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Opinion Article

Tracking the Progression of Cancer over the Duration of the Disease

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DESCRIPTION

Even though it is impractical to examine malignancies throughout time that have not been affected by treatment, the temporal dynamics of cancer evolution continue to be a mystery. Because of this, the majority of our understanding of how malignancies spread comes from deductions made from a single moment in time: the point in a cancer's lifecycle when it is taken from the body and analysed in a lab. Fortunately, the cancer genome offers a rich but covert record of cancer progression thanks to continuous mutations that distinctively designate clonal lineages inside the tumour. In this review, we explain how the genome of a cancer can be examined to reveal the historical history of mutation and selection, and we highlight why both neutral and selective evolution are important in the development of cancer. We contend that good research into selection in cancer requires some comprehension of what selection is not like. Reviewing the evidence for punctuated evolution in cancer, we conclude that both slow and punctuated genome evolution are consistent with punctuated phenotypic evolution. We come to the conclusion that a better understanding of the connection between genotype change and phenotype change is crucial in order to map and predict evolutionary trajectories during carcinogenesis.

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. As a result, historical deduction based on the make-up of removed tumours is how we understand how tumours form. In other words, the majority of the information that informs our understanding of the temporal process of cancer evolution was gathered at a single time point: the point in the process when the tumour ends up on the specimen table. Fortunately, the tumour genome has made this situation not as bad as it may seem gives a rich, covert record of a tumor's development. Errors in DNA replication cause fresh mutations to be added to the daughter cells' genomes each time a cell divides. Additionally, epigenetic markers are only partially replicated. Somatic Copy Number Changes (SCNAs), other structural rearrangements, and larger-scale chromosomal or part-chromosomal losses or amplifications are also seen with notable frequency in several malignancies. Because tumours are clonally formed, all of the cells in the tumour will inherit the mutations in the first cancer cell. These naturally occurring (epi)-genetic abnormalities are what record the ancestry of the cells in the tumour. The order of clone development can be deduced by comparing the sets of mutations found in various tumour cells, whereas later-arising subclones are distinguishable by their sharing of a specific unique collection of variants. Phylogenetics methodologies used to study cancer are based on the logic of this type of analysis. Additionally, if a given mutation type occurs frequently (for instance, the same amount of mutations are added throughout each cell division; this appears to be the case with C>T transitions inside particular three-base pair motifs, for example). The relative time that the lineage developed can therefore be estimated by counting the amount of mutations of the type that are specific to a given 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 that have passed), may 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.

Evolutionary selection also plays a significant influence in altering the cancer genome, in addition to mutation. When a set of cells within a tumour is evolutionarily "favoured" over another, selection refers to the circumstance in which the "favoured" cells produce more offspring than the "unfavoured" cells. The favouring is the result of the cell developing a novel adaptive phenotypic characteristic that gives it a competitive edge in the tumor's present microenvironment (context). When two cells are present in a microenvironment that is deficient in nutrients, the low metabolic demand cell may develop more quickly than the high metabolic demand cell. Positively selected clones (favoured) become substantially less prevalent as a result of selection, whereas any mutation in the selected (favoured) population becomes more common in the tumour population as a whole. As a result, selection is crucial in determining how frequently different mutations occur inside a tumour.

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