

tively). Length of labor did not correlate with either umbilical vein or artery's glucose concentration ($p = 0.82$ & 0.83 respectively).

CONCLUSION: Our data indicates that labor is associated with increased umbilical vessel glucose measurements in normal pregnancies. These differences are not related to any variation in maternal glucose levels. While this increase in cord glucose concentration may be related to fetal stress in labor, cord pH and oxygen content changes (indicating significant hypoxic/acidotic stress) were not seen in the laboring patients.

	Umbilical Vein			Umbilical Artery		
	Group A Vag delivery n=20	Group B C section n=15	P	Group A Vag delivery n=20	Group B C section n=15	P
Glucose (mg/dl)	90.2 +/- 13.5	74.3 +/- 12.7	0.0013	77.8 +/- 17	62.7 +/- 12.6	0.0085
ph	7.3 +/- 0.04	7.31 +/- 0.04	0.49	7.24 +/- 0.05	7.26 +/- 0.04	0.5
pO2 (mm of Hg)	28 +/- 7.1	27 +/- 7.5	0.84	19.8 +/- 4.7	19 +/- 8	0.72
pCO2 (mm of Hg)	43.8 +/- 6.7	46.4 +/- 7.6	0.3	57 +/- 9.7	61 +/- 8.6	0.24

231 Prenatal hypoxia programs increased hepatic mitochondrial gene expression in guinea pig (GP) offspring

Sheveta Jain¹, Yazan Al-Hasan¹, Loren Thompson¹
¹University of Maryland School of Medicine, Obstetrics & Gynecology, Baltimore, MD

OBJECTIVE: Intrauterine hypoxia is a prenatal insult that contributes to fetal organ dysfunction. Permanent changes in organ function of the offspring may be mediated via programming mechanisms. We previously reported that chronic fetal hypoxia increases mitochondrial enzyme activity associated with fatty acid oxidation (FAO; MCAD, medium chain acyl dehydrogenase) and oxidative phosphorylation (OXPHOS; CCO, cytochrome c oxidase) in 90d old GP livers compared to their normoxic controls (SGI2012 Abstract #F120). We hypothesize that increased mitochondrial enzyme activity by intra-uterine hypoxia is mediated via upregulation of target genes [Hepatic MCAD (protein & mRNA), PPAR α & γ (mRNA, peroxisome proliferator-activated receptors)] associated with mitochondrial protein expression.

STUDY DESIGN: Pregnant GPs were exposed to either room air (normoxia NMX) or 10.5% O₂ (hypoxia HPX) for 14d prior to term (65d). Fetal GPs were allowed to deliver and male offspring were selected for study and housed in room air. At 90d, offspring (N=11-12/grp) were anesthetized, body and liver weights measured, and right liver lobes excised and frozen (-80°C). MCAD protein of isolated mitochondrial fractions were measured by Western analysis and normalized to Porin. MCAD & PPAR α & γ mRNA levels were measured by RTPCR using appropriate primers, normalized to 18srRNA.

RESULTS: Chronic hypoxia had no effect on body or liver weights. Exposure to prenatal hypoxia increased ($p < .05$) both MCAD protein (NMX vs HPX 0.35 ± 0.10 vs 0.83 ± 0.17) & mRNA (NMX vs HPX 0.57 ± 0.05 vs 0.81 ± 0.05) levels of offspring livers. PPAR α (NMX vs HPX 0.14 ± 0.02 vs 0.25 ± 0.03) but not PPAR γ mRNA (NMX vs HPX 0.21 ± 0.04 vs 0.28 ± 0.05) levels were increased ($p < .05$) by prenatal hypoxia in offspring livers.

CONCLUSION: These results suggest that in-utero exposure to chronic hypoxia upregulates hepatic MCAD enzymatic activity via transcription factors that regulate FAO, suggesting that prenatal hypoxia programs altered liver metabolism in the offspring (NIH HL49999).

232 Correlation of serum fructosamine and recurrent pregnancy loss

Stephanie Romero¹, Margarita Sharshiner¹, David Branch¹, Robert Silver¹

¹University of Utah, Maternal Fetal Medicine, Salt Lake City, UT

OBJECTIVE: Pre-gestational diabetes is associated with an elevated risk of pregnancy loss. However, it is unclear whether subclinical levels of glucose intolerance are associated with pregnancy loss, especially re-

current pregnancy loss (RPL). Thus, our objective was to compare maternal serum fructosamine (a marker of glycemic control) in patients with and without RPL.

STUDY DESIGN: Case-control study design with 134 women with unexplained RPL, defined as two or more pregnancy losses with no more than one live birth and 134 age-matched controls with at least one full term uncomplicated pregnancy and no more than one pregnancy loss. No cases or controls had a clinical diagnosis of pre-gestational or gestational diabetes. Maternal serum fructosamine was measured using quantitative spectrophotometry.

RESULTS: The groups were similar with regard to age, race and ethnicity. The mean BMI of cases was 26.4 (17.8-51.2) compared to 26.4 (17.8-44.4), $p = 0.91$. Fructosamine levels were higher in women with RPL (225.3 ± 38.5) compared to controls (189.3 ± 19.5 , $p < 0.001$). This was also seen when the cases and controls were stratified by BMI (see table). However, the proportion of women with elevated levels of fructosamine considered diagnostic of diabetes ($\geq 285 \mu\text{mol/L}$) was similar in cases and controls (6.0 versus 12.7%; $p = 0.092$).

CONCLUSION: Cases and controls had a similar proportion of women with elevated levels of fructosamine considered diagnostic of clinically relevant glucose intolerance. However, maternal serum levels of fructosamine were increased in women with RPL compared to controls. Thus, subclinical levels of glucose intolerance may be associated with an increased risk of RPL. Although these data support further investigation into the mechanisms of pregnancy loss associated with glucose intolerance, they do not support testing for subclinical glucose intolerance on women with RPL.

Fructosamine levels stratified by BMI

BMI	Cases	Controls	p
<25	230.11 +/- 30.5	188.27 +/- 18.6	<0.001
≥ 25	216.05 +/- 26.76	185.72 +/- 16.96	<0.001

Fructosamine measured in $\mu\text{mol/L}$.

233 Transplacental transfer of pravastatin

Tatiana Nanovskaya¹, Svetlana Patrikeeva¹, Maged Costantine¹, Gary Hankins¹, Mahmoud Ahmed¹

¹University of Texas Medical Branch, OG/GYN, Galveston, TX

OBJECTIVE: Determine the bidirectional transfer of pravastatin across the dually perfused term human placental lobule and its distribution between the tissue, maternal and fetal circuits.

STUDY DESIGN: The technique of dual perfusion of placental lobule (DPPL) was utilized to determine the Maternal-to-Fetal (n=11) and Fetal-to-Maternal (n=10) transfer of pravastatin. The concentration of pravastatin in the maternal reservoir (50 ng/mL) was equal to the reported mean plasma concentration of the drug in patients who received a dose of 40 mg of pravastatin daily. Pravastatin was co-perfused with its [3H]-isotope and the marker compound antipyrine (AP, 20 $\mu\text{g/mL}$) and its [14C]-isotope. The concentration of pravastatin in the perfused tissue, the maternal and fetal circuits was determined using liquid scintillation spectrometry. Inside-out vesicles (IOV) prepared from placental brush border membranes were utilized to investigate pravastatin interactions with efflux transporters.

RESULTS: Pravastatin was transferred from the maternal to the fetal circuit and vice versa. In the Maternal-to-Fetal direction $14 \pm 5\%$ of the drug was retained by the tissue, $68 \pm 5\%$ remained in the maternal circuit, and $18 \pm 4\%$ was transferred to the fetal circuit. The normalized transfer of pravastatin (Clearance index) to AP in the Fetal-to-Maternal direction (0.48 ± 0.07) was higher than its transfer in the Maternal-to-Fetal direction (0.36 ± 0.07 , $p < 0.05$). Furthermore, pravastatin inhibited the ATP-dependent uptake of the [3H]-paclitaxel and [3H]-estrone sulfate by IOV.

CONCLUSION: The transfer (20%) of pravastatin across the dually perfused placental lobule suggests that fetal exposure to pravastatin is plausible. Moreover, the higher transfer of pravastatin in the Fetal-to-Maternal direction than the reverse as well as inhibition of the ATP-