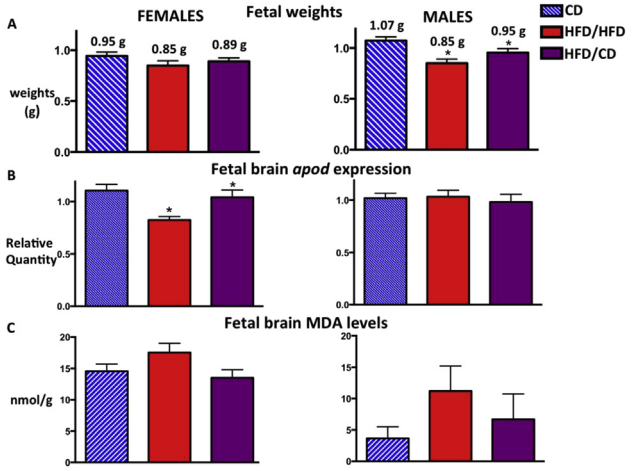
exposed to MATOB (HFD/HFD and HFD/CD) had significantly higher MDA levels than Ms (p=0.02, Fig 1C).

CONCLUSION: MATOB had a more significant effect on M embryo size. However, MATOB had more significant effects on F brain *apod* expression and MDA levels, two parameters related to brain oxidative stress. Maternal diet in pregnancy also impacted fetal brain gene expression of *apod* in Fs only. MATOB and diet in pregnancy appear to have sex-specific influences on embryo size and on the fetal brain. Future evaluation of the effects of MATOB on fetal neurodevelopment should consider fetal sex and maternal diet.



207 The intestinal microbiome is regulated by diet in a novel primate model of polycystic ovarian syndrome (PCOS)

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OBJECTIVE: Polycystic ovarian syndrome (PCOS) is a disorder characterized by oligo-ovulation and hyperandrogenism. PCOS confers risk of infertility, obesity, insulin resistance and diabetes. Afflicted gravidae are at risk of perinatal complications (i.e. gestational diabetes, preeclampsia, hypertension, cesarean delivery and preterm birth). Further, hyperandrogenism confers insulin resistance upon consumption of a high fat diet. The microbiome is altered in obesity and diabetes and is implicated in childhood obesity via cesarean delivery. However, it is unclear if the microbiome is a cause or symptom of altered metabolism. Thus, in order to examine the role of the microbiome in insulin resistance and obesity in PCOS, we examined the influence of diet and androgens on the microbiome in a novel non-human primate model.

STUDY DESIGN: Young female Rhesus macaques received either a cholesterol (control, n=20) or testosterone (experimental, n=20) implant. Additionally, these groups were further distinguished by diet, which resulted in four distinct cohorts (n=10 per cohort, Table 1). Oral and anal swabs and stool were collected before (0) and at 3, 6, 12, and 18 months post-treatment. 16S sequencing was performed on isolated DNA.

RESULTS: Within the oral and intestinal microbiome, we found differences between baseline and all treatment time points (p=0.001, Fig A). For instance, Ruminococcus, Catenibacterium, and Treponema species in the intestinal microbiome are similar between cohorts at baseline, but these bacteria separate the cohorts based on diet as treatment progresses (Fig B). To further investigate cohort separation based on diet, we generated a heatmap showing that multiple taxa are altered by diet (p=0.001) rather than by testosterone treatment (p≤0.52) within the intestinal microbiome (Fig C).

CONCLUSION: These results demonstrate a minimal role for testosterone in altering the intestinal microbiome, whereas a high fat diet significantly alters the microbiome of exposed female macaques. These data further demonstrate that diet influences the microbiome rather than the microbiome acting independently of diet.

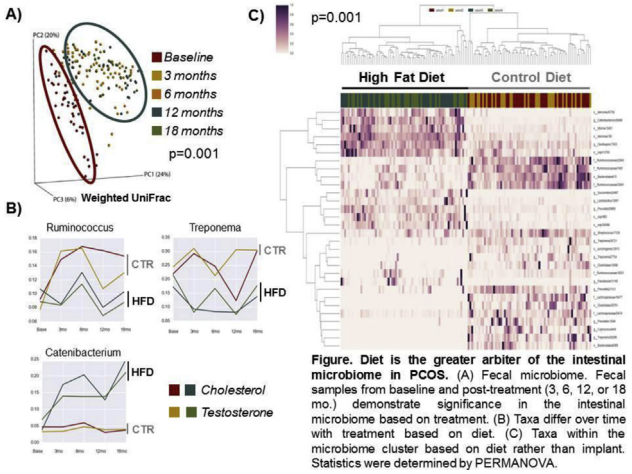

Figure. Diet is the greater arbiter of the intestinal microbiome in PCOS. (A) Fecal microbiome. Fecal samples from baseline and post-treatment (3, 6, 12, or 18 mo.) demonstrate significance in the intestinal microbiome based on treatment. (B) Taxa differ over time with treatment based on diet. (C) Taxa within the microbiome cluster based on diet rather than implant. Statistics were determined by PERMANOVA.

	Implant	Diet
Cohort 1	Cholesterol	Control
Cohort 2	Testosterone	Control
Cohort 3	Cholesterol	High fat
Cohort 4	Testosterone	High fat

Table 1. Non-human Primate Model of PCOS.

208 Obesity delays the onset of lactation and alters the fatty acid composition of milk

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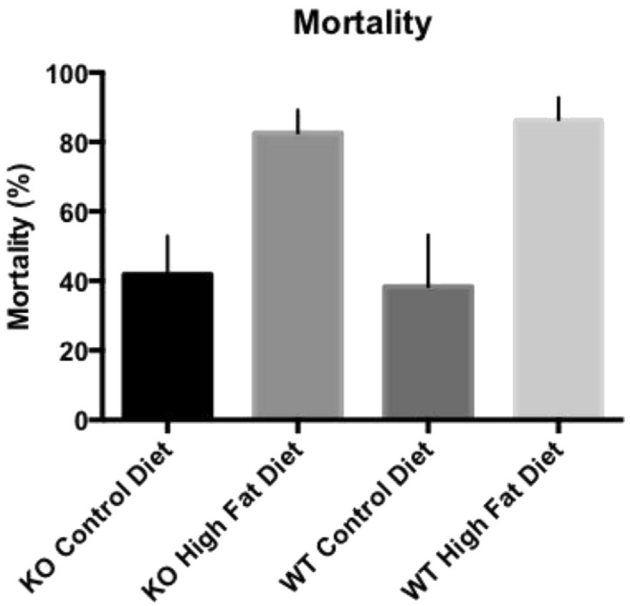
OBJECTIVE: Obesity is a known risk factor for delayed onset of stage II lactogenesis in epidemiologic studies. The underlying mechanism of this association remains elusive. Serotonin is a regulator of mammary gland function and is elevated in obese individuals. We hypothesized that obesity promotes inflammation resulting in early mammary gland involution through a serotonin dependent mechanism.

STUDY DESIGN: Mice fed a high fat diet (HFD) starting at 5 weeks of age were compared to mice fed a control diet (LFD). Wild type (WT) mice were compared to those genetically deficient for TPH-1, the rate-limiting enzyme in peripheral serotonin synthesis. Serum serotonin concentrations were collected prior to diet initiation, and on d 1 and 10 of lactation. Milk yields were measured daily from d1-10 of lactation. Milk samples were collected daily and analyzed using gas chromatography to determine fatty acid profiles. Mice were euthanized on d 10 of lactation and mammary glands were harvested. The number of intact alveoli and mean alveolar diameter were measured from histologic sections. Cytokine expression profiles were determined using PCR arrays.

RESULTS: Twelve wild type (WT) mice (HFD=4, LFD=8) were compared with sixteen mice deficient for TPH-1 (HFD=6, LFD=10). Total pup mortality was higher in the HFD (86%) than the LFD (37.3%) dams (p<.001). Milk yield was decreased on d 1 in

WT dams on HFD but not in TPH-1 KO dams. WT mice had higher serum serotonin levels than TPH-1 KO mice at baseline (p<.001). Mean alveolar diameter did not differ among groups. Pup weights were not different among groups. Mammary gland expression of Cxcl5 and Ccl22 were increased in the WT HFD group compared to controls (p<.05). Expression of Cxcl2, Ly96, IL1rap, Il1b were decreased in the HFD WT group compared to controls (p<.05). In the knockout mice, cxcl5 and cxcl2 were elevated and ccl22 was lower in HFD mice compared to controls (p<.05). Mice fed a HFD had elevated levels of the polyunsaturated fatty acids oleic acid and linoleic acid on days 2-6 of lactation (p<.05).

CONCLUSION: Obesity delays onset of successful lactation in a mouse model, but maintenance of lactation is not affected once it is established. The fatty acid composition of milk is altered in the setting of obesity.



209 Implementation of universal screening for depression during pregnancy: Feasibility and impact on obstetric care

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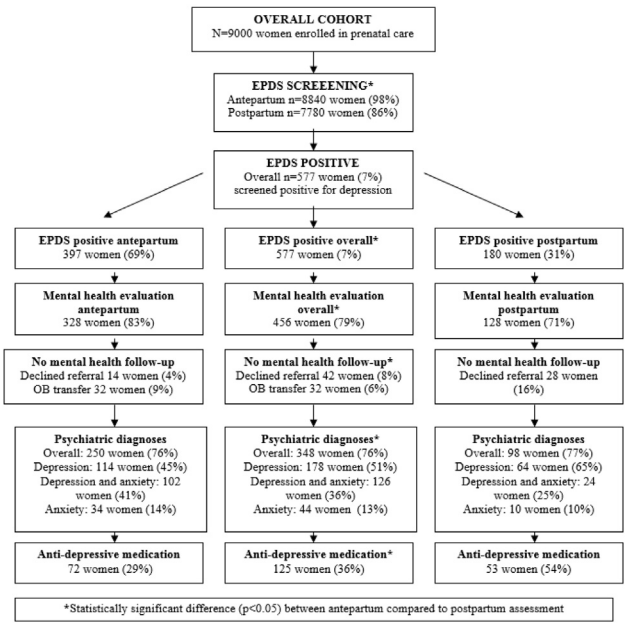
OBJECTIVE: To assess the feasibility of large-scale implementation of universal screening for depression in pregnancy and postpartum using the Edinburgh Postnatal Depression Scale (EPDS).

STUDY DESIGN: Prospective observational cohort study from July 2010 to June 2014 at a large academic medical center. Pregnant women were screened at 28 weeks gestation and again 6 weeks postpartum. An EPDS score≥12 was designated as the cutoff for referral to mental health services for diagnostic evaluation and treatment.

RESULTS: Among 9000 women enrolled in prenatal care, 8840 women (98%) were screened for depression antepartum, of whom 7780 (88%) were screened again postpartum. A total of 577 women (6.5%) screened positive for probable depression: 69% screened positive antepartum and 31% postpartum (p<0.01). All women who screened positive were referred for an evaluation by a mental health professional; 79% of women were evaluated, which was slightly more likely antepartum than postpartum (83% vs. 71%, p<0.01). The majority of women who were not evaluated further either declined a mental health evaluation (33%) or transferred obstetric care (25%). Half of those evaluated were diagnosed with major depression (51%), 36% concurrently with major depression and anxiety, and 13% with a primary anxiety disorder. Thirty-six percent of diagnosed women initiated an antidepressant medication, more commonly postpartum than antepartum (54% vs. 29%; <0.001). Using an EPDS cut-off≥12 antenatally to assess the accuracy in predicting postpartum depressive symptoms, the area under the receiver operating characteristics curve (AUC) was not sufficiently accurate (0.73).

CONCLUSION: This study demonstrates the feasibility of universal depression screening both antepartum and postpartum using the EPDS as an initial screen followed by mental health referral for further diagnostic evaluation and treatment. The population of women who screened positive as well as the acceptance of additional services differed at the two time points, reinforcing the utility of screening both antepartum and postpartum. While universal screening for depression is feasible, further study of the barriers to mental health evaluation and treatment as well as the impact of treatment on obstetric outcomes are needed.

Figure 1. Flowchart of women screened for depression overall and stratified antepartum compared to postpartum (N=9000)



210 Maternal deaths from suicide and drug overdose in Colorado

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OBJECTIVE: Our objective was to ascertain demographic and clinical characteristics of maternal deaths from suicide or drug overdose in Colorado to identify opportunities for prevention.

STUDY DESIGN: We identified maternal deaths (death during pregnancy or within 1 year postpartum) resultant of suicide or drug