Beckwith-Wiedemann syndrome (BWS) is a congenital overgrowth condition with increased likelihood to develop childhood tumors. Children conceived with the use of assisted reproductive technologies (ART) have an increased frequency to have BWS compared to naturally conceived individuals. In ruminants, the use of ART can induce a similar overgrowth condition that phenotypically recapitulates BWS, which is referred to as large offspring syndrome (LOS). It is believed that these two overgrowth conditions are the result of misregulation of a set of genes that are expressed only from the maternally- or paternally-inherited chromosomes. These genes are known as imprinted genes. In this dissertation, we demonstrate that multiple imprinted genes are misregulated in LOS, as in a subset of BWS. Further, we show that global misregulation of non-imprinted genes in addition to loss-of-imprinting characterizes LOS. Importantly, most of the genes with aberrant expression are not associated with differential DNA methylation, an epigenetic modification that can regulate gene expression. Our results lay the foundation to predict the occurrence of LOS and help understand the molecular mechanisms of these congenital overgrowth conditions.