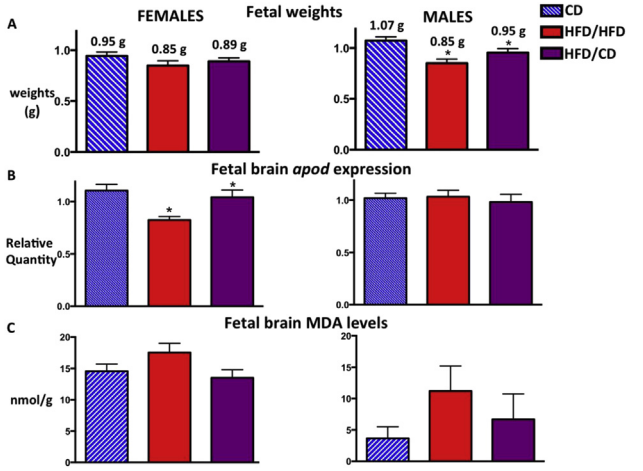
exposed to MATOB (HFD/HFD and HFD/CD) had significantly higher MDA levels than Ms (p=0.02, Fig 1C).

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.

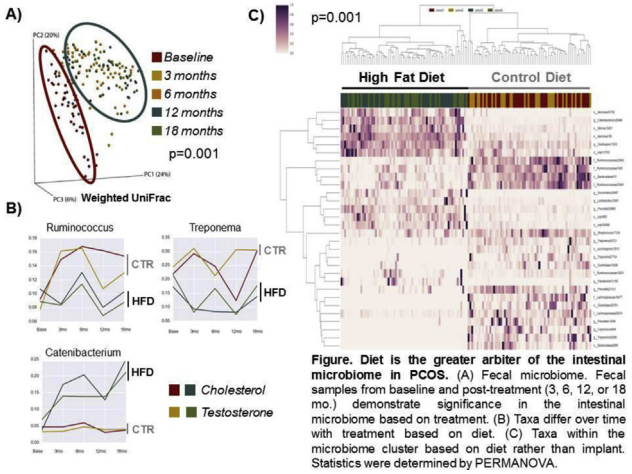

Figure. Diet is the greater arbiter of the intestinal microbiome in PCOS. (A) Fecal microbiome. Fecal samples from baseline and post-treatment (3, 6, 12, or 18 mo.) demonstrate significance in the intestinal microbiome based on treatment. (B) Taxa differ over time with treatment based on diet. (C) Taxa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