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Review Article

Next-Generation Oncolytics Viruses: Smart, Immune-Driven Cancer Destroyers

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ABSTRACT

Cancer is still one of the leading causes of death worldwide because of adverse effects and inadequate tumor clearance. Oncolytic Viruses (OVs) offer a new and promising approach by selectively infecting and destroying cancer cells while protecting healthy tissue. By both directly lysing tumors and strengthening the immune system, these viruses improve anticancer responses in ways that conventional medicines are unable to. This paper aims to provide a comprehensive overview of next-generation oncolytic virotherapy with a focus on their underlying mechanisms, therapeutic potential, and clinical developments. By merging data from preclinical and clinical studies, this review aims to establish how OVs are becoming a crucial part of cancer immunotherapy while recognizing that challenges remain. For methodology, a comprehensive review of recent research, including lab tests and clinical trials, was conducted. Among the numerous DNA and RNA viruses we studied were adenovirus, herpesvirus, reovirus, poliovirus, measles virus, coxsackievirus, and other naturally occurring and genetically modified viruses. The review's primary subjects included tumor selectivity, immune activation potential, genetic changes, delivery strategies, and safety and efficacy findings from human trials. The results demonstrate that OVs effectively target tumor cells by altering the tumor microenvironment and exploiting their unique limitations. Genetically modified viruses that produce cytokines like GM-CSF and IL-12 enhance immune responses. Clinical trials are advancing an abundance of alternatives, and clinically approved drugs such as talimogene laherparepvec (T-VEC) have demonstrated significant benefits. Immune suppression and targeting limitations may be resolved by immune checkpoint inhibitor combined therapies and novel delivery methods. In conclusion, oncolytic virotherapy is a highly advanced and innovative method of treating cancer. Ongoing research is necessary to improve virus delivery, increase therapeutic efficacy, and increase availability-all of which have the potential to significantly change cancer care worldwide.

Keywords: Cancer immunotherapy, Immune activation, Oncolytic viruses, Tumor-selective oncolysis, Viral gene modification.

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INTRODUCTION

The world's second most widespread cause of death, cancer is a complex disease with mysterious causes and processes. Researchers are investigating how stem cells affect tumors and how they contribute to carcinogenesis, metastasis, and recurrence. Novel therapeutic methods for the treatment of cancer may result from an understanding of these features (Yin *et al.*, 2021). Viruses are divided into single-stranded and double-stranded DNA and RNA types, with various viral families emerging. They reproduce, translate, and survive by utilizing cellular components, binding to cell surface receptors or fusion plasma membranes (Fu *et al.*, 2019). Without harming healthy cells, oncolytic viruses infect and multiply within cancerous cells, resulting in cell death. When the first genetically engineered virus appeared in the 1990s, oncolytic virotherapy regained popularity. Recently, a number of viruses

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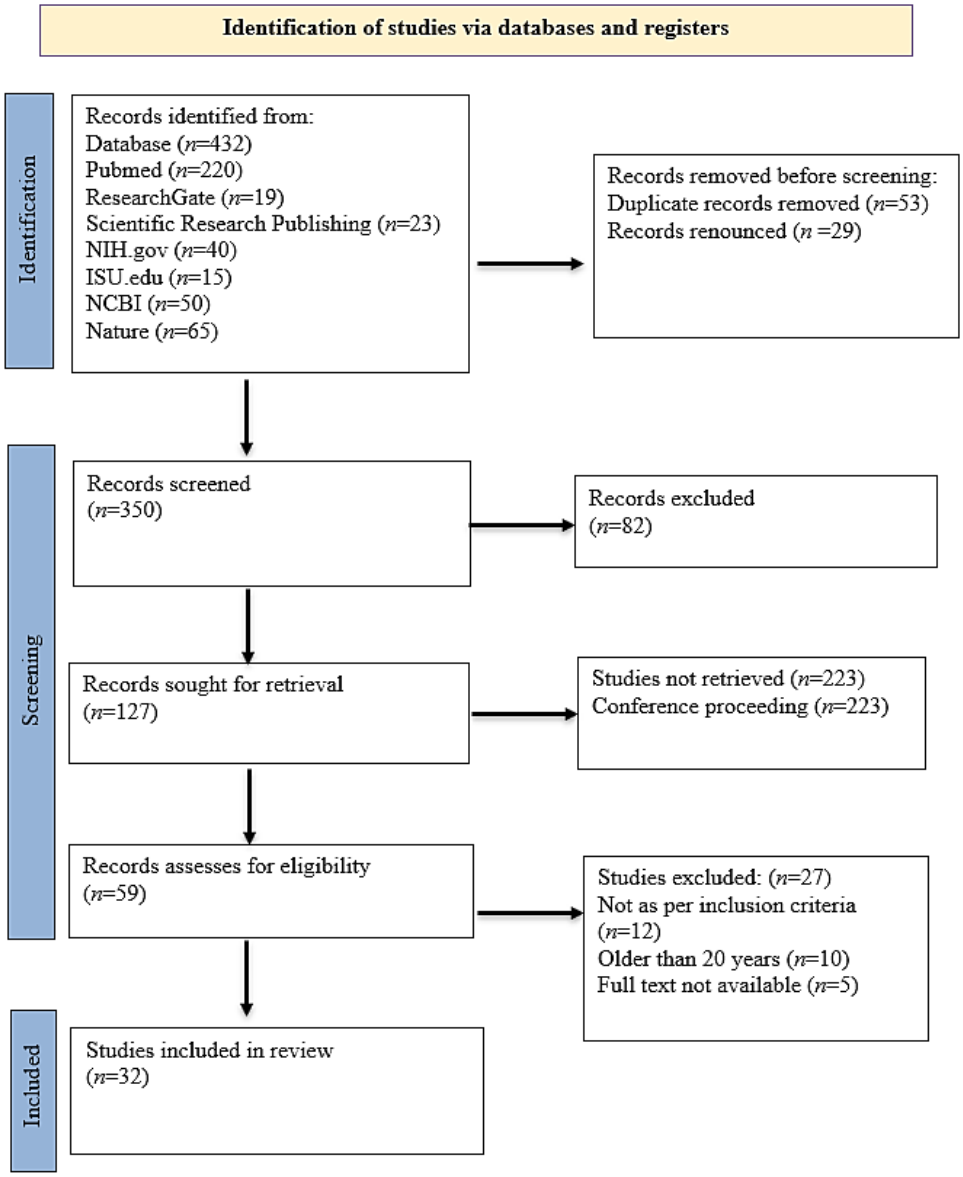

have begun to undergo clinical testing, suggesting advancement (Gupta *et al.*, 2025).

Genetically engineered viruses and attenuated natural viruses are two types of Oncolytic Viruses (OVs). These viruses substantially affect Oncolytic Viral Therapy (OVT) in cancer treatment. Compared to conventional immunotherapies, oncolytic viruses provide a number of advantages, including precise targeting, excellent tumor eradication efficiency, and fewer adverse effects (Fukuhara *et al.*, 2016). The main issue is OV delivery, as systemic OV injection is difficult and requires overcoming innate immune responses, particularly antiviral immunity, and inadequate target distribution (Yoo *et al.*, 2019).

In this review, we briefly outline the various virus types and the anti-tumor mechanism of OVs. We also review the potential uses of OVs in cancer immunotherapy and highlight the particular challenges in developing OV therapies, with an emphasis on the current regulatory approval of OV-based therapies and preclinical and clinical evidence.

MECHANISM OF ACTION OF ONCOLYTIC VIRUSES

OV treatment is the process of using a virus that self-replicates to kill its host cells in cancer cells that have been infected. The virus prevents the cell from making host products and encourages the production of viral products by taking over the cell's protein synthesis. Several viral particles that can infect other cells will be released when the virus-infected host cells lyse (Figure 1) (Thorne *et al.*, 2005).

According to studies, cellular transformation affects oncolytic Measles Virus (MV) replication, and MV-induced oncolysis rises as transformation does. Depending on the degree of transformation, the type 1 interferon response to MV infection

is both markedly diminished and delayed. Treatment with IFNβ restores resistance to oncolysis caused by MV. IFN-induced transmembrane protein 1 is the most down-regulated immune gene, according to gene expression profiling, which shows a lowered basal level of immune-related genes in fully converted cells. A significant factor in MV's oncolytic selectivity is the basal decrease in IFN pathway functions, as stable IFITM1 overexpression boosts cell survival and inhibits viral replication (Ribas and Wolchok, 2013).

Tumor-specific immunity is enhanced by tumor cell lysis, soluble tumor antigens, and molecular factors. T-VEC, a genetically altered herpes simplex virus, has shown promise in treating melanoma and other cancers. Combining it with other immunotherapy techniques is suggested due to its safety profile. Oncolytic viruses offer a new class of cancer treatments (Gotwals *et al.*, 2017).

Oncolytic viruses are ideal for tumor vaccination due to their ability to destroy tumor cells directly, release tumor antigens, and cross-prime antitumor Cytotoxic T Lymphocytes (CTLs). The effectiveness of oncolytic virotherapy depends on balancing direct and indirect killing phases, which can be adjusted by adding immunomodulatory genes. As more transgenes are added, oncolytic vaccinations are expected to become the preferred immunotherapeutic anticancer strategy (Elsedawy and Russell, 2013).

The tumor microenvironment regulates the activation of the innate immune response, which can be reduced by viral combined immunosuppressive drugs like sunitinib. These drugs inhibit natural immune systems, suppressing immunity in tumors and strengthening the virus's antitumor effect. In tumor-bearing mice, sunitinib and vesicular stomatitis virus together may significantly reduce the growth of cancerous tumors like kidney, breast, and prostate cancers (Jha *et al.*, 2013).

OV treatment involves neutralizing antibodies to limit virus growth and dissemination through natural immunity. However, the virus can trigger adaptive immune cells to kill tumor cells. The immune system plays a crucial role in treating OVs, triggering an immune response via signal transduction pattern recognition receptors and pathogen-associated molecular patterns, such as p53, PKR, and phosphorylated retinoblastoma protein pathways. Tumor cells frequently have aberrant signal transduction pathways, which allow the virus to replicate and amplify in those cells (Young *et al.*, 2013).

GENETICALLY ENGINEERED OVS

For the purpose of making oncolytic viruses safe and efficient against cancer, their genomes are usually genetically modified. This is now possible because of developments in genetic engineering along with the understanding of viral genes. These viruses directly infect cancer cells, and by enhancing immune responses, increasing viral replication in tumors, and increasing dosage to reach metastases, their efficacy can be increased. Natural viruses must be modified or targeted to have the greatest possible effect on cancer cells because they do not primarily target tumors (Crance *et al.*, 2003).

To improve the safety and efficacy of oncolytic viruses against cancer, their genomes are usually genetically modified. Examples

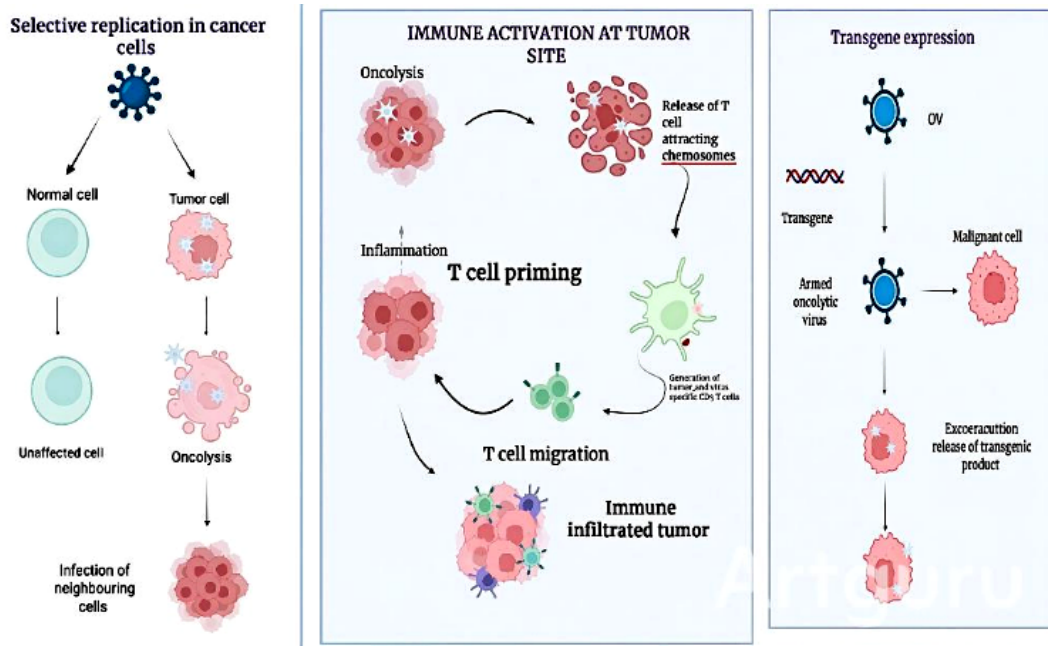

Figure 1: Mechanism of action of oncolytic virus: Oncolytic viruses particularly multiply within cancer cells, inducing oncolysis and immunological activation at the tumor site, whereas modified viruses also generate therapeutic transgenes for particular anticancer effects.

Table 1: Examples of Genetically Modified Oncolytic Viruses and their Target Cancers: Genetically Modified oncolytic viruses are designed to specifically target and kill different types of cancer by adding, eliminating or changing viral genes in order to improve cancer cell selectivity and safety for therapeutic applications.

Virus backbone	New OVS viruses	Gene Modification	Gene Insertion	Cancer type	References
HSV-1	G47Δ	Deleting the alpha47 gene	US1 from gamma34.5	Glioblastoma	(Todo *et al.*, 2001)
HAdV-5	Telomelysin (OBP-301)	E1A and E1B Promoters	HTERT Promoter	Oral squamous cell carcinoma (OSCC)	(Gohara *et al.*, 2022)
HSV	OncSyn	Replace natural viral promoters with tumor-selective logic gates	Inserting miRNA response elements (e.g., miR-122, miR-124) into viral genomes	Glioblastoma, Hepatocellular Carcinoma	(Israyelyan *et al.*, 2007)
Adenovirus	ColoAd1 or Enadenotuvire	Hybrid of Ad11p and Ad3	No foreign gene is inserted	Colon cancer	(Kuhn *et al.*, 2008)
Adenovirus	VCN-01	Δ24 deletion (deletion of 24 base pairs in the E1A CR2 region)	Human recombinant hyaluronidase PH20 (rPH20)	Neuroectodermal tumor, Pancreatic cancer,	(Garcia-Moure *et al.*, 2019)
Adenovirus	AdΔE1B-RLX	E1B gene	Relaxin (RLX) gene	Pancreatic Ductal Adenocarcinoma, Hepatocellular Carcinoma	(Kim *et al.*, 2006)
Adenovirus	Ad.IR-E1A/TRAIL	E1A promoter	Express TRAIL (tumor necrosis factor-related apoptosis-inducing ligand)	Colorectal Cancer, Lung Cancer	(Sova *et al.*, 2004)
Reoviruses	Type 3 Dearing strain	Naturally oncolytic	GM-CSF, IL-12, IFN-β, TRAIL	Human Breast cancer	(Hata *et al.*, 2008)
Coxsackievirus	CVA21	Wild-type Kuykendall strain	No gene is inserted	Breast cancer	(Skelding *et al.*, 2009)
Poliovirus	PVSRIPO	Polio/rhinovirus recombinant	HRV2-derived IRES element	Human breast and prostate cancer	(Holl *et al.*, 2016)

of genetically modified oncolytic viruses and their target cancers are presented in Table 1.

OVT: INNOVATIVE CLINICAL TECHNIQUES AND COMBINATION APPROACHES

Research on Oncolytic Viruses (OVs) began in the early twentieth century. The approval of Imlygic® [Talimogene laherparepvec (T-VEC)] by the FDA in 2015 marked oncolytic viruses' emergence as recognized potential therapies worldwide, notwithstanding previous approvals in the 2000s (Pol *et al.*, 2016). There are two OV products which are yet to be approved and some are approved worldwide for the treatment of cancer. Oncolytic virus–based products in clinical development and clinical trials conducted to expand their therapeutic use are presented in Table 2.

FUTURE DIRECTION

Since Oncolytic Viruses (OVs) can enhance the future prospects of immunologically cold tumors and interact well with current therapies like chemotherapy and radiation, their development continues to progress quickly. There continue to be issues, though, including the variability of clinical trial outcomes, an absence of prognostic biomarkers, and poor efficacy in advanced metastatic tumors. The necessity for more thorough characterization of individuals who respond versus non-responders has been demonstrated by the variability in patient responses. Promising

Table 2: Oncolytic Virus- Based Products in Clinical Development: Clinical trials are currently being carried out to expand the therapeutic use of oncolytic virus-based medications, such as Bracelet-1, RP1, VAXINIA and ONYX-015. These medications showcase modern techniques for treating metastatic and advanced malignancies.

Product name	Virus types	Indication	Clinical trial status	References
Bracelet-1	Poxvirus	Metastatic HR+/HER2-breast cancer.	Undergoing a Phase 2 trial	(Zhao *et al.*, 2023)
RP1 (vusolimogene oder parepvec)	Herpes Simplex Virus Type 1	Advanced melanoma.	Undergoing a Phase 3 trial	(Joshi *et al.*, 2024; Zhao *et al.*, 2023)
VAXINIA (CF33-hNIS-antiPDL1)	Chimeric vaccinia virus	Pancreatic ductal adenocarcinoma	Early-phase clinical trials	(Woo *et al.*, 2020)
ONYX-015	Adenovirus	Breast cancer	Undergoing Early clinical trials	(Parmar *et al.*, 2022)

Table 3: Recently Approved OV Drugs: Recently licensed oncolytic viral medications such as Talimogene laherparepvec (T-VEC), Rigvir, and Oncorine offer novel, regulatory-recognized immunotherapy options for melanoma, nasopharyngeal carcinoma, and similar malignancies. These medications represent significant advancements in cancer treatment.

Product name	Virus types	Approved by	Indication	Approval Date	Injection	References
Talimogene laherparepvec (T-VEC)	Herpes Simplex Virus (HSV)	USA, EU	Advanced melanoma	October 27, 2015	IT	(Ferrucci *et al.*, 2021)
Oncorine (H101)	Adenovirus	China	Nasopharyngeal carcinoma	October 2005	IT	(Liang, 2018)
Delytact	Vaccinia virus	Japan	Malignant glioma	February 2021	IT	(Frampton, 2022)
Rigvir	Enteric cytopathic human orphan virus type 7 (ECHO-7)	Latvia, Georgia	Melanoma	2004 (Latvia) 2015 (Georgia)	IM	(Alberts *et al.*, 2018)

strategies utilising synthetic or cell-based carriers are being studied to enhance OV targeting and accumulation in tumors with the aim to resolve delivery challenges (Yun *et al.*, 2022).

Recently approved oncolytic virus drugs represent significant advancements in cancer treatment and are presented in Table 3.

CONCLUSION

Because of their exceptional capacity to both stimulate antitumor immunity and selectively infect, replicate within, and destroy tumour cells, oncolytic viruses have become a promising class of anticancer agents. The therapeutic potential of OVs has been reinforced by advances in genetic engineering, which have improved safety, improved tumour targeting, and made it possible to incorporate immune-boosting transgenes. Although T-VEC remains the most notable clinically approved OV, ongoing preclinical and clinical studies continue to expand the therapeutic landscape across different cancer types.

Despite these developments, widespread clinical success is constrained by issues like ineffective systemic delivery, pre-existing antiviral immunity, and inconsistent clinical responses. Addressing these issues requires deeper understanding of tumour biology, identification of predictive biomarkers, and the development of innovative delivery platforms. However, we still face challenges. Their current success is limited by problems like pre-existing immunity, which occurs when the body fights the virus too early, and challenges in getting the virus into the bloodstream.

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ABBREVIATIONS

DNA: Deoxyribonucleic Acid; **RNA:** Ribonucleic Acid; **GM-CSF:** Granulocyte-Macrophage Colony-Stimulating Factor; **IL-12:** Interleukin 12; **T-VEC:** Talimogene Laherparepvec; **IFN:** Interferon; **PKR:** Protein Kinase R; **HSC:** Herpes Simplex Virus; **US1:** Unique Short 1 Gene; **HAdV-5:** Human Adenovirus Type 5; **HTERT:** Human Telomerase Reverse Transcriptase; **E1 A,B:** Early Region 1 A,B; **ColoAd1:** Chimeric Oncolytic Adenovirus Derived; **PH20:** Pegylated Recombinant Human Hyaluronidase; **AdΔE1B-RLX:** Adenovirus with Deletion of E1B

Gene Region-Relaxin; **Ad.IR-E1A:** Adenovirus Inverse Repeat E1A Vector; **CVA21:** Coxsackievirus A21; **PVSRIPO:** Poliovirus Sabin 1 Recombinant Poliovirus; **HRV2:** Human Rhinovirus 2; **IRES:** Internal Ribosome Entry Site; **RP1:** RNA Polymerase 1; **VAXINIA (CF33-hNIS-antiPDL1):** Vaccine Candidate Combined with Chimeric Vaccinia Virus (CF33) with Human Sodium Iodide Symporter (hNIS) and Anti-PD-L1i Immune Checkpoint Inhibitor Payloads; **IFITM1:** Interferon-Inducible Transmembrane Protein 1.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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