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Targeting tumors with precision: The rise of oncolytic viruses in molecular oncology research.

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Introduction

Cancer remains a global health burden, challenging scientists and clinicians to continually evolve strategies for more precise and less toxic treatment modalities. Among the many breakthroughs in molecular oncology research, oncolytic viruses (OVs) have emerged as a promising and innovative therapeutic platform. Unlike traditional treatments, OVs are genetically engineered or naturally occurring viruses that selectively infect and kill cancer cells, while sparing normal tissue. Their dual mechanism—direct oncolysis and immune system activation—places them at the intersection of virology, immunology, and oncology. As our understanding of tumor genetics and host-virus interactions deepens, OVs are becoming a central focus in personalized cancer treatment. From preclinical models to clinical trials, their role is being amplified by advances in molecular profiling, which enable precise targeting and customization. This article explores the development, mechanism, and translational potential of oncolytic viruses in the realm of molecular oncology [1, 2].

The unique ability of OVs lies in their selective replication within cancer cells. This is possible because tumor cells often have defective antiviral responses, such as impaired interferon signaling, which makes them more susceptible to viral infection. Once inside the tumor cell, the virus replicates, leading to cell lysis and the release of new viral particles that can infect neighboring cancer cells. However, the benefits of OVs go beyond simple oncolysis. Tumor cell destruction by viral replication results in the release of tumor-associated antigens (TAAs), which are captured by antigen-presenting cells (APCs), triggering a systemic anti-tumor immune response. This mechanism effectively turns the tumor into a vaccine against itself—a feature that makes OVs uniquely suited to complement immunotherapies like immune checkpoint inhibitors [3, 4].

Modern molecular oncology research has allowed for the customization of OVs to improve their specificity, safety, and therapeutic payload. Genetic engineering can be used to delete viral genes that cause toxicity in normal cells while enhancing replication in tumor environments. For instance, modifications in the herpes simplex virus (HSV-1) and adenovirus platforms have led to the development of clinical-grade oncolytic vectors such as T-VEC (talimogene laherparepvec), approved for the treatment of advanced melanoma. Moreover, OVs can be armed with therapeutic transgenes that express immune-stimulating cytokines (e.g., GM-CSF, IL-12) or tumor-suppressing proteins, boosting their anti-cancer efficacy. Such multi-functional viruses represent a new generation of targeted therapies that bridge viral biology with molecular oncology precision [5, 6].

The efficacy of oncolytic virus therapy is closely tied to the molecular characteristics of the tumor. Molecular profiling enables identification of key biomarkers—such as RAS mutations, p53 status, or expression levels of viral receptors—that predict a

patient's susceptibility to specific OVs. This precision-medicine approach ensures that therapy is tailored to exploit the specific vulnerabilities of each tumor. For example, reovirus naturally targets cells with activated Ras signaling—a pathway commonly mutated in colorectal, lung, and pancreatic cancers. Similarly, vesicular stomatitis virus (VSV) demonstrates high selectivity in tumors with impaired interferon responses, providing a molecular rationale for virus selection based on tumor genotype [7, 8].

Recent studies suggest that combination therapies involving OVs and other treatment modalities may unlock even greater therapeutic benefits. Oncolytic viruses can reshape the tumor microenvironment (TME), converting it from immunosuppressive to immunostimulatory, thereby enhancing the effectiveness of immune checkpoint blockade (e.g., anti-PD-1, anti-CTLA-4). In parallel, OVs can be combined with chemotherapy or radiation to sensitize tumors, reduce resistance, and achieve better control of metastatic disease. For example, OV-infected tumors show increased infiltration of CD8+ T cells and natural killer (NK) cells, potentiating an immune-mediated response that can eliminate residual cancer cells systemically. While the promise of oncolytic virotherapy is substantial, several challenges remain. Host antiviral immunity may limit viral replication, especially in patients with pre-existing antibodies. Strategies such as transient immunosuppression or use of novel delivery systems (e.g., stem cells, nanoparticles) are being explored to overcome these limitations [9, 10].

Conclusion

Oncolytic viruses represent a paradigm shift in cancer treatment, bringing a new level of precision, adaptability, and immune engagement. Rooted in molecular oncology research, they exemplify how deep mechanistic understanding can be translated into innovative, patient-specific therapies. As the field progresses, integrating molecular profiling with oncolytic design will be critical in realizing the full potential of this approach. In the coming years, we may witness a future where tumors are no longer merely treated but strategically disarmed at the molecular and immunological level—by a therapy as intelligent as it is unconventional.

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