

Recombinant Poliovirus

Mechanisms to elicit antiviral immunity, a natural host response to viral pathogen challenge, are of eminent relevance to cancer immunotherapy. "Oncolytic" viruses, naturally existing or genetically engineered viral agents with cell type-specific propagation in malignant cells, were ostensibly conceived for their tumor cytotoxic properties. Yet, their true therapeutic value may rest in their ability to provoke antiviral signals that engage antitumor immune responses within the immunosuppressive tumor microenvironment. Coopting oncolytic viral agents to instigate antitumor immunity is not an easy feat. In the course of coevolution with their hosts, viruses have acquired sophisticated strategies to block inflammatory signals, intercept innate antiviral interferon responses, and prevent antiviral effector responses, e.g., by interfering with antigen presentation and T cell costimulation. The resulting struggle of host innate inflammatory and antiviral responses versus viral immune evasion and suppression determines the potential for antitumor immunity to occur. Moreover, paradigms of early host:virus interaction established in normal immunocompetent organisms may not hold in the profoundly immunosuppressive tumor microenvironment.

In a review, Gromeier et al. explain the mechanisms of recombinant nonpathogenic [poliovirus](#), [PVSRIPO](#), which is currently in phase I clinical trials against recurrent glioblastoma. We focus on an unusual host:virus relationship defined by the simple and cytotoxic replication strategy of poliovirus, which generates inflammatory perturbations conducive to tumor antigen-specific immune priming ¹⁾.

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Gromeier M, Nair SK. Recombinant Poliovirus for Cancer Immunotherapy. *Annu Rev Med*. 2018 Jan 29;69:289-299. doi: 10.1146/annurev-med-050715-104655. PubMed PMID: 29414253; PubMed Central PMCID: PMC6013836.

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