1132 Maternal *Campylobacter rectus* Infection Attenuates Murine Placental Growth Factor Expression

**Y.A. BOBETSIS**¹, D. LIN¹, E.L. RICHE¹, C.M. CHAMPAGNE¹, S.P. BARROS², and S. OFFENBACHER¹, ¹ University of North Carolina, Chapel Hill, USA, ² State University of Campinas - Piracicaba, Chapel Hill, NC, USA

**Objective:** Previous human studies have demonstrated that maternal periodontal disease increases the risk for intrauterine growth restriction. We have developed a murine model using the periodontal pathogen *Campylobacter rectus* (*C. rectus*) to study the mechanisms of fetal growth restriction (FGR). Our hypothesis is that *C. rectus* infection induces a reduction in the expression of placental growth factors, which may contribute to an impaired development of the placenta and of the fetus. **Methods:** Pregnant BALB/c mice were challenged with *C. rectus* or saline at embryonic gestation day (ED) 7.5. Mice were sacrificed at ED 16.5 and placentas were collected for histological analysis or for total RNA extraction. Differential gene expression analysis was performed using Agilent 60-mer oligonucleotide microarrays comparing mRNA expression from placentas of FGR fetuses from challenged dams versus placentas from control dams. After LOWESS normalization, data were analyzed using the Bootstrap method at a false positive discovery rate of <0.05. **Results:** Among the many differentially expressed genes a strong pattern involving suppression of growth factors and related signaling and receptor pathways was noted. mRNA expression of insulin-like growth factor II (IGF-II) and placental growth factor (PGF) showed a 2.12 (unchallenged: 4.82±1.79; FGR: 2.27±0.56) and 2.45 (unchallenged: 3.26±0.45; FGR: 1.33±0.16) (Mean±SE) fold decrease respectively in the placentas of the growth restricted fetuses compared to the control placentas. Histological findings were consistent with placental growth factor suppression including a decrease of the labyrinth/decidua ratio in the FGR placentas. **Conclusion:** IGF-II and PGF have been reported to play an important role in murine placental development, nutrient exchange and fetal growth. Our data suggest that *C. rectus* infection in pregnant dams is associated with reduced expression of these growth factors, which may, in part contribute to abnormal placental development and impaired fetal growth. Work supported by NIDCR DE-012453 and UNC University Research Council #3-12708.

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