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The Role of Molecular Biology in Cancer Treatment: Advances, Applications, and Future Perspectives

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Abstract:

Cancer is a genetic disorder primarily caused by several mechanisms, including the activation of oncogenes, the breakdown of tumor suppressor genes, or mutagenesis resulting from external influences. During the latter part of the 20th century, our understanding of cancer biology has advanced remarkably. Currently, we comprehend cancer at the genetic and epigenomic levels, having identified the cell initiating neoplastic transformation and outlined the processes behind tissue invasion. The advancement of diagnostic instruments and therapies through a molecular biological framework enables the implementation of sensitive, noninvasive testing for cancer patients. This knowledge has allowed novel drugs to be designed that act on specific molecular targets, the immune system to be trained and manipulated to increase its efficiency, and ever more effective therapeutic strategies to be developed. The accurate diagnosis, prognosis, and prediction of cancer patients' responses to treatment are crucial for optimizing therapy, minimizing adverse effects, and targeting interventions. In this regard, molecular biology has gained significant prominence in oncology. However, we remain distant from achieving victory in the battle against cancer; hence, biomedical research in oncology must persist as a worldwide imperative.

Keywords: Cancer, oncogenes, tumor, tumor suppressor genes, Molecular Methods

Introduction :

Cellular genetic modifications result in the state termed cancer. Oncogenes must be activated for cancer to manifest, which might transpire in one of two manners. Oncogenes may be activated by tumor viruses that invade cells or may arise from the transformation of normal genes, referred to as proto-oncogenes, into oncogenes. For instance, the alteration of proto-oncogenes, such as

SRC developing into the v-SRC oncogene via mutation, and ABL proto-oncogenes generating the BCR-ABL oncogene through gene fusion. A single cell's oncogenic alteration can initiate tumorigenesis. Tumor development is initiated by oncogenic changes resulting from gene mutations in proto-oncogenes, leading to the transformation of normal cells into malignant ones. Metastasis signifies the ability of cancer cells to disseminate from the primary tumor to other regions of the body, complicating treatment and often yielding suboptimal outcomes. Targeted medicines that seek to disrupt the molecular progression of cancer enhance patient outcomes, with advancements in diagnostic and treatment options guided by research into these pathways. Research indicates that the Rous sarcoma virus may facilitate the transmission of malignancy among animals in instances of solid tumors, such as sarcomas. Rous was awarded the Nobel Prize for this achievement. Subsequent study demonstrated that tumors might also develop in the absence of viral DNA (Imran et al., 2017). In the early 1900s, Peyton Rous conducted pioneering research on the Rous sarcoma virus. He discovered a virus that induces cancer in hens, and in 1966, he received the Nobel Prize in Physiology or Medicine for his groundbreaking study. A pivotal moment in cancer research occurred when Rous identified a virus capable of inducing tumors, therefore elucidating the viral etiology of some cancer forms. This discovery stimulated further investigation into oncogenic viruses, significantly enhancing our understanding of the molecular processes underlying cancer and creating new opportunities for study in virology and cancer biology. The pervasive impact of cancer, which influences millions of lives and is a principal cause of morbidity and death worldwide, underscores the pressing want for innovative strategies in its comprehension and management (Gulati and Singh, 2024).

Role of Oncogenes:

Oncogenes are genes that possess the capacity to induce cancer and play a pivotal role in the progression of many cancer types. The finding of the SRC oncogene in a chicken retrovirus in 1970 was a pivotal moment in cancer research. Proto-oncogenes, often normal genes, can acquire unregulated states through mutations, leading to uncontrolled cell proliferation and, ultimately, cancer (Croce, 2008). The oncogenic allele's dominant nature necessitates only a single copy at the genomic level to alter the normal function of a gene. Oncogenes may originate from cellular sources or viral entities (Milner, 2003). Certain proto-oncogenes can alter their function and transform into oncogenes by duplication, addition, insertion, deletion, or chromosomal rearrangement. The uncontrolled overexpression of the protein caused by these mutations may lead to tumor growth. These mutations may arise from viral infections, radiation, chemical exposure, injury, or disease, among many external, internal, or combination factors (Croce, 2008).

Role of Tumor suppressor genes:

The inhibition of cellular proliferation and the cessation of tumor development are mostly achieved by tumor suppressor genes. The inactivation of tumor suppressor genes in many malignancies abolishes their inhibitory regulation on cell growth, leading to uncontrolled cell proliferation and the development of cancer. Loss-of-function mutations in tumor suppressor genes incapacitate their ability to control cell proliferation. A single copy of a tumor suppressor gene is often sufficient to inhibit cell proliferation; however, both copies are necessary to initiate tumor growth. These alterations exert a recessive influence. The essential functions of the protein products of tumor-suppressor genes encompass the creation of checkpoint-control proteins that halt the cell cycle in the presence of DNA damage or chromosomal anomalies. The enzymes involved also facilitate DNA repair and apoptosis. These proteins function as hormone receptors to inhibit cellular growth and proliferation. In addition to these roles, intracellular proteins regulate or inhibit advancement through a certain stage of the cell cycle (Dakal et al., 2024).

The tumor suppressor gene p53 meticulously regulates cell cycle progression and programmed cell death. Upon DNA damage, p53 may stop the cell cycle to allow for DNA repair or, if the damage is irreparable, induce programmed cell death (apoptosis). This is achieved by activating many genes that control and oversee the cell cycle. Alterations in the p53 gene can inhibit its function as a tumor suppressor, leading to unregulated cellular proliferation and compromised DNA repair mechanisms. Mutations in the p53 gene are considered the most prevalent genetic alterations in human malignancies, occurring in 50% or more of cases (Liu et al., 2024). Tumor suppressor genes may be studied at the DNA, mRNA, and protein levels in normal and tumor cells using a variety of techniques. A heterozygosity test may determine the likelihood of acquiring a certain kind of cancer. Polymerase chain reaction (PCR) amplification, RNase protection assays, single-strand conformational polymorphism, and denaturing gel electrophoresis are among the methods used to assess genetic changes. Immunometric approaches can be used on tissue samples and tumor cell lysates to analyze changed proteins, such as p53 (Ahmed et al., 2013).

Molecular Diagnosis of Cancer:

The accurate diagnosis of cancer patients is a critical issue in their clinical management. Immunohistochemistry, immunofluorescence, and DNA and RNA analysis using in situ hybridization and fluorescent in situ hybridization are among the methods developed for malignancy subtyping. The molecular biology approaches utilized to subtype cancer specimens such as allele-specific PCR, next-generation sequencing (NGS), Sanger sequencing, pyrosequencing, snapshot assays, and mass spectrometry-based assays using semiconductor or fluorescence detectors. Next-generation sequencing (NGS) has elucidated the complete spectrum of cancers and identified recurring mutations amenable to treatment with innovative therapies. Genomic-level investigations will continue to exert significant influence for an extended period (Lopez-Chavez et al., 2015).

Retroviral Therapy:

Retroviruses (RVs) were applied as an alternate strategy compared to standard tumor therapy by delivering genes into mammalian cells (Fig 1). The most often employed retrovirus for this reason is the Moloney murine leukemia virus (MoMLV). In the past twenty years, recombinant viruses have been intentionally created to enhance their application in transgenic animal development, reliable siRNA administration, and clinical gene therapy studies. Recent research indicates that RVs can effectively address severe immunodeficiency diseases in clinical studies. Despite the potential advantages, using RVs for gene therapy has inherent risks. (Goncalves, 2017). In a comparative experiment, two sets of vectors were generated and assessed; one was defective and required a supplementary retrovirus, while the other has inherent replicative capabilities. The defective group transduced less than 1%, but the replicative viruses had a transduction rate over 85%. This work illustrates the prospective application of RVs in the development of cancer gene therapy (Cooray, Howe and Thrasher, 2012). Research on retroviral vector insertions, particularly their capacity to induce oncogenesis, has garnered increased interest in recent years. For several years, researchers have utilized viral insertion sites to identify potential oncogenes and cancer signaling pathways. The efficacy of this strategy has, nevertheless, expanded due to the development of novel techniques such as high-throughput PCR for insertion site cloning, the availability of genetically modified animals, and the culmination of the mouse genome project (Fung and Gerson, 2014).

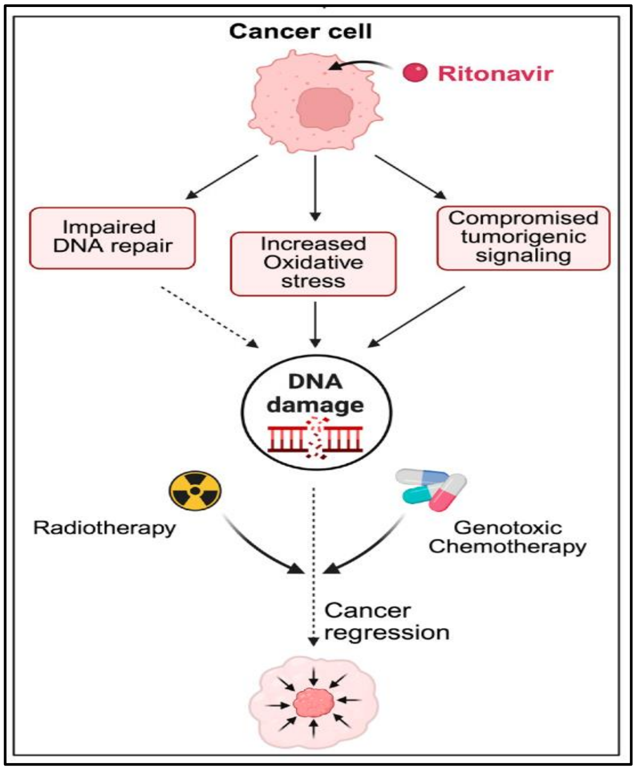

Figure 1: Depiction of RTV operations. RTV slows DNA repair, alters oxidative stress, and interferes with survival pathways in cancer cells. The resultant development of DNA damage renders cancer cells more susceptible to radiation and/or genotoxic chemotherapy, hence justifying the use of combination treatments (Pomella et al., 2025).

Transcription Activator-Like Effector Nucleases (TALENs):

The TALEN system is related to the ZFN system as it necessitates DNA-binding domains and a nuclease domain for genome editing. TALENs, conversely, have a stronger affinity for their target sites and differentiate individual nucleotides rather than recognizing a triplet of nucleotides (Fig 2) (Campbell et al., 2013). Furthermore, TALENs are more straightforward to design than ZFNs. Two specifically engineered TALENs are required to identify the target gene's DNA sequences on complementary strands for the application of TALENs in cancer therapy. Upon dimerization of the TALENs' FokI nuclease cleavage domain, it cleaves the target gene sequence, resulting in double-stranded DNA breaks. The target gene is modified by the end-joining DNA repair process, which rectifies the damage resulting from the DNA break due to the alteration in the reading frame. This approach also facilitates the removal of pre-existing mutations. TALEN technology is an advanced gene editing technique for addressing cancer cells, as it can modify complex cancer genes and target any gene inside the genome (Wu et al., 2014).

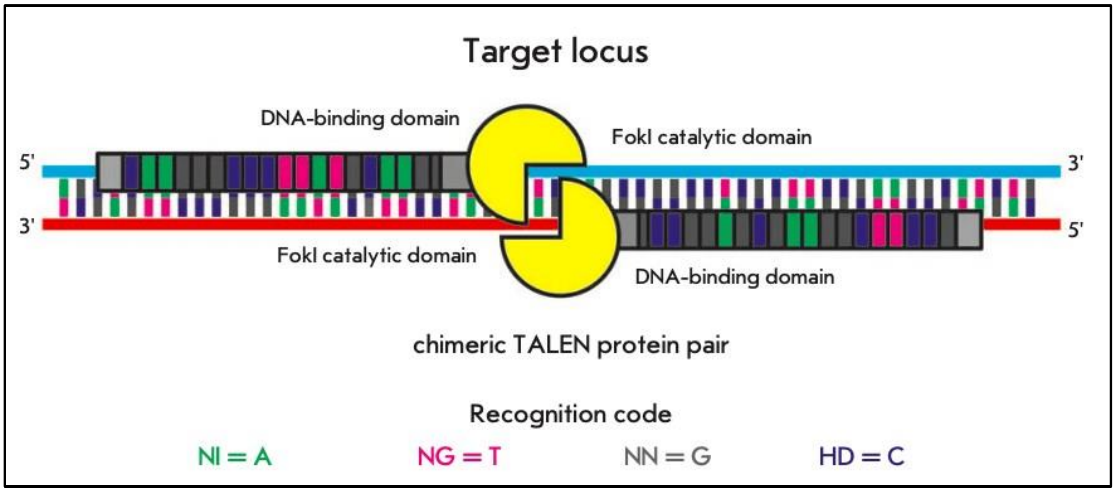

Figure 2: Introducing a double-strand break using chimeric TALEN proteins. (Nemudryi, 2014).

The CRISPR/Cas9 System:

The field of genetics has experienced a transformation due to the genome-editing method referred to as CRISPR-Cas. This approach effectively elucidates the functional organization of the genome and identifies causal genetic variations by enabling targeted mutation of any DNA sequence within the genome of any organism, both in vitro and in vivo (Fig 3). The utilization of this technology in cancer research, diagnosis, and therapy has extensive ramifications (Shamshirgaran et al., 2022). The CRISPR-Cas9 system is a very powerful genome editing method employed to treat cancer by excising oncogenic genes and replacing them with healthy counterparts. The system consists of a specifically designed single-guide RNA (sgRNA) and the Cas9 endonuclease. Utilizing crRNA and tracrRNA, the sgRNA identifies a specific target sequence and guides the Cas9 enzyme to cleave the DNA at that locus. The numerous bacterial and archaeal repeat sequences recognized as genes and their mechanisms of action have led to the classification of CRISPR-Cas systems into three main types (I-III). Specialized Cas endonucleases in type I and III systems transform pre-crRNAs into crRNAs. Every crRNA molecule, which includes multiple Cas proteins, then recognizes and cleaves target sequences that are complementary to the crRNA. The type II system serves as the nucleus of the genome engineering tool because to its utilization of fewer Cas enzymes (Bhatia and Yadav, 2023). Although CRISPR-Cas9 may be administered in vitro to a targeted site and is very successful for malignancies characterized by single gene alterations, it poses significant challenges for metastatic cancers (Yang et al., 2025).

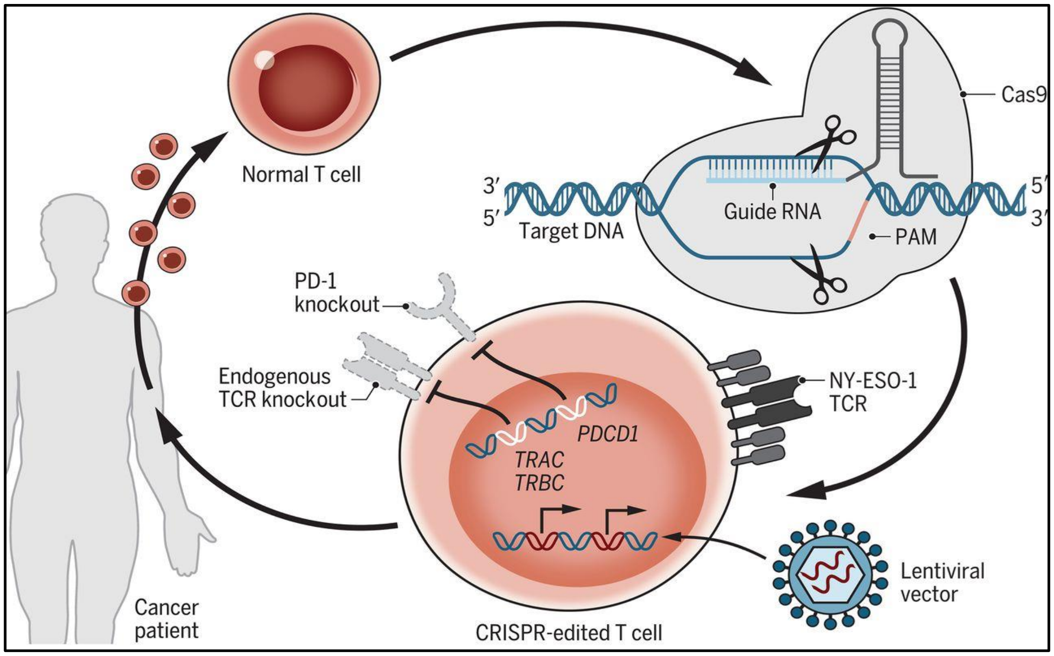

Figure 3: CRISPR-Cas9 engineering of T cells in cancer patients (Stadtmauer et al., 2020).

The CRISPR/Cas9 technology possesses several benefits over ZNFs and TALENs. Initially, because to CRISPR's dependence on ribonucleotide complex synthesis instead of DNA recognition, the target design process is simplified, more cost-effective, and eliminates the need for laborious cloning phases. Moreover, CRISPR has superior efficacy compared to ZFNs and TALENs, enabling it to target any specific DNA sequence inside the genome. The host genome may be directly edited by injecting RNA that encodes the Cas protein, and several genes can be simultaneously modified by introducing various gRNAs. This method is superior for GC-rich target areas, as it is more rapid than traditional approaches and is unaffected by DNA methylation (Chen et al., 2021)

Conclusion:

The advancements in molecular biology have profoundly transformed cancer research and treatment. This investigation has traversed the intricate domain of cancer-associated genes. Molecular biology has elucidated the genetic underpinnings of cancer and has also generated essential diagnostic and treatment instruments. We have successfully elucidated the genetic intricacies of malignancies using techniques such as NGS, which has facilitated the development of personalized therapeutic strategies. Notwithstanding the introduction of other therapeutic alternatives, the CRISPR-Cas9 system continues to be at the forefront of innovation. This innovative genome-editing technology provides patients worldwide with renewed hope for precise and personalized cancer treatment. Molecular biology has provided us with the means to combat it while also elucidating the complex aspects of its progression. The future of cancer therapy appears increasingly promising as we continue our investigation, discovery, and invention.

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Declaration of Competing Interest:

The researcher declares that he has no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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