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Title: Investigation of Neat1's mechanism of splicing factor regulation via detained intron levels

Abstract
Neat1 is long non-coding RNA (lncRNA) with tumor suppressive functions in PDAC. Previous work has shown that Neat1 deficiency leads to wide dysregulation of gene expression patterns in transformed fibroblasts, suggesting wide gene regulatory functions of Neat1. My preliminary analysis of transcriptomic data from Neat1 deficient fibroblasts has shown that splicing-associated genes are those most significantly upregulated in Neat1 deficient cells, and that this trend may be conserved in a human cancer cell line. Further, alternative splicing analysis of Neat1-deficient cells identifies an almost unidirectional decrease in detained introns, whose inclusion in transcripts negatively regulate gene expression. Strikingly, these detained intron alterations are enriched within genes encoding splicing factors. These findings suggest a conserved role for Neat1 in regulation of splicing factors and may hint at a potential mechanism by which Neat1 regulates splicing factor expression via detained intron levels.