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Perspective

Somatic Mutations in Cancer Cells *via* DNA Sequencing

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DESCRIPTION

Cancer is a hereditary condition in which genomic abnormalities impact gene expression, protein activity, and signalling pathways, which are essential for cell survival and proliferation. Many computational methods have been developed to target genetic variants using a variety of somatic mutation characteristics, such as sequence context, occurrence frequency, and impacts of mutations on transcriptome. These methods are used to identify genetic variants that impart greater rate. The interaction between transcriptomic and genomic modifications is still important to recognise the patterns by which mutations manifest their effects to connect the patterns to potential biological interpretation. Some mutations cause premature stop codons and reduce the number of mRNA transcripts, while others alter the amino acid sequences of proteins to change how they function. To overcome this deficiency, mutation has an effect on a gene that currently exists before it extends to the expression of adjacent genes in the transcriptome, pathways, or networks. Analytical challenges arise from associations between mutations and the genes which they are associated with. Regulatory variants, promoter methylation, Copy Number Variation (CNV), and copy-neutral Loss Of Heterozygosity (LOH), among other mechanisms, are used to regulate the mRNA dosage in cancer cells, which in turn regulates the protein concentration. It is still unknown how Single Nucleotide Variants (SNVs) and small insertions and deletions (indels) identified only in cancer cells (i.e. somatic mutations) relate to gene expression. There are numerous scientific explanations for how somatic mutations affect the genes which already exist. Truncation mutations can cause mRNAs to carry premature stop codons, which are usually removed by a mechanism known as Nonsense-Mediated mRNA Decay (NMD). As a result, Truncation mutations would reduce the quantity of mRNA transcripts and protein products. However, stop-gain mutations may escape NMD if they occur outside of the NMD-target areas, such as within 50 bp of the transcripts' 3′ end. Missense mutations (MS), a significant subset of cancer-related mutations, generally alter the amino acid composition of proteins and have an impact on the functions of the proteins in which they are located (e.g. catalytic efficiency, receptor activity, phosphorylation). Lack of association between a candidate mutation and its coding genes does not necessarily exclude the possibility that the mutation is a factor, but it may also imply that other mechanisms may exist through which mutations exert their effects. This is important for the purpose of identifying genetic variants. The effects of mutations in DNA repair genes, which primarily result in the accumulation of somatic mutations in cancer cells. Whole-Exome Sequencing (WES) somatic mutations identified in 12 different cancer types using information produced by the Cancer Genome Atlas (TCGA) project. Mutations in the same group are considered to have comparable processes, grouping mutations based on these characteristics provides a logical technique to differentiate the patterns via which mutations impact transcriptional profile. Somatic mutations are associated to posttranscriptional modifications (measured by the Reverse Phase Protein Array (RPPA) technology, protein level) and transcriptional levels (measured by RNA-sequencing, mRNA level).

CONCLUSION

Each gene exhibits differential expression in mutant samples in contrast to WT samples based on mutation clusters or mutation types. The mutation has the most impact on transcriptional level among the three characteristics, particularly in tumour suppressor genes. In order to address the functional impact of somatic coding mutations in terms of their expression and protein products, the relationships between mutation characteristics and mRNA/protein expression levels in various cancer types have been identified. The most significant factor affecting the amount of gene expression was the type of mutation, and that mutation clusters that were linked to gene expression. When analysing somatic mutation data for functional ramifications, the mutation characteristics were crucial variables.

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