myometrium volumes with and without power Doppler was collected at the first visit. All participants were followed until delivery. Each woman who had a preeclampsia was matched with one woman who delivered at term without pregnancy complications. First-trimester placental volume; vascularization index (VI); flow index (FI); and vascular flow index (VFI) of the entire placenta and the subplacental myometrium as well as mean uterine artery PI were separately measured for all cases and controls by an ultrasound technician blinded to the pregnancy outcome. Analyses were performed using non-parametric tests and area under the ROC curves were used to compared each marker for the prediction of preterm and term preeclampsia.

RESULTS: 1034 women were recruited over a 1-year period including 16 (1.5%) women who developed term preeclampsia and 4 (0.4%) who developed preterm preeclampsia. Women who developed preterm preeclampsia showed significant lower placental VI, placental VFI, subplacental VI and subplacental VFI compared to controls (all with $p<0.05$). However, women who developed term preeclampsia did not show any difference in terms of placental volume, placental vascularization or subplacental vascularization compared to controls. Measurements of AUC suggest that placental VI (0.87; 95%CI: 0.75-0.99; $p=0.02$) and placental VFI (0.83; 95%CI: 0.70-0.95; $p=0.04$) could be useful for the prediction of preterm preeclampsia.

CONCLUSION: First trimester placental and subplacental myometrium vascularization are significantly reduced in women who will subsequently developed preterm but not term preeclampsia.

75 Adverse outcomes are similar in preeclampsia with or without proteinuria: validation of a newly proposed definition of preeclampsia

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OBJECTIVE: To investigate the clinical performance of a newly proposed definition of preeclampsia by comparing outcomes of women with nonproteinuric preeclampsia to those with a traditional diagnosis.

STUDY DESIGN: Women with any signs or symptoms of preeclampsia (PE) were enrolled in a prospective, multicenter, observational study between 20 and 41 weeks. Blood was collected for biomarker development and subjects were managed according to local institutional protocols. Final diagnoses were adjudicated by an independent expert panel. Maternal and perinatal outcomes were compared between subjects with a traditional diagnosis of preeclampsia (Tr PE - mild, severe, or superimposed PE, eclampsia, or HELLP syndrome, per ACOG, 2002), and subjects with nonproteinuric preeclampsia (NP PE) who represent a new component in the 2013 ACOG diagnostic guidelines (gestational or chronic hypertension with any of the following, BP > 160/110, plts<100K, LFTs >70, Cr > 1.1, or persistent CNS or abdominal symptoms). Data stratified by gestational age and compared by X^2, $p<0.05$.

RESULTS: Of 1,223 evaluable subjects, 661 were diagnosed with Tr PE (54% of cohort), and 837 by the revised PE criteria (68%), representing a 27% increase. As compared to women with Tr PE, subjects with NP PE experienced the same rates of C/S, preterm birth <37 wk, SGA, perinatal mortality, and maternal adverse outcomes, regardless of gestational age category (Table 1). Only the rate of early preterm birth < 34 wks was lower in the women with NP PE (44 vs 60%).

CONCLUSION: The inclusion of nonproteinuric preeclampsia in the revised preeclampsia guidelines is justified by high rates of adverse outcomes equal to those women with traditional preeclampsia. This study confirms that proteinuria is sufficient, but not necessary, to identify women at risk of adverse outcome. The effect of an expanded definition of preeclampsia on rates of intervention (e.g. preterm birth) and adverse outcomes is unknown, and deserves further study.

Pregnancy outcome for traditional vs nonproteinuric preeclampsia

< 35+0 weeks (N=757)	Traditional Preeclampsia (N=465)	Nonproteinuric Preeclampsia (N=76)
C-section	329 (70.8)	55 (72.3)
Preterm <37 wk	400 (86.0)	65 (85.5)
Early preterm <34 wk	281 (60.4)	34 (44.7) *
SGA	204 (43.9)	31 (40.8)
Stillbirth	7 (1.5)	1 (1.3)
Neonatal death	10 (2.2)	2 (2.6)
Maternal adverse outcome	17 (3.7)	3 (3.9)
MAO or perinatal mortality	34 (7.3)	6 (7.9)
35-37 weeks (N=224)	**Traditional Preeclampsia (N=115)**	**Nonproteinuric Preeclampsia (N=49)**
C-section	55 (47.8)	27 (55.1)
Preterm < 37 wk	78 (67.8)	28 (57.1)
SGA	30 (26.1)	14 (28.6)
Maternal adverse outcome	2 (1.7)	1 (2.0)
≥ 37 weeks (N=242)	**Traditional Preeclampsia (N=81)**	**Nonproteinuric Preeclampsia (N=51)**
C-section	33 (40.7)	25 (49.0)
SGA	15 (18.5)	12 (23.5)
Maternal adverse outcome	1 (1.2)	1 (1.9)

Data reported as N (%). *$p<0.05$ by X^2. SGA, BW < 10%; MAO-maternal adverse outcome (abruption, renal failure, pulmonary edema/distress, fatty liver, TTP, DIC, stroke, and retinal detachment).

76 The role of LOX-1 receptor in sex-specific altered fetal programming of cardiovascular function in a preeclampsia-like murine model

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OBJECTIVE: Adult male mice exposed to preeclampsia in utero develop elevated blood pressure (BP) and decreased endothelium dependent relaxation compared to controls, most evident at 6 months of age, while female offspring do not. We previously showed that prenatal pravastatin prevented these changes. Lectin-like oxidized low density lipoprotein receptor 1 (LOX-1) is an endothelial cell receptor that is upregulated under inflammatory and oxidative conditions, and is associated with hypertension. Our objective was to evaluate the role of LOX-1 in these gender-specific phenotypic changes, and its response to prenatal pravastatin treatment, using a well-established animal model of preeclampsia.

STUDY DESIGN: CD-1 mice were injected with Adv-sFlt-1 and randomly allocated to pravastatin (sFlt-prav) or water (sFlt) until weaning. A control group was injected with adenovirus carrying the murine immunoglobulin G2α Fc fragment (mFc). Offspring were sacrificed at 3 and 6 months. RNA was extracted from the aorta, and mRNA expression of LOX-1 was measured using quantitative RT-PCR. Statistical analysis was performed using ANOVA and Student t tests.

RESULTS: There were no differences in LOX-1 mRNA expression between groups or gender at 3 months. At 6 months, LOX-1 mRNA in sFlt offspring was upregulated by more than 2 folds compared to mFc controls ($p=0.01$). Prenatal pravastatin reduced the mRNA to a level not significantly different from either sFlt-1 or control (Figure). In addition, at 6 months male offspring had significantly higher LOX-1 mRNA compared with females (6.25 ± 0.87 vs 3.19 ± 0.36, $p=0.01$).

CONCLUSION: Increased expression of LOX-1 parallels the temporal and gender-specific changes in adult cardiovascular function