Title: Low dose aspirin influence on biomarkers of endothelial disruption and inflammation

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Objective: To determine the low dose aspirin (LDA) on biomarkers related to inflammation, CRP, and endothelial activation, S-ICAM, which are potentially important in the pathogenesis of preeclampsia

Study Design: This was a prospective cohort study. Women aged 18-45 years with singleton gestation and who were defined as high risk for preeclampsia per United States Preventive Services Task Force guidelines were enrolled as cases. A convenience control sample of low risk women were enrolled as controls. Daily LDA was initiated between 11 and 16 weeks and continued until 36 weeks. Serum CRP and S-ICAM were assayed prior to aspirin initiation at 11-16 weeks and repeat assays were obtained at 28-32 weeks. Primary outcome included changes in CRP and S-ICAM levels over time reported as mean and standard error. T test and chi-square tests assessed comparisons of demographic data. Initial analyses were performed using mixed models for repeated measures to detect aspirin effects

Results: 132 women were enrolled: 67 on LDA, 65 were controls. Comparison of demographic variables identified significant differences in BMI, occupation, educational levels and insurance status between groups. No significant treatment effects were noted in CRP levels(ng/ml) between cases and controls at 11-16 weeks(14.16 ± 1.37 vs. 11.17 ± 1.38 SE) and at 28-32 weeks (13.53 ± 11.61 vs. 10.48 ± 1.18 SE). S-ICAM levels(ng/ml) were higher in cases than in controls at 11-16 weeks(77.8 ± 2.18 Evs 70.4 ± 1.6 SE) with a decline in cases and increase in controls at 28-32weeks(76.7 ± 3.2 SE vs 75.5 ± 2.2 SE). S-ICAM levels showed a significant treatment effect, P=.0097, in our repeated measures model. The direction of effect was to lower this value during the treatment interval

Conclusion: We observed a treatment effect of LDA on reduced endothelial activation in this cohort study suggesting a mechanism of action for this intervention beyond known effects related to thromboxane metabolism