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Understanding Cancer Genetics: Unravelling the Complexity of Tumorigenesis

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INTRODUCTION

Cancer remains one of the most challenging health concerns of our time, affecting millions of lives worldwide each year. Despite advances in treatment and early detection, the intricate mechanisms underlying cancer development continue to elude complete understanding. In recent decades, however, significant strides have been made in the field of cancer genetics, illuminating the genetic basis of various cancers and paving the way for targeted therapies and personalized medicine approaches. This article delves into the complexities of cancer genetics, exploring the role of genetic mutations, oncogenes, tumour suppressor genes, and the promise of genomic medicine in the ongoing battle against cancer. Cancer arises from the uncontrolled growth and proliferation of cells, often triggered by genetic alterations that disrupt normal cellular functions. These alterations can be inherited (germline mutations) or acquired (somatic mutations) during an individual's lifetime due to environmental factors or spontaneous errors in DNA replication. Central to cancer genetics are oncogenes and tumour suppressor genes, which play opposing roles in regulating cell growth and division: Normally, proto-oncogenes regulate cell growth and differentiation. When mutated or activated, they become oncogenes, promoting uncontrolled cell division and tumour formation. Examples include HER2 in breast cancer and BRAF in melanoma. These genes normally inhibit cell division, repair DNA damage, and promote apoptosis (programmed cell death).

DESCRIPTION

Mutations that inactivate tumour suppressor genes, such as TP53 (p53), BRCA1, and BRCA2, can lead to unchecked cell growth and cancer development. Genetic mutations are fundamental in driving cancer initiation and progression. These mutations can occur in various genes involved in critical cellular processes. Mutations impairing DNA repair mechanisms (e.g., BRCA1/2) increase susceptibility to cancer, as damaged DNA accumulates, leading to further mutations and genomic instability. Alterations in genes controlling the cell cycle (e.g., cyclin-dependent kinases) can disrupt the orderly progression from one phase of the cell cycle to the next, promoting uncontrolled cell division. Some cancers have a strong hereditary component, where specific gene mutations increase the likelihood of developing certain types of cancer. Mutations in DNA mismatch repair genes (MLH1, MSH2, etc.) predispose individuals to colorectal and other cancers. Mutations in BRCA1 and BRCA2 significantly increase the risk of breast, ovarian, and other cancers. Recent advancements in genomic sequencing have revolutionized cancer research and clinical practice.

CONCLUSION

The era of precision medicine has emerged from our growing understanding of cancer genetics. By identifying specific genetic alterations driving individual tumours, targeted therapies can be developed to selectively inhibit oncogenic pathways. Target mutations in kinases involved in cell signalling pathways, such as EGFR inhibitors in lung cancer (e.g., gefitinib). Exploit deficiencies in DNA repair pathways (e.g., BRCA mutations) to selectively kill cancer cells, as seen in ovarian and breast cancers. Immunotherapy has revolutionized cancer treatment by harnessing the body's immune system to target cancer cells. Cancer genomics plays a crucial role in predicting patient response to immunotherapy. High TMB correlates with increased response to immune checkpoint inhibitors (e.g., PD-1/PD-L1 inhibitors) across various cancer types.

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CONFLICT OF INTEREST

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