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Original Research Article

Oncolytic Virotherapy in Cancer: A Comprehensive Systematic Review of Recent Data and Current Landscape

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Abstract: Oncolytic virotherapy (OV) is an emerging and innovative approach to cancer treatment. By inducing virus-mediated immune responses, oncolytic viruses enhance tumor specificity, stimulate antitumor immunity, and selectively infect and lyse cancer cells. In this study, we conducted a systematic review of oncolytic virotherapy, summarizing the major types of oncolytic viruses and their applications in combination therapies. Different viral vectors possess distinct biological characteristics, and diverse strategies have been employed in the design and optimization of OVs. Given their ability to modulate immune responses and the tumor microenvironment, oncolytic viruses show strong potential when combined with conventional cancer treatments. In particular, combinations of OVs with immunotherapies and CAR-T cell therapies are discussed in detail. Nevertheless, the selection of combination strategies should take into account tumor location, standard treatment modalities, and the expression of relevant biomarkers to maximize therapeutic efficacy.

Keywords: Oncolytic Virotherapy, Cancer, Landscape, virus, Systematic Review.

INTRODUCTION

Oncolytic virotherapy is an emerging cancer treatment strategy that selectively replicates within and lyses cancer cells while largely sparing normal tissues [1]. Oncolytic viruses (OVs) exploit cancer-specific mutations and include both naturally occurring and genetically engineered viral platforms [1]. Prestwich *et al*., demonstrated the critical role of host immune responses in OV efficacy, highlighting virus-mediated immune activation as a primary driver of antitumor effects rather than direct oncolysis alone [2]. Kelly *et al*., provided a historical perspective on the use of non-pathogenic viruses that selectively target human tumors, tracing developments in this field back to the 1980s [3]. Over time, OV development has shifted from a focus on improving target specificity, selective replication, and direct tumor lysis toward the engineering of viral vectors capable of enhancing intratumoral immune responses and modulating tumor neovascularization and metabolism [4].

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The mechanisms employed by different OVs vary depending on viral vector characteristics and tumor cell type. These mechanisms include interactions with specific cellular receptors, suppression of antiviral pathways in tumor cells, and induction of immunogenic cell death (ICD) [4]. The design and selection of viral vectors require consideration of multiple factors, as OVs typically elicit broader antitumor immune activities compared with targeted therapies or immune checkpoint inhibitors (ICIs) [4, 5]. In addition, OVs can remodel the tumor microenvironment (TME) by influencing neovascularization, tumor metabolism, and the rigid extracellular matrix barrier [4].

Given the complexity of OV mechanisms, combination therapies can provide enhanced antitumor efficacy [6, 7]. This article presents a systematic review of oncolytic virotherapy, exploring diverse studies across different viral platforms and combination strategies. Using the PRISMA methodology, we conducted a systematic literature search and summarized key findings related to OV vectors, therapeutic mechanisms, and combination treatment approaches.

METHODOLOGY

Search Strategy for Oncolytic Virotherapy in Cancer Treatment:

We conducted a literature search using two databases, PubMed and Google Scholar (which includes MEDLINE), employing the search terms "cancer treatment" and "oncolytic virotherapy." Boolean operators ("AND," "OR") and Medical Subject Headings (MeSH) were applied to refine the search and ensure the inclusion of studies relevant to the objectives of this review. Table 1 summarizes the databases searched and outlines the stepwise process used to identify studies pertinent to oncolytic virotherapy in cancer treatment.

Table: 1 Types of databases we used in our study

Type of Database	Keywords	Search Strategy	Filter Used	Number of Records Identified
PubMed	Oncolytic Virotherapy, Cancer Treatment	"Oncolytic Virotherapy" OR "Cancer Treatment"	The complete English text from the last decade is available without restriction.	70
Google Scholar	Oncolytic Virotherapy, Cancer Treatment	Oncolytic Virotherapy, and Cancer Treatment	Released sometime between 2020 and 2023.	48

Inclusion and Exclusion Criteria

Only articles published in English were included, and studies in other languages were excluded. From PubMed, we selected studies published within the past decade, while from Google Scholar, we included articles released between 2020 and 2023. An independent review process was conducted to identify and remove duplicate records. Article titles and abstracts were screened, and studies that did not align with the study design were excluded. Ultimately, only research articles that met the review criteria and provided significant contributions to the advancement of oncolytic virotherapy for cancer treatment were included.

RESULTS

Based on the initial search, 118 records were identified across multiple databases and registries. After the removal of 34 duplicate records, 84 unique studies remained. Title and abstract screening excluded 54 records that did not meet the inclusion criteria, leaving 30 reports for full-text evaluation. These 30 studies were assessed in detail for eligibility. Studies were excluded if they lacked an appropriate study methodology, including seven primary research articles, one conference paper, one case series, and one research protocol. The case series did not meet the predefined inclusion criteria, the seven primary research studies were categorized as basic research and excluded, and the conference paper was excluded as a meeting abstract. Following these exclusions, 20 studies were deemed eligible and included in the final analysis.

Using a systematic approach guided by predefined criteria, the study selection process progressed through identification, screening, and eligibility assessment. The PRISMA flow diagram illustrating this process is presented in Figure 1. A summary of recent studies on oncolytic virotherapy in cancer treatment is provided in Table 2, while the distribution of included studies by cancer type is shown in Table 3.

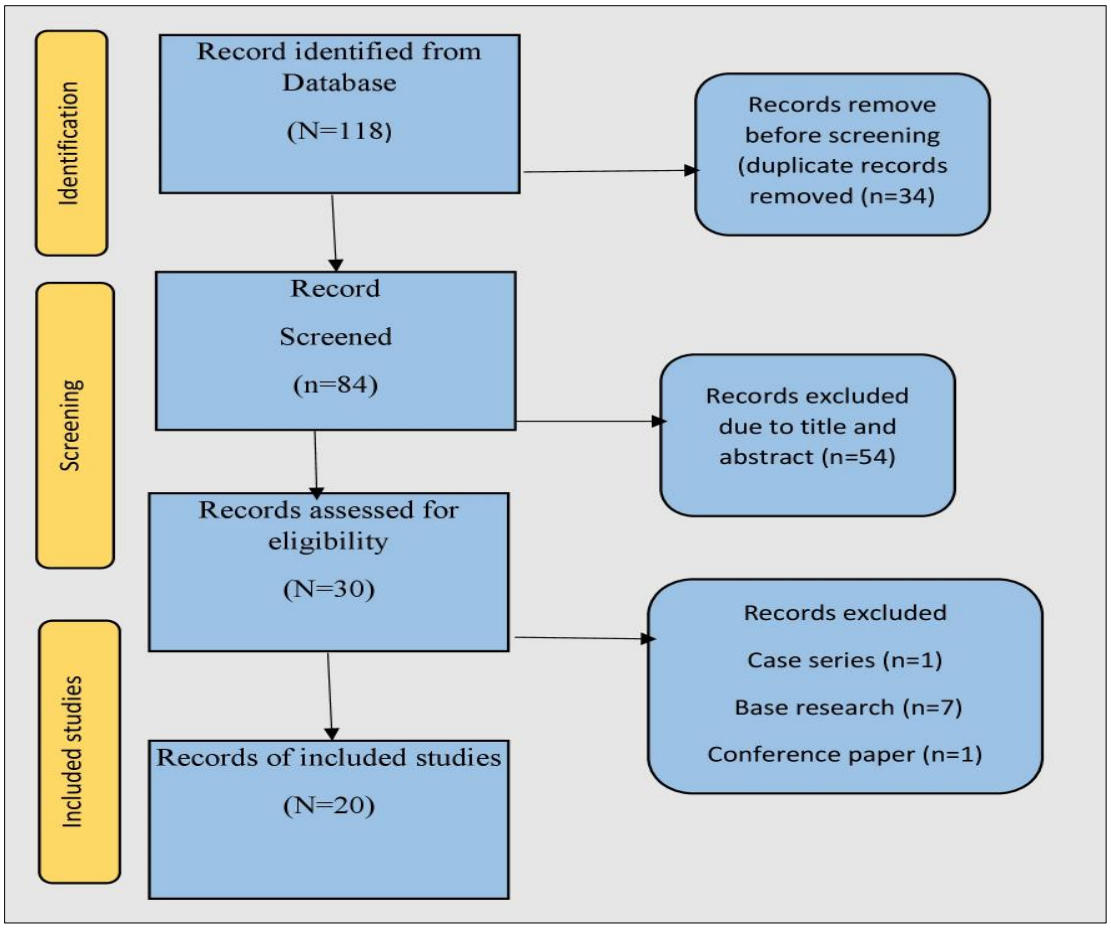

Figure 1: The PRISMA flow chart with the search results of our study

Table 2: Summarized of some recent studies for oncolytic virotherapy in cancer treatment

Author/Year	Quality Assessment Tool Used	Database Used	Conclusion
Abd-Aziz and Poh, 2021 [8]	SANRA	PubMed	The review provides insight into different characteristics of oncolytic viruses for effective applications in preclinical and clinical trials, addressing strategies to overcome limitations.
Jafari., 2022 [9]	SANRA	PubMed	The review explores the role of antibody-based therapeutics in combination with oncolytic viruses, contributing to understanding this immuno-virotherapy approach.
Feola., 2022 [10]	SANRA	PubMed	The review explores the crosstalk between oncolytic viruses and the immune system for cancer treatment, presenting insights into resistance mechanisms.
Lan., 2020 [11]	SANRA	PubMed	The study discusses the development of oncolytic virotherapy, focusing on genetic modification and combination therapy, providing a comprehensive overview of recent advances in this field.
Goradel., 2022 [12]	SANRA	PubMed	The study reviews oncolytic virotherapy as promising immunotherapy against cancer, emphasizing resistance mechanisms to oncolytic viruses.
Russell, 2019 [13]	SANRA	PubMed	The review explores oncolytic viruses' priming time for cancer immunotherapy, presenting perspectives on their application and potential benefits.

Author/Year	Quality Assessment Tool Used	Database Used	Conclusion
Chiocca and Rabkin, 2014 [14]	SANRA	PubMed	The review discusses oncolytic viruses and their application to cancer immunotherapy, providing insights into this therapeutic approach.
Ylösmäki, 2020 [15]	SANRA	PubMed	The study reviews the design and application of oncolytic viruses for cancer immunotherapy, presenting an overview of different strategies in this field.
Wei, 2018 [16]	SANRA	PubMed	The study explores fighting cancer with viruses, focusing on oncolytic virus therapy in China, providing insights into the progress in this region.
Deng and Wang, 2023 [17]	SANRA	PubMed	The study reviews research advances in the clinical application of oncolytic viruses for the treatment of gynecologic cancers, providing insights into current developments.
Marotel *et al*., 2020 [18]	SANRA	PubMed	The review explores the two faces of NK cells in oncolytic virotherapy, presenting perspectives on their functions in this context.
Hofman *et al*., 2021 [19]	SANRA	PubMed	The review article to explore the role of macrophage-mediated responses in oncolytic virotherapy efficacy.
Perez, M. C. *et al*., 2019 [20]	SANRA	Google Scholar	The observational study evaluates alipogene laherparepvec use for melanoma in the United States (COSMUS-1), providing insights into real-world clinical practice.
Louie, K. S. *et al*., 2020 [21]	SANRA	Google Scholar	The real-world use of alipogene laherparepvec in Germany is explored through a retrospective observational study using a prescription database, offering insights into prescription patterns.
Sun, J. *et al*., 2020 [22]	SANRA	Google Scholar	The observational study (COSMUS-2) investigates alipogene laherparepvec use for melanoma in the anti-PD-1 era in the United States, contributing real-world evidence in the context of evolving treatment approaches.
Kleemann, J. *et al.*, 2021 [23]	SANRA	Google Scholar	The retrospective study provides evidence for the effective use of T-VEC in old and oldest-old patients with melanoma.
van Akkooi, A. C. J. 2021 [24]	SANRA	Google Scholar	A retrospective chart review study assesses the real-world use of alipogene laherparepvec in unrespectable stage IIIB-IVM1a melanoma in four European countries, providing insights into its application across different regions.
Carr, M. J. 2022 [25]	SANRA	Google Scholar	Talimogene laherparepvec (T-VEC) is evaluated for the treatment of advanced locoregional melanoma after the failure of immunotherapy, presenting findings from an international multi-institutional experience.
Stahlie, E. H. A. *et al*., 2022 [26]	SANRA	Google Scholar	Two cohorts evaluating stage IIIB-IVM1a melanoma patients treated with T-VEC, concluded the overall performance was good.

Table 3: Characteristics of Included Studies by Types of Cancer

Characteristics	Number of Studies (n)
Brain	13
Breast	21
Gastrointestinal	7
Genitourinary	15

Characteristics	Number of Studies (n)
Gynecologic	5
Head and neck	2
Lung	10
Melanoma	2
Pediatric	3
Sarcoma	2
Other solid tumors	35
Hematological tumors	3
Total Cancer Cases	118

Virus Vectors in Oncolytic Virotherapy Studies

Oncolytic viruses (OVs) can be categorized into four groups based on their nucleic acid type: single-stranded RNA (ssRNA), double-stranded DNA (dsDNA), double-stranded RNA (dsRNA, e.g., reoviruses), and single-stranded DNA (ssDNA, e.g., parvoviruses). The dsDNA group primarily includes three virus vectors: adenovirus, vaccinia virus, and herpesvirus. In contrast, ssRNA virus vectors are more diverse, encompassing both positive-sense and negative-sense viruses. Table 4 summarizes the studies included, organized by virus vector type, and each type will be discussed individually in this section.

Adenovirus

Adenoviruses are a family of non-enveloped, icosahedral, medium-sized viruses with linear, double-stranded DNA that primarily infect epithelial tissues lining the respiratory tract [27]. Oncolytic adenoviruses achieve tumor specificity through several strategies: modifying viral genes to limit replication in normal cells while allowing replication in tumor cells, placing viral genes that initiate replication under the control of tumor-specific promoters, or altering viral coat proteins involved in host cell infection [28]. One common approach involves attenuating replication via the E1B gene. The E1B-55kD protein is essential for viral replication through the p53 pathway, and loss of E1B function prevents replication in normal cells [29]. In preclinical studies, the mutant adenovirus dl1520 significantly reduced tumor size [29–31]. Using overactive cellular promoters to drive viral replication is another strategy, although its effectiveness can vary depending on tumor type [29]. From a safety perspective, the most frequently reported adverse effects are flu-like symptoms, with occasional transient transaminitis and reversible hyperbilirubinemia [32–33]. In 2005, the recombinant oncolytic adenovirus H101 was approved by the China Food and Drug Administration (CFDA) for use in combination with chemotherapy in patients with refractory head and neck carcinoma [34]. Subsequent clinical trials have continued to show promising results.

Herpes Simplex Virus (HSV)

Human herpesviruses are among the largest and most complex viruses, with capsids composed of approximately 3,000 proteins [35]. The HSV-1 genome consists of 152 kb of linear double-stranded DNA, encoding around 90 genes, including multiple viral glycoproteins (gB, gC, gD, gH, and gL) and cellular receptors that facilitate virion fusion [36]. After fusion, viral genes are transported to the nucleus and expressed in a regulated sequence according to gene type: immediate early (IE), early (E), and late (L) [36]. One strategy for designing oncolytic herpesvirus therapies involves inserting a therapeutic transgene into the viral genome while deleting one or more essential viral genes, with the transgene typically carried on a plasmid [37]. Another approach uses conditionally replicating vectors, in which the virus preferentially infects, replicates in, and lyses tumor cells [37]. HSV-1 is currently one of the most commonly used strains in oncolytic virotherapy, with examples including Talimogene laherparepvec (T-VEC), G207, and G47Δ [38–40].

Reoviruses

Reoviruses are non-enveloped, icosahedral viruses composed of an outer capsid and an inner core, which contains a genome of ten double-stranded RNA segments. Their replication cycle occurs entirely in the cytoplasm [41]. Junctional adhesion molecule-A (JAM-A), an IgSF protein and a receptor for reoviruses, is expressed in many cancer types and is associated with tumor progression and the tumor microenvironment [42–43]. Ras transformation enhances three steps of viral replication: more efficient uncoating, an increased ratio of infectious to noninfectious viral particles, and more effective virus release. This makes reoviruses a potential therapeutic option for cancers with high rates of Ras mutations [44–45]. Examples of reoviruses used in cancer treatment include REO 008, REO 014, REO 016, and REO 021 [45]. Reolysin was approved by the FDA as an orphan drug in 2015 for ovarian cancer, pancreatic cancer, and glioblastoma [45].

Vaccinia Virus

Vaccinia virus (VV), a member of the poxvirus family, has a linear double-stranded DNA genome encoding approximately 200 genes [46]. Its replication occurs in the cytoplasm, and viral entry is mediated by fusion of the virion with the host cell membrane via glycosaminoglycans (GAGs) on the cell surface [46–47]. The entry-fusion protein complex includes eight viral proteins: A16, A21, A28, G3, G9, H2, J5, and L5 [48]. VV exists in two forms: intracellular mature virus (IMV) and extracellular enveloped virus (EEV), with the A34R gene promoting increased EEV production [46]. As an oncolytic agent, VV is often engineered with a deletion of the viral thymidine kinase (TK) gene, which restricts viral replication to rapidly dividing cancer cells [49]. Pexastimogene devacirepvec (JX-594) is an example of a targeted oncolytic vaccinia virus approved by the FDA as an orphan drug for hepatocellular carcinoma in 2013 [50–51].

Other Ovs

In addition to the virus types discussed above, several other viruses have potential as oncolytic vectors. Coxsackievirus, Seneca Valley Virus (SVV), and poliovirus belong to the Picornaviridae family and contain single-stranded RNA (ssRNA) genomes. Coxsackievirus A21 targets ICAM-1 and DAF, which are overexpressed in human melanoma cells [52]. Seneca Valley Virus isolate 001 (SVV-001), developed in 2002, is an oncolytic RNA virus that induces cytotoxicity in tumors with neuroendocrine characteristics [53], however, a phase II trial of NTX-010 showed no significant benefit in patients with extensive-stage small-cell lung cancer (SCLC) [54]. Poliovirus, also a Picornaviridae member, typically causes paralytic poliomyelitis, but a prototype intergeneric poliovirus chimera was engineered to infect and replicate in cell lines derived from malignant gliomas [55].

The Paramyxoviridae family includes negative-sense single-stranded RNA [ss(-)RNA] oncolytic viruses such as measles virus and Newcastle disease virus (NDV) [4]. Measles virus uses CD46 as its receptor, with replication occurring in the cytoplasm [56]. NDV 73T-R-198, a recombinant NDV, can selectively replicate in and kill tumor cells [57].

Other classifications of oncolytic viruses not covered here also exist. The key characteristics of the various OVs discussed are summarized in Table 5.

Table 4: Summarization of Included Studies by Virus Vectors

Virus Type	Number of Studies (n)
Adenovirus	30
HSV-1	23
Reoviruses	19
Poxviruses	12
Newcastle Disease Virus	5
Measles Virus	3
Seneca Valley Virus	2
HVJ-E Virus	2
Gamma-Herpes Virus	1
Parvovirus	1
Retrovirus	1
Others (Specify)	4
Total	97

Table 5: Characteristics of Types of Viruses

Virus	Attributes	Oncolytic Treatment for Malignancies
Adenovirus	dsDNA, enveloped, icosahedral	Due to their adaptability and effective infection of a wide range of cells, Adenoviruses show promise for oncolytic virotherapy. Efficacy may be restricted by pre-existing immunity.
HSV-1	dsDNA, internal helix, enveloped	Targeted oncolytics may be possible for HSV-1 due to their natural affinity for specific cancer cells. Considerations include potential neurotoxicity and pre-existing immunity.
Reoviruses	Naked, icosahedral, dsRNA	Reoviruses exhibit potential as oncolytic virotherapy agents by selectively destroying cancer cells while sparing healthy tissue. Limited tropism for specific cancer types is a consideration.
Poxviruses	Enveloped, Complex, dsDNA	Poxviruses, like vaccinia, with tumor-selective replication, have been adapted for oncolytic virotherapy. Immunogenicity remains a challenge

Virus	Attributes	Oncolytic Treatment for Malignancies
Newcastle Disease Virus	ssRNA, Helical, Enveloped	NDV is being investigated for its potential in cancer treatment, particularly against solid tumors. Limited human research is available.
Measles Virus	ssRNA, helical, enveloped	The measles virus shows promise for oncolytic virotherapy due to its ability to target and infect cancer cells specifically. Pre-existing immunity may limit efficacy.
Seneca Valley Virus	ssRNA, Enveloped, Icosahedral	Seneca Valley virus demonstrates oncolytic activity against neuroendocrine tumors. Limited human safety and efficacy data are available.
HVJ-E Virus	Icosahedral, enveloped, (-)RNA-containing	HVJ-E virus shows promise for oncolytic virotherapy specific to cancer cells. Limited clinical data are available, necessitating further research.
Gamma-Herpes Virus	dsDNA, Icosahedral, enveloped	Certain gamma-herpes viruses have been modified for tumor-selective replication in oncolytic virotherapy. Potential immunogenicity and limited clinical use are considerations.
Parvovirus	ssDNA, naked, icosahedral	Parvovirus is being investigated for its potential to induce cancer cell death and has demonstrated oncolytic potential. Limited human clinical trial data are available.
Retrovirus	ssRNA, icosahedral, enveloped	Retroviruses with selective replication in cancer cells have been adapted for gene therapy and oncolytic virotherapy. Incorporation into the host genome is a concern.
Others (Specify)	Varying traits based on the type of virus	Each specific virus has different properties and oncolytic potential; more investigation and clinical trials are needed for a comprehensive understanding.

Combination Therapy of Oncolytic Viruses (OVs)

Oncolytic viruses (OVs) have shown potential for cancer treatment through direct tumor lysis and modulation of the tumor microenvironment. Combining OVs with other therapeutic approaches may produce synergistic effects, which are discussed in the following sections.

Immune Checkpoint Inhibitors (ICI) and OVs

Immune checkpoint inhibitors (ICIs) have been widely used across various cancer types and have demonstrated clinical effectiveness. ICIs are drugs that target checkpoint molecules to regulate the immune response, with well-known examples targeting CTLA-4 and PD-1 [58–59]. There are two main strategies for combining ICIs with OVs: inserting anti-ICI antibody genes into the OV genome or administering ICIs before or after OV therapy [58].

For example, Shi *et al*., engineered a ΔTK-Armed-VACV encoding anti-PD-1 and anti-4-1BB antibody genes, which activated tumor-specific cytotoxic T lymphocytes in mice [60]. Similarly, combining a tumor-selective oncolytic vaccinia virus with anti-PD-1 or anti-CTLA-4 antibodies enhanced antitumor activity compared to virotherapy alone in murine models [61]. Wang *et al*., developed an OV co-expressing a PD-L1 inhibitor and GM-CSF, which elicited systemic T-cell responses following intratumoral injection [62]. Additionally, an oncolytic vaccinia virus expressing a superagonist IL-15 fusion protein combined with an anti-PD-1 antibody improved outcomes in mouse tumor models [63]. While most studies remain preclinical in vivo, several in vitro studies also support these combination strategies [64–70].

Chemotherapy and OVs

Combining traditional chemotherapy with OVs is another promising approach. In mouse tumor models, the combination of paclitaxel with oncolytic vaccinia virus produced significant therapeutic benefits, achieving 50% complete and durable responses without increasing toxicity [71]. Synergistic antitumor effects were also observed with vaccinia virus combined with cyclophosphamide (CPA) [72]. Most evidence currently comes from in vivo or in vitro studies; however, a 2019 case report described a patient treated with GL-ONC1 in combination with chemotherapy, resulting in decreased CA-125 levels and significant tumor reduction on CT imaging [73].

CAR-T and OVs

Chimeric antigen receptor (CAR)-T cell therapy has emerged as a novel treatment for relapsed or refractory hematological malignancies. CAR-T cells are engineered with chimeric antigen receptors that contain intracellular domains capable of activating immune responses. OVs can be designed to deliver immunostimulatory cytokines, chemokines, or immune checkpoint-targeting molecules, which may enhance CAR-T efficacy [74].

For instance, Watanabe *et al*., demonstrated that OAd-TNFa-IL2, an oncolytic adenovirus expressing TNF-α and IL-2, improved the antitumor activity of meso-CAR T cells in mice by modifying the tumor microenvironment [75]. CAR-T therapy combined with a modified oncolytic vaccinia virus expressing CXCL11 increased both total and antigen-specific T-cell infiltration in tumors and enhanced antitumor efficacy [76]. Additionally, an oncolytic adenovirus expressing a PD-L1 blocking mini-antibody enhanced the effectiveness of HER2.CAR T cells compared to either therapy alone [77]. VanSeggelen *et al*., found that CAR-T cells loaded with low doses of virus maintained their function while successfully delivering the virus to target cells [78]. Despite these promising findings, the evidence for OV and CAR-T combination therapy is still preliminary, and further studies are required to validate its clinical effectiveness.

Future Perspective

Oncolytic virotherapy has evolved from the use of naturally occurring viruses that incidentally reduced tumors to the deliberate engineering of viruses designed to selectively target and kill cancer cells. Modern oncolytic viruses (OVs) are modified to enhance tumor-specific infection, promote efficient viral replication, and maximize anticancer effects. Alterations to the viral capsid can improve cell entry and infection efficiency, while genetic modifications can accelerate replication and increase tumor cell lysis. These innovations allow the virus to act more precisely and effectively within the tumor microenvironment.

Various OV designs have demonstrated promising results across multiple cancer cell lines and animal models, highlighting their potential as standalone therapies. However, most preclinical studies have focused on OVs as monotherapies. There is growing interest in exploring combination therapies, where OVs are used alongside novel immunotherapies, targeted therapies, or conventional treatments such as chemotherapy and radiotherapy. The success of such combinations depends on careful selection of strategies that consider tumor type and location, the nature of the conventional therapy, and the presence of specific biomarkers such as microsatellite instability (MSI), tumor mutational burden (TMB), and PD-L1 expression [74].

Despite encouraging preclinical findings, clinical evidence for OV-based combination therapies remains limited. Further well-designed clinical trials and mechanistic studies are needed to clarify the factors that influence treatment efficacy, identify potential barriers, and determine the real-world applicability of OVs in cancer therapy. Expanding our understanding of these parameters will be critical for translating OV therapies from the laboratory to routine clinical practice.

CONCLUSION

Innovative oncolytic virotherapy represents a cutting-edge approach to cancer treatment, harnessing viruses with diverse structures and mechanisms to selectively eliminate malignant cells while sparing healthy tissue. This approach relies on the unique properties of oncolytic viruses (OVs), which differ in shape, replication cycle, and cellular targeting strategies. Major OVs under investigation include Parvovirus, herpes simplex virus (HSV), Newcastle disease virus (NDV), Reoviruses, Coxsackievirus, and Echoviruses. Each virus has distinct biological characteristics, tropisms, and therapeutic potential, emphasizing the need for extensive preclinical and clinical research to fully understand their efficacy and safety profiles.

OVs exploit the vulnerabilities of cancer cells, particularly defects in signaling pathways and altered metabolic or structural features, to gain entry and replicate preferentially within tumors. Unlike traditional therapies, OVs can recognize noncanonical cell surface receptors, abnormal communication pathways, and microenvironmental conditions unique to malignant tissues, enabling precise targeting. Studying viral entry mechanisms—for example, HSV binding to specific tumor receptors or Coxsackievirus recognition of ICAM-1 and DAF—provides insights into optimizing delivery, enhancing cytotoxicity, and minimizing off-target effects. Visual models and graphics help illustrate these virus-tumor interactions, aiding in the understanding of cancer tropism and viral targeting efficiency.

The field of oncolytic virotherapy is highly dynamic. Research is increasingly focused on developing combination strategies with immunotherapies, targeted drugs, and conventional cancer treatments to maximize therapeutic outcomes. Several oncolytic immunotherapies have been licensed internationally, and novel OVs are being evaluated in clinical trials and approved by regulatory agencies such as the FDA. Market analyses suggest that the oncolytic virotherapy industry is poised for significant growth, driven by the potential of OVs to achieve precise tumor cell killing with limited side effects, cost-effectiveness, and compatibility with other therapies.

As the understanding of viral biology, tumor interactions, and therapeutic design deepens, oncolytic virotherapy has the potential to transform cancer treatment paradigms. With ongoing

innovation and clinical validation, OVs may emerge as a leading modality in oncology, offering a versatile and potent alternative to conventional therapies and existing immunotherapies.

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