251 FIRST TRIMESTER UTERINE ARTERY DOPPLER, PAPP-A AND 3D POWER DOPPLER OF THE INTERVILLOUS SPACE IN PATIENTS AT RISK FOR PREECLAMPSIA JULIANA GEBB[1], ELLEN LANDSBERGER[1], IRWIN R. MERKATZ[1], PEER DAR[1], [1]Montefiore Medical Center/Albert Einstein College of Medicine, Obstetrics & Gynecology and Women's Health, New York, New York

OBJECTIVE: Uterine artery pulsatility index (UAPI), PAPP-A levels and 3D power Doppler (3DPD) of the intervillous space are potential screening markers for preeclampsia (PEC) in the first trimester. As PEC most commonly develops in women with known risk factors, we sought to compare these markers between patients with risk factors for PEC (high risk-HR) and those without (low risk-LR).

STUDY DESIGN: We conducted a prospective observational study of singleton pregnancies undergoing first trimester screening. Risk factors for PEC included nulliparity, history of PEC, chronic hypertension, pregestational diabetes, thrombophilia, sickle cell disease, lupus or renal disease. UAPI was measured in both arteries and PAPP-A was measured as part of the aneuploidy screen. 3DPD volumes of the intervillous space were acquired and then analyzed with the VOCAL program (GE Kretz, Zipf, Austria) to determine the Vascularization Index (VI), Flow Index (FI) and Vascularization Flow Index (VFI). Values were compared between HR and LR groups using the student's t-test. $P<0.05$ was considered statistically significant.

RESULTS: 255 patients enrolled. 127 patients had 1 or more risk factors for PEC and 128 patients had no risk factors. All 3DPD indices were significantly lower in the HR group (VI 19.8±11.6 vs.25.5±12.8, FI 50.2±7.9 vs.53.6±9.1, VFI 10.5±7.2 vs.14.2±8.2). In contrast, no differences were identified in PAPP-A and UAPI values between the groups (PAPP-A 1.35±0.8 vs. 1.3±0.7, UAPI 1.77±1.0 vs. 1.85±1.2).

CONCLUSION: Patients at risk for PEC have decreased 3DPD indices, suggesting reduced blood vessel density and corpuscle volume in their intervillous spaces during the first trimester. This finding implies a higher incidence of abnormal placentation in this group. Our results further imply that 3DPD may provide a more direct assessment of the abnormal placentation process than do UAPI or PAPP-A levels.

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252 THE FREQUENCY OF ELECTIVE DELIVERY AT 34.0-36.9 WEEKS' GESTATION AND ITS' IMPACT ON NEONATAL OUTCOMES IN WOMEN WITH STABLE MILD GESTATIONAL HYPERTENSION (MGHTN) JOHN BARTON[1], LUCY BARTON[2], NIKI ISTWAN[3], CHERYL DESCH[3], DEBBIE RHEA[3], GARY STANZIANO[3], BAHA SIBAI[4], [1]Central Baptist Hospital, Obstetrics & Gynecology, Lexington, Kentucky, [2]University of the South, Sewanee, Tennessee, [3]Matria Healthcare, Clinical Research, Marietta, Georgia, [4]University of Cincinnati, Cincinnati, Ohio

OBJECTIVE: There is concern about the rate of late-preterm (L-PT) birth and its' impact on neonatal morbidity. We examined frequency of elective delivery and neonatal outcomes in women with stable mGHTN delivering L-PT.

STUDY DESIGN: 1882 singleton gestations with mGHTN without proteinuria enrolled for outpatient surveillance were studied: 607(32.3%) delivered due to labor/PROM or maternal/fetal reasons; 642(34.1%) electively at ≥38 weeks); 633(33.6%) electively L-PT for mGHTN. Neonatal outcomes compared between those delivering L-PT vs. 37.0-37.9 weeks and ≥38 weeks using Student's ***t***, Mann-Whitney U, or Pearson's chi-square statistics.

RESULTS: Table shows neonatal outcome in 1275 with mGHTN and elective delivery. NICU admission occurred in 88/323 (27.2%) delivered L-PT, 31/310 (10.0%) at 37.0 weeks ($p<0.002$) and 34/642 (5.3%) at ≥38 weeks ($p<0.002$). Ventilatory assistance was required in 23/323 (7.1%) delivered L-PT and 3/642 at ≥38 weeks ($p<0.002$). RDS was diagnosed in 19/323 (5.9%) delivered L-PT, 7/310 (2.3%) at 37 weeks ($p=0.044$) and 6/642 (0.9%) at ≥38 weeks ($p<0.002$). There was no maternal/perinatal mortality.

Outcome *Median (range)	34 wks (n=39)	35 wks (n=100)	36 wks (n=184)	37 wks (n=310)	≥38 wks (n=642)
NICU admission	56.4%	38.0%	15.2%	10.0%	5.3%
Days in NICU*	10 (1, 30)	7 (1, 35)	5.5 (1, 20)	7 (1, 22)	3.5 (1, 31)
Asst ventilation	10.3%	10.0%	4.9%	3.9%	0.5%
Total nursery (d)*	7 (1, 30)	4 (1, 35)	3 (1, 23)	3 (1, 22)	2 (1, 31)

CONCLUSION: We found that 33% of patients with stable mGHTN without proteinuria had iatrogenic elective delivery L-PT which may be one of the reasons for the increased rate of L-PT birth in the US. This practice was associated with increased rates of neonatal complications and neonatal length of stay.

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253 VASCULAR ENDOTHELIAL GROWTH FACTOR (VEGF) SINGLE NUCLEOTIDE POLYMORPHISMS (SNPS) IN UNEXPLAINED STILLBIRTHS (SB) FRANCESCA FERRARI[1], FABIO FACCHINETTI[2], HUAIZHI YIN[1], MONICA LONGO[1], GEORGE R SAADE[1], [1]The University of Texas Medical Branch, Obstetrics and Gynecology, Galveston, Texas, [2]University of Modena and Reggio Emilia, Obstetrics and Gynecology, Modena, Italy

OBJECTIVE: VEGF plays a crucial role in placental vasculogenesis. VEGF imbalance has been implicated in the pathogenesis of preeclampsia and other perinatal complications. Previous studies suggest that SNPs in the VEGF gene contribute to abnormal vascular function in various medical conditions. Our objective was to compare the distribution of VEGF gene SNPs between unexplained SB and control in a well-characterized cohort.

STUDY DESIGN: Placentas were obtained from SB and healthy controls. Following an extensive workup, SB were classified as unexplained using a modified version of the Wigglesworth system. Placental DNA was extracted using Qiagen kits, and evaluated for six VEGF SNPs (−2578C/A, −936C/T, −1154 G/A, −634 G/C, −460C/T, −405G/C) by real time PCR using specific taqman probes. The genotype and allelic distributions were analyzed by Chi-square ($p<0.05$ was considered statistically significant).

RESULTS: Placentas from 33 unexplained SB and 33 healthy controls were included. All SNPs were in Hardy-Weinberg equilibrium. Genotypes −2578 C/A and −460 C/T were significantly less frequent in SB compared to controls ($p<0.005$). VEGF-460 T allele occurred more frequently in SB placentas compared with controls ($p=0.02$). No significant associations between SB and the genotypic or allelic frequencies of the other SNPs were found.

CONCLUSION: Genotypes −2578 C/A and −460 C/T are protective, but the VEGF-460 T allele is associated with unexplained stillbirths. A role for placental VEGF imbalance in the etiology of stillbirths is also supported by the well-characterized association between these SNPs and altered VEGF production. Further studies focusing on VEGF for prediction and/or prevention of stillbirths are warranted.

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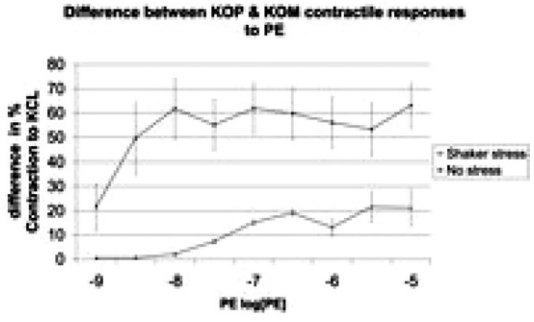
254 EFFECT OF POSTNATAL STRESS ON THE ALTERED FETAL PROGRAMMING OF VASCULAR FUNCTION. MAGED COSTANTINE[1], FRANCESCA FERRARI[1], EGLE BYTAUTIENE[1] BENJAMIN BYERS[1], PHYLLIS ORISE[1], GEORGE R SAADE[1], MONICA LONGO[1], [1]The University of Texas Medical Branch, Obstetrics and Gynecology, Galveston, Texas

OBJECTIVE: We have previously shown that heterozygous mice offspring born to transgenic mothers lacking endothelial nitric oxide synthase (NOS3) have altered vascular function in later life compared to offspring born to wild type mothers. Our objective in the current study was to determine the effect of postnatal stress on the altered vascular function in this animal model of fetal programming.

STUDY DESIGN: Homozygous NOS3 knockout (KO) and wild type mice (WT) were cross-bred to produce maternally-(KOM) and paternally-derived heterozygous (KOP) litters (n=10-12 per group). Offspring from both groups were stressed by placing them in a special caging system attached to a shaking platform that provided programmed intermittent shaking. Animals were sacrificed at 14 weeks of age, and the carotid arteries were prepared for in vitro vascular reactivity. Vascular responses to cumulative concentrations of phenylephrine (PE), in the presence and absence of the non-selective NOS inhibitor (L-NAME) were determined. Response following stress were compared with those obtained in similar unstressed offspring. ANOVA followed by Neuman-Keuls post hoc test were used for statistical analysis (significance: $P<0.05$).

RESULTS: KOM mice offspring had significantly higher contractile responses to PE before and after L-NAME incubation when compared to KOP. However, the difference in these contractile responses between KOP and KOM was significantly less at every PE concentration when animals were stressed compared to non-stressed animals (figure).



CONCLUSION: Postnatal stress decreases the difference in vascular function induced by fetal programming. This is largely the result of worsening the vascular function in the normal offspring. The effects of postnatal stress appear to parallel the fetal programming effects of an adverse uterine environment.

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