Biomedical Research 2022; Special Issue: S1-S2

ISSN 0970-938X
www.biomedres.info

Oncogenes and their perception to cancer.

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Accepted on April 27, 2022

Description

Oncogenes are mutated genes that play a role in cancer formation. Everyone has genes that are known as proto-oncogenes when they are not altered. When proto-oncogenes are altered or amplification occurs as a result of DNA damage (such as carcinogen exposure), the proteins produced by these genes might affect the cell's development, proliferation, and survival, potentially leading to the creation of a malignant tumour. Scientific study has clearly proven the importance of oncogenes in human cancer over the last five decades. Many efforts have been undertaken to understand the causal function of activated oncogenes in cancer formation since their discovery in human tumours. All of this research has demonstrated that oncogene expression is essential not just for cancer development but also for disease maintenance, keeping oncogenes in the spotlight as important anti-cancer treatment targets. Tumors form often when oncogenic expression is induced by tissue-specific promoters in genetically modified mouse models, but they regress when the inducing stimulus is turned off, implying that oncogenes are cancer's Achilles' heel (allowing the body to target the disease). This novel cancer model is consistent with the fact that, in human malignancies, all malignant cells carry the same beginning oncogenic genetic mutations, regardless of cellular heterogeneity within the tumour [1].

Proto-oncogenes are regular genes in our bodies that assist regulate cell division, growth, and even death rates. This equilibrium is what keeps our bodies healthy and functioning properly. An instance of an oncogene is the HER2 gene, which creates the HER2 protein. This protein aids in the healthy division and development of breast cells. Extra copies of this gene may result in an overabundance of HER2 protein, causing cells to proliferate faster. Some breast cancer and ovarian cancer cells include the HER2 oncogene. The role of proto-oncogenes in normal cell growth and differentiation, as well as the idea that oncogene proteins could be used as new targets for cancer chemotherapy, are both current research topics that are direct outgrowths of Howard's important contributions to cancer research. Howard's concept for the origin of cancer genes (who had properly anticipated many parts of current understanding of the molecular modifications necessary for the formation of malignant tumours) has proven correct on all three major elements. To begin with, mutations are important in transforming proto oncogenes to oncogenes and inactivating tumour suppressor genes. Second, both strongly transforming retroviruses and non-virus induced malignancies, including human cancers, contain comparable oncogenes. Third, reverse transcription is responsible for the insertion of oncogenes into retroviral genomes as well as some proto-oncogene and tumour suppressor gene alterations in non-virus induced cancers. The discovery that oncogenes are mutated forms of normal cell genes (proto-oncogenes) drew attention to proto-oncogenes' roles in normal cells and the nature of the molecular changes that turn proto-oncogenes into oncogenes [2,3].

Completely unregulated expression of oncogenes and tumour-suppressor genes has been studied extensively in tumour growth for centuries. Oncogene expression and their possible function in immune cell abnormalities during carcinogenesis and tumour growth, however, have not even been adequately investigated [4]. Despite the fact that proto-oncogenes are expressed in all cells, including immune cells, abnormalities in proto-oncogenes have been thoroughly characterised and analysed mostly in tumour cells. In general, oncogene function during multistep carcinogenesis is thought to be based on a growth advantage afforded by the proto-oncogene product's altered function. The damaged cells are no longer under the supervision of a carefully regulated network of mitogenic and antimitogenic signals, which normally controls their proliferation. As a result, cells containing oncogenes grow preferentially and independently, contributing to the expansion of the pool of altered cells that are selected during carcinogenesis [5]. It's worth noting that the oncogenic function of a gene is typically determined using cellular model systems that involve primary or immortalised cell lines. In cellular model systems, the multistep aspect of tumour growth is ignored.

Conclusion

Discovering proto-oncogenes, oncogenes, and tumour suppressor genes is assisting researchers in better understanding both the processes that lead to cancer formation and progression, as well as cancer treatment techniques based on the specific impacts of oncogene products. The promise of developing novel therapeutic techniques for cancer treatment that target signalling pathways that are unique to tumour cells appears to be promising in the near future. The discovery of oncogenes and the understanding of their functions have clearly influenced the development of such techniques.

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