**Conclusion:** Blood pressure in early pregnancy may indicate impaired cardiovascular adaptation and be an indication of later hypertension.

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### 233. Risk of cardiovascular mortality in women with a history of spontaneous preterm birth: A nationwide cohort study

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**Introduction:** Data suggest an association between a history of spontaneous preterm birth (sPTB) and cardiovascular disease (CVD) in later life. Therefore, we hypothesized that women with a history of sPTB have a higher cardiovascular mortality (CVM) risk.

**Methods:** Women who gave birth between 1995–2015 (registered in the National Birth Registry) were analyzed for death due to CVD through linkage with the National Death Registry. After excluding women with hypertensive pregnancy disorders and/or intra-uterine growth restriction, CVM for women with sPTB was analyzed prospectively in two different cohorts: including all births per woman and including only the first birth of a woman. Women with a sPTB were compared to women with no preterm birth. Cox-regression models with survival curves were executed.

**Results:** Of 1,476,048 parous women 6.9% had a history of sPTB. sPTB was associated with a 1.65-fold higher CVM risk (95% CI 1.34–2.04). Recurrent sPTB (HR 2.57; 95% CI 1.45–4.56) and sPTB under the 32 weeks gestational age (HR 2.59; 95% CI 1.64–4.10) were significantly associated with the highest CVM risk.

In the 1,166,476 nulliparous women, 6.4% had a sPTB. sPTB was associated with a 1.38-fold higher risk for CVM (95% CI 1.02–1.87). In this cohort the highest CVM risk was also found in women with a sPTB < 32 weeks gestational age (HR 3.19; 95% CI 1.84–5.56).

**Conclusion:** These data suggest that sPTB is associated with CVM. The CVM risk is highest in women with recurrent sPTB and sPTB < 32 weeks gestation.

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### 235. Evaluation of the fullpiers model and PLGF as predictors of adverse outcomes in women with hypertensive disorders of pregnancy

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**Introduction:** Singling out high-risk patients from the diverse hypertensive disorders of pregnancy (HDP), and not only preeclampsia, is a challenge for clinicians.

**Objectives:** The aim of the study is to evaluate the performance of the fullPIERS model and the placental growth factor (PLGF) to predict adverse outcomes in women with HDP.

**Methods:** A prospective cohort study carried out at a teaching hospital in Brazil enrolling pregnant women admitted with a systolic blood pressure  $\geq$  140 mmHg and/or a diastolic blood pressure  $\geq$  90 mmHg from the 20th week of gestation. First 48 h of admission worst clinical and laboratory data were recorded and adverse maternal outcomes were evaluated up to 14 days. Admission maternal plasma PLGF concentrations were measured.

**Results:** 351 women were included in the fullPIERS model analysis, 20 (5%) developed one of the combined maternal adverse outcomes. The fullPIERS model had poor outcome discrimination within 48 h [AUC 0.639 (95% CI 0.458–0.819)] and 14 days [AUC 0.637 (95% CI 0.491–0.783)]. PLGF analysis included 392 women. PLGF < 5th percentile predicted maternal adverse outcomes within 48 h in women with gestation < 35 weeks with negative predictive value (NPV) of 0.98 (0.9–0.99) and AUC ROC of 0.672 (CI 95% 0.5–0.9). PLGF had good performance to predict delivery within 14 days in women presenting before 35 weeks, AUC ROC 0.720 (0.64–0.80). PLGF < 5th percentile predicted small for gestational age (SGA) newborn with NPV 0.87 (0.75–0.94) and AUC ROC 0.698 (0.60–0.79), in women with gestation < 35 weeks.

**Conclusion:** The fullPIERS model and PLGF were limited predictors of maternal adverse outcomes in pregnant women with HDP, including preeclampsia, in our sample. PLGF appears to be an additional tool to predict delivery within 14 days and SGA newborn in women before 35 weeks gestation.

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### 240. Decidual inflammation in normal and preeclamptic pregnancies

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**Introduction:** Preeclampsia is characterized by reduced trophoblast invasion in the uterine wall decidua, harmful placental inflammation, and elevated systemic inflammation and sFlt-1 levels. Danger sensors like toll-like receptor (TLR)2, TLR4 and the nod-like receptor protein (NLRP)3 inflammasome initiate inflammation. These sensors have been associated with placental inflammation in preeclampsia, and maternal cholesterol and uric acid levels represent relevant activators. We have previously shown that trophoblasts have potent inflammatory properties, but it has not been determined how these sensors affects their communication with maternal immune cells in the decidua.

**Objectives:** We aimed to investigate cell specific inflammation through TLR2, TLR4 and NLRP3 in decidual trophoblasts and maternal immune cells, and assess the contribution to the harmful placental inflammation in preeclampsia.

**Methods:** Decidual biopsies obtained from 44 normal and 48 preeclamptic pregnancies during cesarean sections were analyzed by immunohistochemistry for cell markers and TLR2, TLR4, NLRP3 and IL-1 $\beta$  expression. Automated protein quantification was done in MATLAB. Decidual explants and trophoblasts were primed and stimulated with cholesterol crystals in vitro. IL-1 $\beta$  response was quantified by ELISA. Serum total cholesterol, uric acid, sFlt-1 and C-reactive protein (CRP) were measured by enzymatic assays or ELISA.

**Results:** TLR4, NLRP3 and IL-1 $\beta$  were markedly expressed by both trophoblasts and maternal immune cells in the decidua, while TLR2 was mainly expressed by maternal immune cells. Cholesterol crystals induced IL-1 $\beta$  in trophoblasts. Serum cholesterol levels were elevated in preeclamptic compared to healthy pregnancies, correlating to concentrations of hsCRP and sFlt-1. Quantification of danger sensor protein expression comparing normal and preeclamptic pregnancies will be presented.