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Perspective

Epigenetic Biomarkers in Cancer Genome

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DESCRIPTION

The temporal dynamics of cancer evolution remain a mystery since it would be difficult to look at malignancies across time that has not been influenced by therapy. Because of this, the majority of our knowledge on the spread of malignancies is derived from observations made at a single point in time; the period when a malignancy is removed from the body and examined in a laboratory. Fortunately, the cancer genome provides a detailed but hidden record of the development of the disease due to ongoing mutations that clearly identify clonal lineages within the tumour. We stress the importance of both neutral and selective evolution in the genesis of cancer and describe how the genome of a tumour can be studied to establish the historical history of mutation and selection. We draw the conclusion that both slow and punctuated genomic evolution are consistent with punctuated phenotypic evolution after reviewing the evidence for punctuated evolution in cancer. We conclude that to map and predict evolutionary trajectories during carcinogenesis, a better knowledge of the relationship between genotype change and phenotype change is essential. How do tumours spread? This fundamental question is still challenging to address for the apparent reason that it is rarely practicable to observe tumour progression over time in both human beings and model systems. So we understand how malignancies form through historical deduction based on the composition of excised tumours. In other words, most of the data that contribute to our comprehension of the temporal process of cancer evolution was obtained at a single time point: the stage of the process when the tumour ends up on the specimen table. It turns out that this predicament is not as dreadful as it may sound because the tumour genome provides a secret but comprehensive record of a tumour's growth. Every time a cell divides, mistakes in DNA replication introduce new mutations into the genomes of the daughter cells. Limited fidelity is also present in the copying of epigenetic markers, such as DNA methylation. Many malignancies also exhibit a substantial incidence of larger-scale chromosomal or part-chromosomal deletions or amplifications Somatic Copy Number Changes (SCNAs) and other structural rearrangements. Because tumours are clonally produced, these naturally occurring (epi) genetic changes serve as a record of the ancestry of the tumor's cells. The first cancer cell's mutations will be present in every cell of the tumour, but later-arising subclones can be distinguished because they share a specific, distinct set of variants; as a result, the order of clone development can be inferred by comparing the sets of mutations found in various tumour cells. Phylogenetics methodologies used to study cancer are based on the logic of this type of analysis. If a certain type of mutation accumulates at a consistent rate, it is possible to estimate the relative period at which a lineage originated by counting how many mutations of that type is unique to that lineage. The term "molecular clock" refers to a constant mutation rate. If the rate at which the molecular clock "ticks" is known, the absolute time of an event—where time is measured in the number of cell divisions can also be calculated. These techniques have been used to study a range of malignancies and have revealed fresh information about the time and sequence of mutation accumulation.

Finding neutrality and selection in the cancer genome

Whatever the biological mechanism underlying clonal selection, it ultimately leads to the relative expansion of the chosen clone inside the tumour. In contrast to the "null" condition, where the genome develops without selection, the clonal expansion is manifested in the cancer's genome by a "over-representation" of the mutations in the selected clone. Therefore, in theory, detecting selection just requires identifying the distinctive "clonal outgrowths." A variety of bioinformatics algorithms have been developed to identify the "clusters" of mutations that occur often in tumour next-generation sequencing data. As the selected clone spreads, all of the numerous passenger mutations in the clone are carried along to higher frequency, making the selected clone visible against the milieu of unselected mutations in the tumour. It is important to note that the passenger mutations in the clone, rather than the drivers themselves, largely reveal the evolutionary dynamics of the clone. As a result, selection influences both driver and passenger mutations in the clone, however passenger mutations are often more illuminating because they are more prevalent. Since evolution is a blind process, there have been many "unsuccessful" mutations in a genome as large as the human genome for every "successful" driving mutation.

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