SERUM INSULIN, GROWTH HORMONE, IGF-1, PROLACTIN AND LH IN RAM LAMBS TREATED WITH GLUCOSE

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(Key Words: Sheep, Glucose, Reproduction)

Ten, 6-month-old Debouillet ram lambs (avg BW 125.7 lbs) were assigned to one of two treatments to examine effects of glucose on endocrine profiles. Rams were allowed ad libitum access to a pelleted alfalfa diet (17% CP) and were fed corn at 1.5 lbs/day throughout the experiment. Five rams received (i.p.) 50 g Glucose (100 mL 50% dextrose) and five rams received (i.p.) 100 ml 0.9% saline (0 glucose, control) daily for 10 consecutive days. One hour after treatment on the last day, rams received 50 μg GnRH and serum samples were collected every 15 minutes for 7 hours (jugular venipuncture). Animals were weighed one day before and 13 days after the first treatment. A tendency was noted for decreased weight in glucose-treated rams apparently because feed intake was depressed. Exogenous glucose tended to suppress growth hormone and IGF-1 concentrations. Mean serum prolactin (PRL) concentrations in both groups were higher than those reported for adult rams which supports the concept that PRL concentrations are higher at the beginning of rapid testicular growth in young growing animals. Over the 7-hour period, serum luteinizing hormone concentrations were similar. Exogenous glucose resulted in a rapid and sustained increase in serum insulin as early as 30 minutes after a 50 g i.p. injection of glucose, but major effects on growth hormone, IGF-1 and prolactin were not detected. Likewise, injected glucose and the resultant elevated insulin failed to influence pituitary release of LH in response to a GnRH challenge.

EFFECTS OF BY-PASS PROTEIN ON PUBERTY IN BEEF HEIFERS

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(Key Words: Nutrition, By-Pass Protein, Heifer)

Undernutrition depresses reproductive function in all mammalian females. Nutrient intake is a key modulator for attainment of puberty in females, however the mechanisms by which nutrition influences the onset of puberty are not well understood. Current research is directed toward developing supplements which elicit a biological response which decreases days to puberty.

Sixty-seven pre-puberal beef heifers will be utilized in an attempt to determine which supplemental treatment accelerates the onset of puberty. Additional measurements to be taken include carry over effects of supplementation on conception rates, milk production, and weaning weights. Supplemental feeding groups consist of heifers receiving five pounds of one of three treatments three times weekly. Treatments consist of control, by-pass protein, or by-pass protein plus fat supplements. By-pass proteins have been shown to positively effect reproduction possibly through increasing secretion of follicle stimulating hormone (FSH) and/or insulin. Fat supplementation in ruminant females has been demonstrated to positively effect reproductive function by increasing serum cholesterol concentrations which in turn increases serum concentration of progesterone.

Data collected will focus on skeletal measures, age at puberty, ovarian dynamics, and serum metabolic hormones. Ultrasonography will be used for determination of back fat thickness, udder fat, and ovarian activity. Serum cholesterol and hormone concentrations will be measured every three weeks by blood sampling. Skeletal measures will include body weight change and pelvic area. Body condition scores will also be assigned.

The first year of this multi-year trial is currently in progress at the NMSU Range and Livestock Research Center, Corona, NM.

GENOMIC CONTROL OF FOLLICULAR ATRESIA

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(Key Words: Apoptosis, Follicular atresia)

Greater than 99% of ovarian follicles present at birth never ovulate (or undergo atresia). Until recently, mechanism(s) responsible for atresia had not been identified. Apoptosis, or programmed cell death, is now described as a possible mechanism responsible for atresia. Several "death genes" have been isolated and proven to be responsible for the apoptotic death related to follicular atresia. These genes include bax, bcl-x short and ice. In order to manipulate these genes and either rescue atretic follicles or initiate their demise, the trigger mechanism must be identified. Preliminary research suggests that oxidative stress may be involved in cell death and specific superoxide dismutases (SOD) have been identified. This research will attempt, initially, to determine whether SOD's are responsible for programmed cell death and mechanisms that control their presence.

The results of this research could lead to either a method of rescuing follicles from atresia or perhaps a reversible non-invasive form of contraception.