istered either intramuscular (IM) or intranasal (IN). Mice were given booster doses of the same formulation and the same route of administration at 4 and 8 weeks. Serial serum and vaginal washings were obtained at weeks 4, 8, 11, 15, and 27 to determine antibody response. C5a specific IgG and IgA antibody responses were determined by colormetric ELISA. Mice were also challenged vaginally with GBS serotypes Ia, III, and V. After 24 hours, vaginal swabs were obtained, grown on blood agar plates, and number of GBS colonies were counted.

RESULTS: Of the various formulations and routes of delivery tested, both PLGA 75:25 30ug and 50:50 30ug IM and IN vaccines elicited a significant systemic IgG immune response with highest titers attained of 1:100,000. IgA immune responses were observed, but were inconsistent. For the vaginal colonization studies, mice vaccinated with PLGA 50:50 30ug and 75:25 30ug via the IM route showed no vaginal colonization with serotypes Ia and III. For GBS serotype V, less protective immunity was observed.

CONCLUSIONS: In the murine model, IM and IN administration of PLGA 50:50 30ug and 75:25 30ug vaccine formulations rendered a strong systemic immune response and prevented GBS colonization of the vaginal vault by serotypes Ia and III. Differences in protection to colonization by serotype V may be explained by differential C5a peptidase expression.

39 Pravastatin upregulates eNOS activity in the sFLT-1 mouse model of preeclampsia

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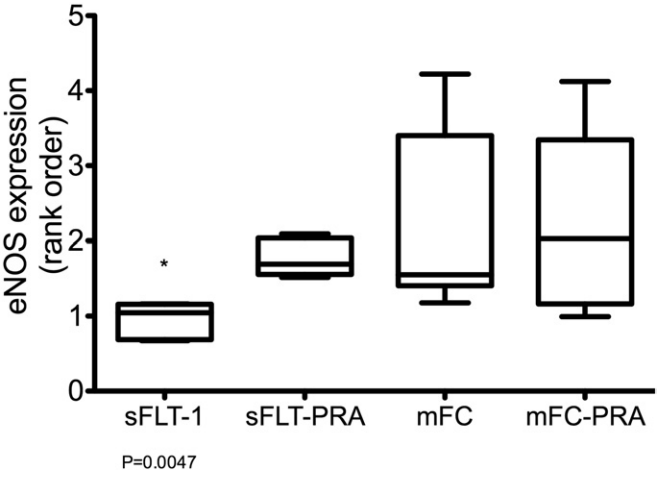
OBJECTIVE: We have previously shown that pravastatin administered to a well-characterized mouse model of preeclampsia decreases the production of the antiangiogenic sFlt-1 and prevents the associated vascular dysregulation. Endothelial nitric oxide synthase (eNOS) is the enzyme responsible for the production of nitric oxide, one of the most potent vascular smooth muscle relaxants. We hypothesize that pravastatin's action is mediated through eNOS, rather than cholesterol reduction or upregulation of mebrane bound vascular endothelial growth receptor-1 (VEGFR-1).

STUDY DESIGN: At day 8 of gestation, CD-1 mice were randomly allocated to injection of 10^9 PFU of the adenovirus carrying sFlt-1 or mFc control via the tail vein, and to receive pravastatin (Pra; 5 mg/kg/day) dissolved in drinking water or control. This resulted in 4 groups: sFlt, sFlt-Pra, mFc and mFc-Pra. Baseline and day 18 sera were collected. On day 18, dams were sacrificed and their aorta and kidneys collected and homogenized for western blot analysis for eNOS protein expression in the aorta and VEGFR1 expression in the kidneys. Western blot protein levels were normalized to actin. Serum cholesterol levels were determined using ELISA. Kruksal-Wallis with Dunn's post-hoc test was performed (statistical significance: $P<0.05$).

RESULTS: Pravastatin treatment in the sFlt group up-regulated eNOS expression by 60%, to levels similar to control mice ($P=0.005$; Figure). Cholesterol levels were not significantly different between groups, despite a trend toward lower levels with pravastatin administration. Renal VEGFR-1 levels were similar across groups.

CONCLUSIONS: Pravastatin administration prior to the onset of preeclampsia prevents the associated vascular dysfunction through pleiotropic effects by upregulating eNOS expression in the vasculature in the mouse model. This supports a role for statins in preventing the vascular abnormalities of preeclampsia.



40 Utility of an intrapartum rapid group B streptococcus test

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OBJECTIVE: Group B streptococcus (GBS) early-onset sepsis is a leading cause of neonatal morbidity and mortality. Two-thirds of infants with GBS sepsis are born to mothers with a negative third-trimester GBS culture—the current screening test. Given the transient nature of GBS colonization we evaluated the test characteristics of a rapid intrapartum GBS test and the current screening test along with the incidence of GBS conversion from the third trimester to intrapartum.

STUDY DESIGN: Women presenting to labor and delivery with an antepartum GBS culture were enrolled. Intrapartum recto-vaginal samples were obtained per CDC guidelines for the culture and rapid test. The intrapartum culture was the gold standard to calculate test characteristics and 95% confidence intervals.

RESULTS: Among 559 women who delivered 563 neonates, GBS prevalence was 19.5% with antepartum culture and 23.8% with intrapartum culture. Compared with intrapartum culture, antepartum culture had sensitivity of 69.2% (60.6-76.9) and specificity of 96.0% (93.7–97.7). The rapid intrapartum test showed sensitivity of 90.8% (84.6–95.2), specificity of 97.6% (95.7–98.9), positive predictive value of 92.3% (86.2–96.2) and negative predictive value of 97.2% (95.1–98.5). The incidence of GBS conversion from the late third trimester to labor was 10.4%. The time interval between the antepartum and intrapartum tests was the same for women who did not convert as for those who converted ($P=0.99$). Compared with women who identified as Caucasian, African-American ($P=0.02$) and Hispanic ($P=0.02$) women were significantly more likely to convert. The incidence of neonatal blood culture was 35% among women who converted to negative versus 17% among women who remained negative.

CONCLUSIONS: This intrapartum rapid GBS test has excellent test characteristics and may be superior to the antepartum culture for accurately detecting intrapartum GBS colonization. Use of this rapid intrapartum test may improve the precision of neonatal sepsis evaluations, and may thus impact the incidence of neonatal GBS sepsis.