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What transposable elements are differentially translated in lung cancer?

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Transposable element (TE) expression is generally silent in somatic tissues, due to significant genomic methylation and other redundant methods of silencing. Cancer tissues, however, exhibit a marked decrease in methylation throughout the genome, which can result in de-repression of transposable element transcription. Because of this phenomenon, TEs may be tumor-specific antigens for use as potential biomarkers and vaccine targets. Lung cancer in particular is in dire need of early screening tools and treatment; the five-year survival rate is 16%. Ideal biomarkers and vaccine targets for lung cancer would be proteins or polypeptides that can be recognized by the immune system. However, TEs are subject to multiple post-transcriptional silencing mechanisms, such that increased hypomethylation does not necessarily result in increased polypeptide expression. Although evidence of increased transposable element RNAs and proteins in cancer tissues (in particular LINE-1 and HERV-K) exist in the literature, translation of other retrotransposon-encoded intermediates and other repetitive transcripts has not been thoroughly investigated. To this end we selectively sequenced translated TE sequences in a conditional mouse model of lung cancer, using ribosomal profiling. Comparing ribosomal footprints of healthy wild-type and transgenic cancerous lung tissues allows us to identify differentially translated elements.

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