load 4 hours prior to study. On D19 of gestation, dams were given IV radiolabeled 18F-fluorodeoxyglucose to measure tissue GU and mPET was performed. ASIPRO (Siemens) software was used to localize regions of interest (ROI), defined as 5 image slices representing maximum concentration of fetus/placenta. For each animal's ROI, the mean GU was calculated in SUVs (standard uptake value). A 3-point scale quantified maternal cardiac GU (0=none, 1=minimal, 2=maximal).

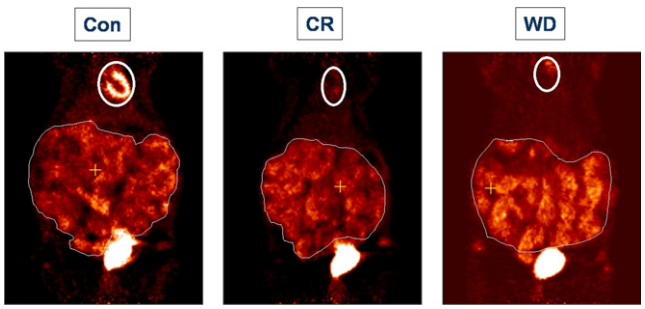
RESULTS: CON weighed more than CR (302±8g vs 268±14g,p<0.01) and WD more than Con and ALL(384±9.6g vs 302±8g, 297±18g p<0.01). Pup weight and litter size were similar between groups. CR and WD dams demonstrated increased fetal-placental GU and decreased maternal cardiac GU compared to CON [Figure]. Similarly, in response to acute lipid load, ALL dams had increased fetal-placental GU with a decrease in maternal cardiac GU [Table].

CONCLUSION: Similar to acute lipid load, chronic maternal CR and WD results in increased fetal-placental GU and decreased maternal cardiac GU. These findings suggest that both calorie restriction and chronic high fat feeding in pregnancy induces greater maternal reliance on lipid oxidation, therefore making more glucose available for transport to the fetus. In pregnant models, mPET may be a valuable tool not only to measure in vivo glucose transport, but also transport of other nutrients, such as lipids.

Maternal cardiac glucose uptake and traced region of interest (ROI)

Group	Fetal=Placental GU (mean SUV)	Maternal Cardiac GU (mean uptake score)
CON	1.23 (±0.2)	2
CR	1.58 (±0.3)*	0.44*
WD	2.09 (±0.4)*	0.43*
ALL	1.7 (±0.3)*	0.57*

*$P < .05$ compared to CON.



229 Risk factors for iodine deficiency in pregnancy

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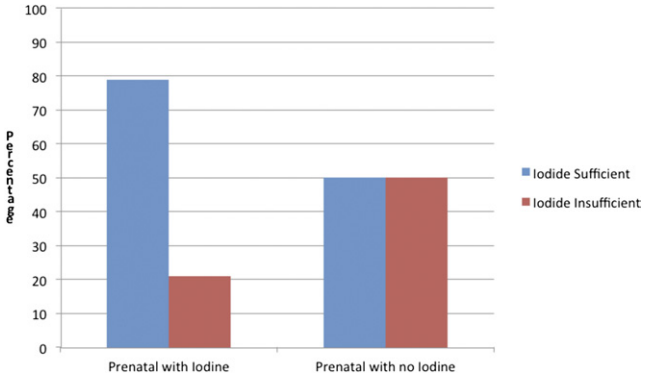
OBJECTIVE: Iodine deficiency during pregnancy is associated with an increased risk for complications of pregnancy and a 13.5 decrease in IQ in exposed offspring. The recommended daily intake of iodine is 220 mcg, of which approximately 100 mcg comes from diet. Surprisingly, many prenatal vitamins sold in the US do not contain any iodine. To assess urinary iodide levels in a population of pregnant women and correlate these results with iodine amounts in their prenatal vitamins and dietary intake of other sources of iodine.

STUDY DESIGN: An observational prospective study was performed from 2010-2012 at a single academic medical center. Urinary iodide samples were collected from pregnant women between 20-28 weeks of gestation. Subjects completed a research validated dietary survey. Prenatal vitamin type, iodine content and compliance were recorded. Bivariable comparisons were made with Chi-square analysis. A multivariable linear regression model was created to control for demographic and dietary variations.

RESULTS: 280 subjects were enrolled in the study. The median urinary iodide level in this population was 130.5 mcg/L (IQR 74 mcg/L). Iodide deficiency (<100 mcg/L) was seen in 35 % of the pregnant patients. (98/280) Severe iodide deficiency (<50 mcg/L) was noted in 11.4 %. (30/280). The iodine content of prenatal vitamins was strongly correlated with iodide level. (p<.001). Taking a prenatal vitamin without iodine was associated with a significantly increased risk for iodide deficiency. (RR 2.2 95 % CI 1.3–3.3). After controlling for demographics and seafood and dairy consumption, only lack of prenatal vitamin iodine content remained significantly associated with risk of iodine deficiency.

CONCLUSION: Iodine deficiency is common among pregnant women and is associated with lack of iodine in prenatal vitamins. Patients should be advised to take a prenatal vitamin containing 150 mcg of iodine from potassium iodide, or an equivalent supplement. Iodine should be included in prenatal vitamins as a matter of public policy.

Maternal iodine insufficiency by prenatal vitamin



230 Effect of labor on glucose concentrations in umbilical veins & arteries

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OBJECTIVE: To assess the effects of labor on fetal circulating glucose concentrations as measured in umbilical vein & artery.

STUDY DESIGN: The study population included two groups - those who had vaginal delivery after undergoing labor (Group A,n=20) and those that underwent elective c-section (controls - Group B,n=15). Patients with medical complications (including diabetes, hypertension, systemic infections, multiple pregnancies) were excluded. Patients in both the groups received lactate ringer's solution. Maternal glucose was measured at the delivery of fetus in both the groups. Umbilical cord vessel samples were collected in heparinized syringes immediately after delivery. From that sample, glucose was analyzed using SureStep Flexx glucometer. The remaining sample was sent for cord gas evaluation using GEM Premier 4000 analyzer's iQM method.

RESULTS: The two groups, A vs B, did not differ significantly in maternal age (26.4 vs 27.3 y; p=0.56), gestational age (39.5+/−0.5 vs 39.2+/−0.2, p=0.09), parity (55% vs 86% multiparous; p=0.07) or birthweight (P=0.96). Maternal glucose was comparable in both the groups (97.46+/−12.9 vs 91.38+/−13.7 mg/dl,p=0.08). Umbilical venous glucose was significantly higher in group A (90.2+/−13.5 vs 74.3+/−12.7,p=0.001). Umbilical artery glucose was also elevated in group A (77.8+/−17 vs 62.7+/−12.6, p=0.008). Umbilical vein and artery pH was similar in both the groups. Also, the umbilical vein and artery pO2 was comparable in both groups (p=0.84 & 0.72 respec-

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

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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 intrauterine 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 (−80oC). MCAD protein of isolated mitochondrial fractions were measured by Western analysis and normalized to Porin. MCAD & PPARα & 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. PPARα (NMX vs HPX 0.14±0.02 vs 0.25±0.03) but not PPARγ 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

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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 recurrent 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 (≥ 285 μmol/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 umol/L.

233 Transplacental transfer of pravastatin

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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 ug/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 vise versa. In the Maternal-to-Fetal direction 14 ± 5% of the drug was retained by the tissue, 68 ± 5% remained in the maternal circuit, and 18±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-