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Methodology Help in Advancement of Analysis and Treatment

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Abstract

Oncogenes are the tumour causing genes and have important role in development of many cancers. In 1970, SRC oncogene was discovered in chicken retrovirus. As a result of some mutation in the otherwise normal proto-oncogenes, their deregulation occurs and uncontrolled proliferation of cells starts and leads to cancer.

Keywords: Diagnosis; Cancer; Prevention; Scintigraphy; Health outcomes; Imaging

Introduction

At genomic level, only single oncogenic allele is required to alter normal gene function because of its dominant property. The origin of oncogene can be cellular i.e., from inside the body or viral i.e. from some virus. Gene Duplication, addition, insertion, deletion or chromosomal translocation, chromosomal re-arrangement of certain proto-oncogenes alters their function and converts them into oncogenes. These mutations overexpress the protein to an uncontrolled level, which may lead to tumour. These mutations may occur due to external factors or internal factors or both like viral infection, radiation or chemicals, injury and disease. Among these mutations, viral infection is the rare cause of oncogene activation in animals but is of great importance for understanding oncogene function [1]. Viral infection Retroviruses or DNA viruses cause viral infections. These viruses infect the host either by inserting oncogenes in host chromosome, interfering proto-oncogene transcription factors/regulators or by inserting homologous sequences corresponding to normal proto-oncogene of host. For example, retrovirus carrying SRC oncogenes infects the host, integrates viral chromosome in host chromosome, further divides the viral progeny and infects the surrounding cells, inducing overexpression of cellular normal genes and deregulated proliferation of cells in order to cause cancer. Types and classification of oncogenes, oncogenes can be classified into five classes based on protein products formed by mutation or deregulation of proto-oncogenes. These include growth factors, growth hormone/factor receptors, serine/threonine kinases, GTPase molecules and, transcription factors [2]. Mutations in the growth factors can lead to several types of cancers such as fibrosarcoma, glioblastoma, osteosarcoma, etc.

Discussion

In several tumors, ligand binding domain deletions of Epidermal Growth Factor Receptor cause successive activation of receptor even in absence of ligand by trans-membrane protein carrying tyrosine-kinase activity [3]. This activation causes interaction with further cytoplasmic proteins like SRC domain and leads to deregulation of numerous signalling pathways. Mostly in gastrointestinal, breast and lung cancers, GFR mutations occur. Similarly, overexpression of Raf-1 kinase and cyclin-dependent kinases due to uncontrolled phosphorylation may cause many cancers such as thyroid and ovarian cancer. Deregulated activation of GTPases such as Ras, causes activation of MAPK pathway and uncontrolled signalling and division of cells cause several cancers such as myeloid leukemia. Transcription factor proteins are products of proto-oncogenes. The mutation, translocation or re-arrangement of these causes overexpression of gene and unwanted consecutive transcription of target gene that leads to any types of cancers such as pancreatic and lung cancer. The oncogenes are targeted to treat oncogenic cancer. Several oncogenes discussed above are targeted by drugs and gene therapies to inhibit, arrest, regulate or senescence their genes. For example, Imatinib or Gleevec is used to treat BCR-ABL. Gefitinib or Iressa, erlotinib or Tarceva are used to target EGFR. VEGF oncogenes are targeted by bevacizumab or sorafenib. Sorafenib is also used to down regulate or inhibit B-Raf oncogene. These agents/drugs are used, sometimes in combination, for chemotherapy to inhibit proliferation of oncogenes or to down regulate signalling onco-proteins in several signalling pathways to treat oncogenic cancers. However, it is difficult to target non-kinase oncogenes through drugs such as Myc and Ras. Tumour suppressor genes in cancer Tumour suppressors play their role by inhibiting cellular proliferation and tumour development. In most of the tumors, inactivation of the tumour suppressor genes eliminates the negative regulation of these genes over cellular proliferation that leads to abnormal cell proliferation, therefore, causing cancer [4]. Tumour suppressor genes have “loss of function” mutations because they develop cancer by inactivating their inhibitory effect on cell proliferation. For a tumour suppressor gene to promote tumour development, both copies of the gene must be inactivated because onecopy is sufficient for controlling cell proliferation. These mutations act recessively. The protein products tumour suppressing genes are found to play the following important functions, Enzymes involved in DNA repair, Checkpoint-control proteins arresting the cell cycle in case DNA is impaired or chromosome abnormality, Proteins promoting programmed cell death, Inhibiting cellular growth and proliferation by acting as receptors for hormones. Intracellular proteins which regulate or inhibit movement through a specific stage of cell cycle [5].Some tumour-suppressing genes act as transcriptional regulatory proteins. For example, the product of WT1 gene which is a repressor protein and acts by suppressing transcription of many growth factor-inducible genes. WT1 is made inactive in Wilms tumors. Insulin-like growth factor II is the target of WT1 gene, over-expressed in Wilms tumors, thereby contributing to abnormal cell proliferation. Retinoblastoma and INK4 Genes Several tumour suppressor genes regulate cell cycle progression through a specific stage e.g. protein

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products of Rb and INK-4 genes. Retinoblastoma is the tumour of the eye. Two mutagenic events are required for the etinoblastoma development in sporadic cases whereas only one mutagenic event is needed in individuals with inherited form of the disease in which it displays autosomal dominant inheritance [6]. In normal cells, Cdk2 and cyclin D complexes regulate the entry through the constraint point, thereby phosphorylating and inactivating pRb. pRb also impedes the entry through the constraint point in the G1 phase of the cell cycle by repressing the transcription of many genes involved in cell cycle advancement. The INK4 tumour suppressor gene also regulates movement through the restriction point by encoding Cdk inhibitor p16. Inactivation of INK4 results in uncontrolled phosphorylation of Rb [7]. The p53 plays its role by regulating cell cycle and programmed cell death. The p53 can arrest the cell cycle upon DNA damage. It allows the DNA to repair or cause the programmed cell death. This is achieved by activating a number of genes involved in controlling and regulating the cell cycle. Mutation in p53 in tumorigenic cells results in uncontrolled cell proliferation and inefficient DNA repair. P53 mutations are estimated to be the most common in tumors of humans, approximately greater than that. Breast cancer and genes are linked to familial breast cancer. Breast Cancer-1 gene consists of 100 Kb DNA and 21 exons. It has a zinc-finger domain like that in the DNA binding proteins. Breast cancer-1 is a tumour suppressor gene. BRCA-2 is located on chromosome. Tumour suppressor genes can be studied at the levels of DNA, mRNA, and proteins in the normal and cancerous cells using various methods. Tests for the detection of heterozygosis can be helpful for identifying individuals predisposed to retinoblastoma and other malignancies. Higher frequency of p53 mutations also offers diagnostic and analytical possibilities. PCR amplification can be used to study the changes along with recent methods such as RNase protection assays, single-strand conformational polymorphism or denaturing gel electrophoresis [8]. Immuno-metric type as says are quite good at measuring altered p53 in tumour cell line lysates and tissue homogenates. One of the primary challenges in the clinical management of cancer patients is to establish the correct diagnosis. As a result, a number of technologies have been developed and are now routinely employed to subtype molecularly cancers. These include immunohistochemistry, immune-fluorescence, and the analysis of DNA and RNA extracted from the lesion- through In situ hybridization and fluorescent in situ hybridization. The cancer specimen is then subtyped using different approaches of molecular biology including Sanger sequencing, pyro-sequencing, allele-specific PCR. Cancer genotyping is performed by snapshot assay, mass spectroscopy based assays and next generation sequencing. The introduction of next-generation sequencing is serving to uncover the true diversity of cancers as well as to define recurring mutations targeted with new therapies [9]. Such genomic level analyses will continue to have an impact for many years. Different treatment techniques and therapies have been applied for the treatment of cancer at different times. Some of the most common methods used include surgery, radiation therapy, chemotherapy, hormonal therapy, immunotherapy, adjuvant therapy, targeted- growth signal inhibition, drugs that induce apoptosis, nanotechnology, RNA expression and profiling, and the latest being CRISPR. Few of these shall be later discussed in this review. Cancer cells can also be killed by gene replacements or by knocking out oncogenes. Oncolytic viruses can be used in combination with chemotherapeutic agents to destroy cancer cells as well. Apart from the conventional methods, retroviruses (RVs) have also been used in cancer therapy. RVs can be and have been used for transferring genes to mammalian cells. Most popular RVs are the ones derived from the Moloney Murine leukaemia virus. In the last twenty years, artificial evolution of RVs has enabled their applications in developing transgenic animals, stable delivery of siRNA and clinical trials forgene therapy. The great potential of RVs was discussed in recent reports about the successful clinical trials of gene therapy in patients with severe. However, there are some probable risks inherently related with that. A comparative experiment was done by designing two groups of vectors. One was intrinsically replicative, the other was defective and so it had a helper retrovirus with it. Under in vitro conditions, the replicative viruses achieved more than transduction while the other transduced only less than 1%. This experiment clearlyindicates the potential of RRVs for developing cancer gene therapy. Recently, interest in investigating retroviral vector insertions has been growing. For many years, viral insertion sites were used to identify possible oncogenes and cancer signalling pathways. The scope of this approach has been broadened by techniques likeinsertion site cloning by high throughput PCR, availability of genetically modified animals and with the completion of mouse genome project. However, numerous investigators have identified hundreds of common sites [10]. These integration sites are usually associated with cancer genes in MoMLV-induced murine haematopoietic malignancies. Generally, majority of the insertions exist outside the coding regions. Therefore, only less than that of the RISs can be considered as the accepted tumour suppressor genes.

Conclusion

In addition, a large, high-quality, head-to-head comparison of and MRI would be needed, especially for women at high-risk of breast cancer, because MRI, alternated with mammography, is currently the recommended screening technique.

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Conflict of Interest

None

References

1. Hamashima C, Shibuya D, Yamazaki H, Inoue K, Fukao A, et al. (2008) The Japanese guidelines for gastric cancer screening. Jpn J Clin Oncol UK 38:259-267.
2. Sabatino SA, White MC, Thompson TD (2015) Cancer screening test use: United States, 2013. MMWR US 64:464-468.
3. Vernon SW (1997) Participation in colorectal cancer screening: a review. J Natl Cancer Inst UK 89:1406-1422.
4. Brawley OW, Kramer BS (2005) Cancer screening in theory and in practice. J Clin Oncol US 23:293-300.
5. Warner E (2011) Breast-cancer screening. N Engl J Med US 365: 1025-1032.
6. Walsh JME, Terdiman JP (2003) Colorectal cancer screening: scientific review. JAMA US 289:1288-1296.
7. Secretan BL, Scoccianti C (2015). Breast-cancer screening—viewpoint of the IARC Working Group. N Engl J Med US 372:2353-2358.
8. Schwartz LM, Woloshin S, Fowler FJ, Welch HG (2004) Enthusiasm for Cancer Screening in the United States. JAMA US 291:71-78.
9. McKinney SM, Sieniek M, Godbole V, Godwin J (2020). International evaluation of an AI system for breast cancer screening. Nature 577:89-94.
10. Fontana RS, Sanderson DR, Woolner LB, Taylor WF, Miller WE, et al. (1986) Lung cancer screening: the Mayo program. J Occup Med US 28:746-750.