Effects of Heat Stress on Ovarian Physiology in Growing Pigs

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Summary and Implications

Ovaries were obtained from growing pigs that had been heat-stressed and were evaluated for alterations in a signaling pathway known to play a critical role in ovarian physiology. Our results indicate that hyperthermia alters this pathway in a short space of time (after 7 days). Identifying how and why heat stress alters ovarian physiology are important in developing therapeutic approaches to prevent the reduction in reproductive performance associated with warm summer months.

Introduction

The US swine industry suffers major economic losses to heat stress during the summer months. Reasons for this fiscal hardship include seasonal infertility and poor sow performance during and immediately following the hot annual seasons. It is likely that the negative effects of heat stress will become more severe if climate change continues as some predict and most models forecast more extreme summer conditions in US pig-producing areas. In addition, genetic selection based upon rapid muscle growth is thought to increase pig sensitivity to heat stress. Heat-stressed pigs have unexplainably increased circulating insulin levels and we believe this detrimentally influences specific components necessary for successful reproduction. **Our central hypothesis was that heat-induced elevated insulin impairs steroidogenesis, oocyte recruitment and oocyte health and is a contributing mechanism to seasonal infertility**. This hypothesis was addressed by *determination of the impact of impaired insulin signaling caused by heat stress on the phosphatidylinositol-3 kinase signaling pathway*.

Materials and Methods

Pigs were exposed to thermal neutral conditions (TN) or 38°C (humidity: 25-35%; HS) for 7 d at the Iowa State University Swine Nutrition Farm. Ovaries were obtained immediately after slaughter, and treated as appropriate for further analysis.

Results and Discussion

Pigs exposed to HS conditions had decreased pathway ($P < 0.05$) action of the phosphatidylinositol-3 kinase signaling at the mRNA level (Figure 1). Additionally, use of immunofluorescence staining for a proxy measurement of the PI3K pathway, pAKT, indicated elevated protein levels following heat stress (Figure 2). The findings are significant since this pathway negatively regulates how quickly follicles are recruited from the resting pool, thus increased level of this protein indicates that this recruitment is being impaired due to increased thermal load.

Collectively, this preliminary data suggest that heat stress may alter follicle recruitment and development in pigs during the warm summer months, which may lag into the early fall months. Additionally, alterations in this pathway due to heat stress may change the level of ovarian hormones that are required for pregnancy maintenance.

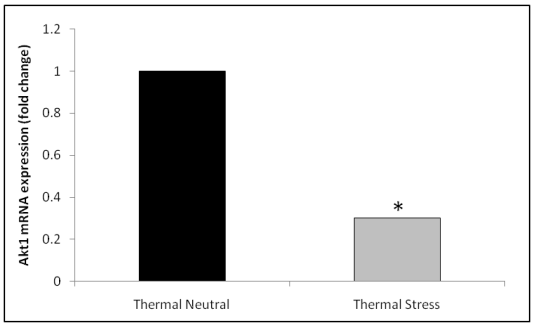

Figure 1. Effect of thermal stress on ovarian Akt1 expression. RNA was isolated from thermal neutral or thermal stressed gilt ovaries (n = 3/treatment) and real-time quantitative RT-PCR used to compare levels of Akt1 mRNA. Unpaired t-test was performed comparing differences in cycle numbers, relative to a house keeping gene, 18S rRNA. Data presented represents fold-change in thermally stressed ovaries relative to thermal neutral ovaries. *indicates different from control; P < 0.05.

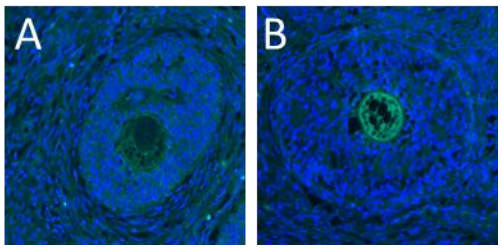

Figure 2. Effect of thermal stress on ovarian pAKT protein. Thermal neutral (A) or thermal stressed (B) gilt ovaries (n = 3/treatment) were sectioned onto slides and immunoflourescence staining to detect pAKT protein (green stain) performed. DNA in nuclei was co-stained using hoechst (blue stain). Increased pAKT was evident in ovaries from thermally stressed gilts.

Acknowledgments

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