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A Short Note on Cancer Genetics

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Editorial

Oncogenes are also known as cellular genes that when mutated or potentially improperly communicated in a way that expands their action bring about a malignant phenotype. Traditional models incorporate src, ras and myc oncogenes. These genes are a lot of the vital parts of cell administrative cycles eg. The src quality is codes for a tyrosine kinase, the rash quality for a G protein and the myc quality for an atomic protein that is associated with DNA replication. Oncogenes were first found in intensely changing retroviruses (Rous Sarcoma Virus). When these infections taint deified however untransformed cells in culture they create a neoplastic phenotype. It was accordingly tracked down that these viral oncogenes were not normally happening viral qualities however got from the cell genome and hence changed or over communicated to create cell change. A single mutated oncogene can't change essential cells and the necessity for oncogene cooperatives is in concordance with the multi-step hypothesis of carcinogenesis got from traditional examinations. Oncogene co-operative typically requires collaboration between oncogenes having a place with various groupings eg. Atomic (eg myc with cytoplasmic eg rash) [1].

These genes can be compared to the brakes of acar, and function in the cell to manage cell division. Loss of genetic matter is additionally a vital occasion in the generation of neoplasia, and the equivalent can be exhibited by cytogenetic methods. Sub-atomic instruments have had the option to additionally characterize the deficiency of genetic matter [2]. Normally there is deficiency of one allele of a TSG while the other is inactivated by point change. The ideas of TSGs were shown first with the Retinoblastoma quality (RB). Ordinarily influenced TSGs incorporate the p53 quality (influenced in practically a large portion of the human malignancies) the Wilms tumour quality the p16gene etc [3].

These have also been called caretaker genes. Inactivation of such genes leads to genomic instability and thus markedly increases the probability of alterations in the oncogenes and the TSGs. DNA mismatch repair genes have been extensively studied and include the hMSH2 and hMLH1 genes which are commonly affected in human malignancies. Again as the case of most oncogenes and TSGs, homologues of such genes can be traced back to the yeasts indicating the fundamental similarity of these biological processes [4]. DNA mismatch repair defects manifest as unusually rapid expansion and contraction of microsatellite repeat sequences. Inherited defects in such genes are exemplified in Hereditary Non Polyposis Colon Cancer (HNPCC), where analysis of microsatellite repeats in leucocyte DNA forms a basis of diagnosing the affected siblings in a family. The affected individuals are subjected to regular investigations including colonoscopy. Increased genomic instability also includes several other aspects, the implications of which are under study [5]. These include aneuploidy including genetic loss and translocations, increased frequency of point mutations, other repeat mediated recombinations, increased tendency for gene amplification etc. The p53 gene which has been described as the guardian of the genome functions as both a caretaker gene and as a TSG. A similar role has been attributed to the Brca II gene. The instability of the cancer genome could contribute extensively to therapeutic resistance, which is perhaps the most frustrating aspect of tumour therapy [6].

The viruses shown to be extensively involved in human cancer are the Hepatitis Viruses (B and C) and the Human Papilloma Virus (HPV) which are involved in liver and cervical cancers respectively. Several other viruses (like Herpes viruses) have also been implicated from time to time. However there is not much hard evidence as yet for the involvement of acutely transforming retroviruses [7]. However Human T Cell Leukaemia Viruses I and II have been shown to be involved in outbreaks of T cell Leukemia especially in Japan. In terms of sheer numbers and morbidity and mortality, cervical and liver cancers are both very important. While there are a number of significant publications regarding the mechanistic basis of these cancers, it is important that these be treated as preventable cancers. The Hepatitis B vaccine is also an important vaccine for cancer prevention. Vaccines for HPV are at the experimental stages; however this knowledge could be useful for early diagnosis screening and behavioural intervention [8].

The peer-reviewed publications included in this review were extracted from PubMed and covered the period between January 1990 and December 2019, as shown in the flow chart in. Since PubMed Medical Subject Heading (MeSH) terms involve synonym control, it yields more precise and inclusive search results. Our literature search approach, therefore, utilized an integration of MeSH terms that incorporated "the disease" (neoplasm), 54 African countries, and combinations of study parameters ('gene or protein or molecular biology or mutation or genetics or genomics') [9]. After extracting African cancer papers, we next filtered those to include only papers pertaining to cancer molecular biology (protein or nucleic acid). Cancer molecular biology papers were then further filtered using "genetic* OR genomic* OR mutation*[MeSH Terms]." The final criteria were that the studies must utilize bio specimens of African origin. Two authors (SOR and OAR) manually verified these publications to ensure the accuracy of terms [10].

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