Addressing the role of cellular senescence in female fertility

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Introduction
Age-related decline in female fertility is characterized by a reduction in oocyte quality and quantity. Although several factors can contribute to this downfall, ovarian oxidative microenvironment, tissue inflammation and fibrosis have received increased attention. Additionally, the role of cellular senescence is still being unraveled. Therefore, we hypothesized that ovarian redox imbalance and cellular senescence contribute to the age-related loss of fertility.

Aim
1. Identify the presence of senescent cells in the ovaries of reproductively-aged mice
2. Evaluate the effect of redox imbalance in ovarian morphology and mice fertility

Experimental Design
Reproductively young C57BL/6J Mice (3 months old) n=3
Reproductively aged C57BL/6J Mice (9-10 months old)

Wildtype (Control) n=3 NRF2 KO mice n=4 HFE KO mice n=3 Hamp1 KO mice n=4

Results

Methodology
WT (C57BL/6J) mice, NRF2 and HFE KO mice and Hamp1 KO mice

Histochemistry Techniques
- Hematoxylin – Eosin
- Follicle Number & Ovarian Morphology
- Picrosirius Red
- Fibrosis (Collagen)
- Sudan Black
- Lipofuscin deposition
- Perls’
- Iron deposition

Fertility Decline Evaluation
- Number of litters
- Pups/litter

Number of follicles at ovary mid-section was obtained by calculating the mean counts of three to four ovary mid-sections. Collagen, Lipofuscin and iron deposition were quantified using the Image J software with blind intervention of two operators. A minimum of three to four sections of the mid-ovary was examined. A t-test was performed between the Old Wild-type (Old Control - OCT) mean and each mean of the other groups to determine the statistical significance.

Conclusions
Decreased follicle pool correlates with increased SB staining. Further studies are needed to understand the mechanisms behind increased induction of cellular senescence in WT animals.