of pro-inflammatory cytokine IL-8. Our research also showed that addition of IL-8 (1 ng/ml) to melanoma cells stimulated cell growth (117%) and suppression of IL-8 by curcumin (100 μM) pre-treatment suppressed human melanoma cell growth (26%) in-vitro. This observation prompted us to check the effect of male sex hormones androstenedione (AD) and testosterone (T) on melanoma cell growth. AD and T also suppressed cell growth and IL-8 secretion, but not as significantly as that of progesterone. However, addition of progesterone (10 μM) along with androgens showed an additive effect on the inhibition of melanoma cell growth and suppression of IL-8 secretion. As steroids (P, AD, T) targeted IL-8 for their action, it was decided to check whether vitamin-D3 also targeted IL-8 secretion and cell growth. Active form of vit-D3 (25 μM) also suppressed IL-8 secretion and cell growth. But, addition of progesterone (50 μM) along with D3 significantly suppressed cell growth and IL-8 secretion. This brought IL-8 into focus as a key molecule regulating melanoma cell growth. In order to check whether IL-8 was *the molecule* involved in regulating melanoma cell growth, IL-8 rescue experiment after curcumin (25 μM) pre-treatment was carried out. IL-8 (100 ng/ml) was able to rescue cell growth completely after pre-treatment with curcumin, suggesting IL-8 was the molecule involved in regulating melanoma cell growth. Literature also suggested important role for IL-8 in regulating melanoma cell growth. Conditional expression of IL-8 in nude mouse by Dr. Singh et al., indicated in-vivo role of IL-8 in melanoma growth and metastasis. **Conclusion:** Both, in-vitro and in-vivo studies suggested an important role for IL-8 in regulating melanoma growth and metastasis. So, IL-8 could be targeted to arrest melanoma growth and metastasis in-vivo. *Hence, IL-8 could be a potential target for melanoma treatment.*

Reproductive Endocrinology

FEMALE REPRODUCTION: BASIC MECHANISMS

Ovulation Induction Results in Altered Growth and Metabolic Dysfunction in Mice Offspring

Royce Harner, BA[1], Saul Lira Albarran, MD[1], Celine Chalas, PhD[1], Annemarie Donjacour, PhD[1], Xiaowei Liu, MD[2], Paolo Rinaudo, MD PhD[3].
[1]UCSF, San Francisco, CA, USA, [2]University of CA-San Francisco, San Francisco, CA, USA, [3]University of California San Francisco, San Francisco, CA, USA.

MON-LB001

Nearly 15% of couples are affected by infertility and a large proportion of individuals will need to use ovulation induction (OI) or assisted reproductive technologies (ARTs) to conceive. Previous studies have shown that offspring conceived by ARTs are predisposed towards increased insulin resistance and glucose intolerance. However, the long-term effects of OI alone on offspring health have not been studied. This rodent study was designed to elucidate the effects of maternal superovulation on offspring growth and development.
C57Bl/6 females were either naturally mated (control= C) or super-ovulated (5 IU PMSG; 5IU hCG, OI group) and mated to C57Bl/6 males with one Agouti viable yellow (Avy) allelic mutation. The Avy allele contains an intracisternal A particle whose methylation levels determine expression of the agouti protein which alters coat color and can be used as a phenotypic readout for global methylation. Offspring (n= 108 control and n = 69 OI) were followed through 13 weeks of age to measure birth parameters, growth rate, fasting glucose, GTT, and body composition (EchoMRI). Parametric and non-parametric tests were used as indicated. Only results with $p<0.05$ are reported.
Results: Surprisingly, while litter size was not different (C = 7, OI = 6), superovulated mothers had fewer surviving pups (C=6.5 pups, OI=5 pups). No major differences in coat color frequencies were observed between the two groups, suggesting no changes in DNA methylation. All OI pups had decreased anogenital distance (males C = 2.1mm, OI = 1.7mm; females C = 1.74mm, OI = 1.48mm), while OI female had lower birthweights (C = 1.38g and OI = 1.23g). Starting at four weeks of age, OI male had lower weight compared to control males. As early as 3 weeks, significant differences in fasting glucose levels were noted (C = 162 mg/dL, OI = 149.5mg/dL). Additionally, superovulated males had lower lean mass at 8 weeks of age (tested by EchoMRI: C = 23.6g, OI = 19.3g) and higher insulin levels at 13 weeks (120 min post injection, C = 339 mg/dL, OI = 213 mg/dL). In summary, we found that the process of OI alone has profound effects on offspring development in a sexual dimorphic fashion. Additional studies will be performed up to 30 weeks of age. Funding: R01 HD092267-01to P

Genetics and Development (including Gene Regulation)

ENDOCRINE DISRUPTING CHEMICALS

Examination of Hepatic Gene Expression Following Developmental Exposure to Dieldrin in Trachemys Scripta and Discovery of a Novel Hepacivirus

Mara H. O'Brien, PhD[1], Ian P. Callard, BSC,PhD[2].
[1]University of Connecticut Health Center, Farmington, CT, USA, [2]Boston University, Boston, MA, USA.

SAT-LB131

The Massachusetts Military Reservation (MMR) is a Superfund site where ground water has been contaminated by a mixture of pollutants. Exposure to these chemicals is a public health concern and reproductive impairments have been observed in a population of turtles (*Chrysemys picta*) endemic to this site. We hypothesize that developmental exposure to endocrine disrupting compounds originating from the MMR might lead to abnormalities seen in adult animals. Upon examination of egg yolk from turtles at the impacted site, we found the presence of dieldrin and *p,p'*-DDE. Turtles from a reference site were also found to have *p,p'*-DDE present in the yolk. In order to investigate these chemicals in the laboratory we used a closely related turtle (*Trachemys scripta*) and applied vehicle, dieldrin, or *p,p'*-DDE to the eggshells. Absorption of *p,p'*-DDE through the eggshell was limited. Although there were variations in absorbance, we were able to achieve levels of dieldrin in the yolk similar to what was seen in animals from the impacted site. Following *in ovo* exposure to dieldrin, we used RNAseq to examine hepatic gene expression in neonates and found that several transcripts were repressed at least 1.5-fold in the dieldrin-treated animals. QPCR was carried out to