compared with CON males ($P \leq 0.001$). In OVER, 66 and 80% of DMR were hypermethylated in females and males compared with CON females and males, respectively ($P \leq 0.001$). These data demonstrate that maternal nutrition affects the DNA methylation patterns in the pancreas tissue of offspring in a diet-specific manner and that the changes in pancreatic DNA methylation are gender dependent.

Key Words: DNA methylation, maternal nutrition, pancreas
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307 Effect of maternal melatonin supplementation during mid to late gestation on fatty acid composition in maternal and fetal plasma and perirenal adipose tissue collected from bovine fetuses at 240 days of gestation.

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Melatonin supplementation during mid to late gestation increases uterine blood flow, thereby altering the flux of nutrients delivered to the developing fetus by the dam. Changes in the amount and composition of fatty acids available to the fetus can alter the long-term growth and developmental potential of the offspring in postnatal life. The objective of this study was to determine the effect of supplementing melatonin to beef heifers during mid to late gestation on fatty acid composition of maternal, umbilical, and fetal plasma as well as fetal perirenal adipose tissue. A total of 32 pregnant heifers were treated with (MEL) or without (CON) two 24-mg melatonin implants every 30 d starting on d 180 and ending on d 240 of gestation. On d 240 of gestation (approximately 85% of gestation), 6 CON and 6 MEL heifers were randomly selected to undergo Cesarean sections to collect fetal blood and tissues. Maternal blood (MB) was collected from the tail vein of the dams immediately prior to the surgery. Before excising the fetus, the umbilical cord was clamped on the fetal and maternal ends to collect blood from the umbilical artery (UA) and umbilical vein (UV). Fetal peripheral blood (FB) was collected during exsanguination, and perirenal (PR) adipose tissue was dissected from the fetal kidney following evisceration. Plasma and PR adipose tissue samples were directly derivatized for fatty acid quantification on a gas chromatography system to determine fatty acid concentration and percentage by internal calibration. Data were analyzed using the GLIMMIX procedure of SAS 9.4 (SAS Inst. Inc., Cary, NC), and statistical significance was determined at $P \leq 0.05$. There was no difference in total plasma fatty acid concentrations between the CON and MEL groups in MB, UA, UV, FB or PR ($P > 0.324$). Total SFA, MUFA, and PUFA in UA, UV, MB, FB, or PR also did not differ between treatments ($P \geq 0.11$). However, MEL tended to increase C22:6 fatty acid concentration in MB ($P = 0.065$), UV ($P = 0.079$), and FB ($P = 0.068$). Additionally, there was a tendency for increased ($P = 0.080$) C20:5*n*-3 fatty acid in PR adipose tissue in fetuses from MEL-treated dams. Both C22:6 and C20:5*n*-3 fatty acids improve fetal development and immune function. Therefore, additional research is warranted to determine the specific effect of MEL on these fatty acids and their long-term impacts on offspring growth and physiology.

Key Words: fatty acids, melatonin, perirenal adipose tissue
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308 Fetal brown fat deposition is increased by melatonin implants in sheep.

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The major cause of lamb mortality in grazing systems at birth is starvation and/or exposure to cold complex, with hypothermia being a primary contributing factor. In precocial species such as sheep, the ability to produce the heat required to prevent hypothermia during the first 12 h after birth relies heavily on the nonshivering thermogenesis, via brown adipose tissue (BAT). Therefore, insufficient BAT deposits, or inability to metabolize BAT, are the major factors contributing to lamb death resulting from starvation/exposure. Circulating concentration of melatonin is associated with proper amount and functionality of BAT. However, there is limited information on the effect of increased melatonin levels, via maternal supplementation, on fetal BAT deposition. The objective was to establish the effect of melatonin implants (M) in single- and twin-bearing ewes, on fetal BAT deposits. Corriedale ewes were synchronized, superovulated, mated to Suffolk rams, and managed under commercial grazing conditions. Single (S)- and twin (T)-bearing ewes received 0 (M0), 1 (M1), or 2 (M2) commercial 18-mg melatonin implants (Regulin) at 100 d of gestation ($n = 8$ per group). Ewes were euthanized at d 140 of gestation, and total fetal perirenal fat (BAT) was excised and weighed. The effect of litter size (S vs. T), number of implants (M0, M1, or M2), and their interaction on fetal biometrics were analyzed using ANOVA. A rank × treatment interaction ($P = 0.002$) was observed for total BAT, where SM2 fetuses tended to have 18% more BAT compared with SM0 ($P = 0.1$) and SM1 fetuses ($P = 0.09$; 22.8 ± 1.6 vs. 19.3 ± 1.5 or 19.2 ± 1.5 g), whereas TM1 fetuses had approximately 35% more BAT compared with TM0 ($P = 0.0002$) and TM2 fetuses ($P = 0.0003$; 22.9 ± 1.1 vs. 17.1 ± 1.0 or 16.9

± 1.1 g). Single and twin M2 fetuses tended to be 5 to 8% heavier compared with single and twin M0 and M1 fetuses (4.1 ± 0.1, 3.8 ± 0.1, and 3.9 ± 0.1 kg, respectively; $P = 0.09$). In addition, M2 fetuses, compared with M0 and M1 fetuses, showed greater fetal thorax diameter (34.6 ± 0.4, 33.8 ± 0.3, and 33.9 ± 0.3 cm, respectively; $P = 0.047$) and presented a trend for increased crown–rump length (44.1 ± 0.5, 43.0 ± 0.4, and 42.6 ± 0.5 cm, respectively; $P = 0.056$). These results indicate that maternal melatonin implants from d 100 of gestation increases BAT deposition, especially in twin fetuses, and may increase BW. Both effects may have important implications for newborn lamb survival under commercial grazing conditions. Funded by CONICYT project number 11150998.

Key Words: brown adipose tissue, lamb survival, melatonin

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309 Changes in fetal muscle microRNA expression from exposure to ergot alkaloids in utero.

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MicroRNA (miRNA) are small noncoding RNA that help regulate gene expression and metabolic function. Some miRNA, called MyomiR, are expressed predominately in skeletal muscle and regulate myogenesis. The objective of this study was to identify miRNA in skeletal muscle of fetuses exposed to ergot alkaloids in utero. Thirty-six pregnant Suffolk ewes (78.24 kg ± 9.5) were randomly assigned to dietary treatments of endophyte-free tall fescue seed (E−; 0.0 μg ergovaline + ergovalinine/ewe per day) or endophyte-infected tall fescue seed (E+; 1,722 μg ergovaline + ergovalinine/ewe per day) at specific stages of gestation (d 35–85 or d 86–133) in a 2 × 2 factorial arrangement of treatments. Fetal and maternal necropsies were performed at d 133 of gestation. Semitendinosus (ST) muscle was removed from each fetus and immediately frozen in liquid nitrogen for storage at −80°C. Total cellular RNA was extracted using the *mir*Vana miRNA Isolation Kit (Ambion, Austin, TX). Quality analysis of RNA was performed using an Agilent 2100 Bioanalyzer, with a RNA integrity number threshold of 7.0. The translational control RNA from 3 fetuses per treatment from the ST was used for miRNA sequencing and data analysis (LC Sciences, Houston, TX). MicroRNA sequencing yielded 113,252,743 reads with 92,177,228 mappable to the ovine reference genome. Of the mappable reads, 27% were specific to the *Ovis aries* genome and 18% were specific to mammals. There were 4,242 unique miRNA identified by sequencing, which included 208 that were specific to the *Ovis aries* genome and 676 that were mammalian but novel to *Ovis aries*. Known MyomiR (miR-1, miR133a, miR133b, miR206, miR-208b, miR-486, and miR-499) in skeletal muscle were present in our samples but not ($P > 0.05$) differentially expressed due to treatment. miR-148b, miR-300-3p, miR-431-3p, miR-299-3p, and miR-541-5p were upregulated ($P < 0.05$) in E+/E+ compared with E−/E− fetal ST muscles. miR-652, miR-628, miR-2427, miR-22-3p, miR-8118-p5, miR-376d, and miR-677 were downregulated ($P < 0.05$) in E+/E+ versus E−/E−. Skeletal muscle miR-148b has been shown to reduce glucose uptake in response to insulin in humans. miR-541 promotes vascular smooth muscle cell proliferation. Exposure to ergot alkaloids in utero alters miRNA expression in fetal skeletal muscle.

Key Words: fescue, fetal muscle, MyomiR

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310 Genetics is the essential factor for the precocious puberty in Nellore heifers.

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There is a paradigm that Nellore heifers' reproductive life starts at ≥24 mo of age due to nutritional conditions of low-quality pasture. This study aimed to determine the genetics and nutrition effects on puberty attainment in Nellore (*Bos indicus*) heifers. Fifty-eight weaned heifers (174 ± 6 kg initial BW and 8 ± 1 mo of age) were assigned to 28 feedlot pens. Heifers were born from 4 sires: 2 were precocious (P; negative EPD to age at first calving; $n = 33$) and 2 were nonprecocious (NP; positive EPD to age at first calving; $n = 25$). Heifers of each EPD were randomly assigned to 2 nutritional strategies (high ADG [HG; 0.7 kg] or low ADG [LG; 0.3 kg], resulting in 4 treatments: heifers from P sires were submitted to either HG (PHG; $n = 17$) or LG (PLG; $n = 16$) and heifers from NP sires were submitted to either HG (NPHG; $n = 12$) or LG (NPLG; $n = 13$). The HG heifers were fed with 75% concentrate diet, whereas the LG heifers were given 93% forage in their diet. Blood samples were collected at 9, 14, 18, 24, and 28 mo of age for plasma IGF-I determination. Transrectal ultrasonography and progesterone concentration were assessed weekly to determine puberty onset. The proportion of heifers that attained puberty at 18, 26 and 30 mo of age and BW at puberty were assessed by GLIMMIX procedure using a binomial and normal option, respectively. The MIXED procedure, with repeated measure analysis, was used to assess the interaction between treatment and time in IGF-I concentration. There was a treatment effect ($P < 0.01$) in the percent of heifers that attained puberty by 18 (62, 0, 0, and 0% for PHG, PLG, NPHG, and NPLG, respectively), 24 (100, 6, 54, and 0% for PHG, PLG, NPHG, and NPLG, respectively) or 36 mo of age (100, 100, 100, and 38% for PHG, PLG, NPHG, and NPLG, respectively). The BW at puberty was 360, 340, 468, and 390 ± 15 kg for PHG, PLG, NPHG, and NPLG, respectively. Plasma IGF-I concentrations were higher in P heifers than NP cohorts in all ages and in the same ADG ($P < 0.01$, treatment × age interaction). In conclusion, EPD of age at first calving was the