**Characterization of *RTL1* imprinted gene expression in Large Offspring Syndrome**

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Misregulation of imprinted genes is linked to a fetal overgrowth condition in bovine and humans known as large offspring syndrome (LOS) and Beckwith-Wiedemann syndrome, respectively. Imprinted genes have parental-allele specificity of expression and are key for regulating fetal development in mammals. The paternally expressed *RTL1* mRNA has been found to be involved in fetal growth. Therefore, we hypothesized that fetuses with LOS have loss-of-imprinting in the *RTL1* locus. The goal of this project was to characterize *RTL1* expression in LOS. To do this, we designed genomic primers for *RTL1*'s single exon and performed polymerase chain reactions (PCR) followed by Sanger sequencing in order to identify DNA sequence polymorphisms that may be used to determine allele-specificity in *Bos taurus indicus* X *Bos taurus taurus* F1 LOS fetuses. We identified five single nucleotide polymorphisms (SNP) in RTL1's single exon. Sequencing results showed that the bull is heterozygous for all five SNPs and the DNA pool of fetuses contain both alleles at the five positions. The SNPs were a T/C in the position 21:65,779,646, an A/G in position 21:65,779,739, a G/A in position 21:65,780,358, an A/G in position 21:65,782,216 and a T/A in position 21:65,782,250. If the F1 hybrids inherit a different allele from each parent, then we would be able to identify loss-of-imprinting as biallelic expression of this gene. Alternatively, we could detect biallelic silencing, as that mechanism for loss-of-imprinting has been documented at this imprinted domain. Therefore, we will determine if biallelic expression occurs in F1 hybrid LOS individuals through Sanger sequencing of the amplified transcript. In addition, we will perform quantitative reverse transcriptase PCRs to determine if level of expression of this gene varies between LOS and control fetuses. Results will be presented.