In Wyoming, 27.8% of adults and 10.7% of children are obese. By 2018, medical expenditures for obesity-related health issues in the state are predicted to cost $607 million per year. These statistics illustrate a growing obesity epidemic that will increase healthcare costs in Wyoming and the entire nation. To combat the growing epidemic, the Centers for Disease Control and Prevention recommends increasing the initiation and duration of breastfeeding. Breastfeeding decreases obesity rates and the onset of Type 2 diabetes in the mother and infant. Unfortunately, mothers who are obese or overweight are more likely to terminate breastfeeding prematurely due to inadequate breast milk production. This problem is directly related to the hormone prolactin, which normally stimulates milk production in breast cells. Obese mothers, however, have a blunted prolactin response early postpartum, which delays the onset of copious breast milk production. Scientific understanding of how prolactin controls lactation is improving, but we do not currently understand how obesity alters prolactin production by lactotrope cells in the anterior pituitary gland.

**Objectives**

The goal is to determine how maternal obesity effects prolactin production and lactation.

**Materials and Methods**

Experiments are being conducted in the University of Wyoming Biological Sciences Building. One group of mice is receiving a control diet (10% calories from fat), while the second group receives a high fat diet (60% calories from fat). Blood and pituitary and mammary glands from both groups were collected during lactation to examine changes in prolactin and milk production. All animals are housed and cared for following approved UW Institutional Animal Care and Use Committee guidelines.

**Results and Discussion**

Normally, prolactin levels rise dramatically at the end of pregnancy to initiate lactation. Prolactin is critical to activate expression of lactation-related genes, which produce breast milk proteins and enzymes. Obese mothers have a blunted prolactin response early postpartum, which delays milk production. To investigate

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*Figure 1. A high fat diet induces obesity. Mice were fed a control or high fat diet for eight weeks and weighed weekly. Means were separated by one way ANOVA (p<0.05).*

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this problem at the cellular level, we have generated obese mice. Mice fed a high fat diet for eight weeks have approximately a 20% increase in body weight compared to mice fed the control diet (Figure 1). Next, obese and normal-weight female mice were bred. On lactation day two (L2) and nine (L9), we collected blood and pituitary and mammary glands from obese and normal-weight mothers. Our results indicate that normal-weight mice have an increase in pituitary prolactin mRNA production from L2 to L9, which facilitates normal milk production. In contrast, obese mice do not have an increase in pituitary prolactin mRNA production from L2 to L9, which may negatively affect milk production (Figure 2). Important studies are currently underway to corroborate mRNA data with prolactin protein levels in pituitaries and blood from control and high fat diet mothers. Our preliminary data suggest a correlation between obesity and the synthesis of prolactin, which warrants further investigation.

It is well established that a major complication of obesity is insulin resistance in metabolic tissues, which results in elevated circulating insulin, termed hyperinsulinemia. Our upcoming studies will investigate if obesity-induced hyperinsulinemia causes the decrease in prolactin production by the anterior pituitary gland lactotrope cells. We are using a weigh-suckle-weigh paradigm to examine if maternal obesity affects fetal weight. Lastly, changes in milk protein and fat levels will be examined in obese versus non-obese mothers. Our overall goal is to examine how obesity induces changes in the initiation of lactation and milk composition. We believe that our results will provide a scientific rational to promote breastfeeding to combat obesity.

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