Long non-coding RNA (IncRNA) has been shown to regulate expression of coding genes via epigenetic mechanisms. In the human genome, many IncRNAs cluster with imprinted genes, which are expressed monoallelically, due to constitutive silencing of one parental allele. We are studying a genomic locus containing DIRAS3, a maternally imprinted tumour suppressor gene whose function is abrogated in ovarian and breast cancers. This locus also contains a novel IncRNA, GNG12-AS1, which we have functionally characterized. In normal cell lines with imprinted DIRAS3 expression, the GNG12-AS1 gene is biallelically expressed, with a small minority of splice variants being monoallelic. In cancer cell lines with loss of DIRAS3 imprinting, GNG12-AS1 expression becomes monoallelic. Transcription of GNG12-AS1 attenuates transcription of the active DIRAS3 gene, while high levels of DIRAS3 transcription reciprocally inhibit GNG12-AS1 expression. Imprinted expression of DIRAS3 is positively regulated by CTCF binding at a differentially methylated region upstream of its promoter. GNG12-AS1 transcription levels do not seem to be directly related to CTCF binding but allele specific splicing and the allele ratio seems to be regulated by CTCF-cohesin. The GNG12-AS1/DIRAS3 locus is the first example of imprinted co-transcriptional splicing and a potential model system for the study of long range effects of CTCF-cohesin on splicing and allele exclusion.