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Opinion Article

Genomic Instability in Cancer and Tumorigenes

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DESCRIPTION

Genomic instability has the potential to start cancer, speed up its development, and impact the patient's prognosis as a whole. Genomic instability can be perpetual or limiting through the induction of mutations or aneuploidy, both of which are both enabling and catastrophic. It can be caused by a variety of pathways, including telomere damage, centrosome amplification, epigenetic modifications, DNA damage from endogenous and exogenous sources, and DNA damage from epigenetic modifications. Numerous cancer treatments cause DNA damage, which slows down cell division over time a global level However, it is acknowledged that personalised therapies those that are catered to the specific patient and cancer must also be created. We describe the mechanisms by which genomic instability develops and can cause cancer in this review. We also discuss methods for treating and preventing genomic instability or exploiting the cellular flaws it causes. To combat genomic instability, we specifically identify and describe five key targets: (1) DNA damage avoidance, (2) DNA repair augmentation, (3) addressing DNA repair deficiencies, (4) centrosome clustering impairment, and (5) telomerase activity inhibition. As priority methods for battling genomic instability, we also call out vitamin D and B, selenium, carotenoids, PARP inhibitors, resveratrol, and isothiocyanates

Genomic instability-preventing or promoting cellular processes

Genomic instability is a key factor in the development and spread of cancer. Genetically, this instability can show up in a variety of ways, from straightforward changes in the Deoxyribonucleic Acid (DNA) sequence to chromosomal structural and numerical abnormalities. This section will briefly describe the mechanisms that keep nuclear and mitochondrial DNA stable as well as how these systems could be compromised in cancer cells. Telomeres support chromosomal stability and can prevent or stimulate the development of cancer and more recent research has strengthened the link between Chromosomal Instability (CIN) and telomere dysfunction. Each chromosome's ends, or telomeres, are made up of roughly 5–10 kbp of specialised, tandem repeat, noncoding DNA that is complexed with a number of telomere-associated proteins. These components form a shield that hinders the detection of the chromosomal termini as DNA Double Strand Breaks (DSBs) and the ensuing aberrant repair through Non Homologous End Joining (NHEJ) or Homologous Recombination (HR). Telomeric DNA is lost at a rate of about 100 base pairs (bp) per telomere every cell division because DNA polymerase is unable to fully replicate the ends of linear DNA molecules in the absence of compensatory measures. Normal somatic cells use this telomere erosion to track the history of their divisions, with moderate telomere shortening leading to either replicative senescence, an irreversible cell cycle arrest or apoptosis. This restriction on subsequent proliferation is assumed to have developed to stop the growth of altered cell clones unchecked, as well as to stop further telomere erosion that would follow such abnormal growth and eventually destabilise the telomeres resulting to CI.

The spindle assembly checkpoint, tumorigenes and centrosomes

The centrosome, which is made up of two centrioles and a cloud of proteins that encourage microtubule nucleation, is the main location in dividing mammalian cells where microtubules are organised. One daughter centriole is created next to an existing mother centriole during the semiconservative centrosome duplication process, which only happens once every cell cycle. Most solid and haematological cancers share the characteristic of centrosome amplification, which is the presence of more than two centrosomes during mitosis. This characteristic may cause multipolar mitoses, chromosome missegregation, and consequent genetic imbalances that encourage carcinogenesis. Numerous mechanisms, including as centrosome overduplication, de novo assembly, and mitotic failure following mono- or multipolar division, can result in centrosome amplification. Cytokinesis failure is frequently the final outcome of these structural anomalies, which can lead to tetraploid binucleated cells and genomic instability later on the end outcome over time is a limited population of cells with the capacity to manage extra centrosomes, which could explain the build-up of cancer cells with aneuploidy and centrosome amplification.

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