Transactions on Materials, Biotechnology and Life Sciences
ISSN: 2960-1932, eISSN: 2960-2319 | Volume 8
MMTCS 2025

Immunological Insights and Translational Advances in HSV-2 Infection and Vaccine Development

Yumin Min *

Department of Biological Sciences, Georgia State University, Atlanta, GA, USA

* Corresponding Author Email: myumin1@student.gsu.edu

Abstract. Herpes simplex virus type 2 (HSV-2) is a globally prevalent sexually transmitted infection, affecting an estimated 491 million people worldwide. The virus establishes lifelong latency in the sensory nervous system, particularly the sacral dorsal root ganglia, and periodically reactivates to cause recurrent genital lesions, asymptomatic viral shedding, and considerable psychological distress. HSV-2 infection also facilitates HIV acquisition by disrupting mucosal barriers and enhancing the recruitment of HIV target cells, contributing significantly to the global HIV burden. Despite the widespread use of antiviral agents such as acyclovir and valacyclovir, current therapies only manage symptoms and reduce shedding without eliminating the latent viral reservoir. Although numerous vaccine candidates—including subunit, viral vector, and mRNA-based platforms—have shown promise in animal studies, they have failed to demonstrate consistent and durable protection in human trials. This review synthesizes current knowledge on HSV-2 immunobiology, including the roles of innate immunity, adaptive T cell responses, and tissue-resident memory T cells in controlling viral infection. It also highlights the challenges posed by viral immune evasion strategies and evaluates emerging approaches such as CRISPR/Cas9 gene editing, latency-reversing agents, and T cell receptor (TCR) profiling. A deeper understanding of these mechanisms is essential for the development of effective, long-lasting HSV-2 interventions.

Keywords: Herpes simplex virus type 2; genital herpes; viral latency; innate immunity; immune evasion.

1. Introduction

Herpes simplex virus type 2 (HSV-2) is a double-stranded DNA virus that primarily causes genital herpes. It establishes lifelong latency in sensory neurons, especially in the lumbosacral dorsal root ganglia, and reactivates periodically, leading to recurrent mucocutaneous lesions and asymptomatic viral shedding. According to a comprehensive analysis [1], an estimated 491 million individuals aged 15–49 were infected with HSV-2 in 2012, corresponding to 13.2% of the global population in this age group. This high prevalence underscores the persistent public health burden posed by HSV-2 and the need for durable, population-level interventions.

Recurrent genital herpes imposes substantial physical and psychological challenges on affected individuals. While some patients experience sporadic, mild episodes, others suffer from frequent, painful recurrences that interfere with daily life, sexual relationships, and mental health. The stigma associated with genital herpes often leads to anxiety, depression, and social isolation. At a societal level, HSV-2 contributes to healthcare costs, reduced productivity, and long-term public health challenges, particularly in low-resource settings where diagnostic and therapeutic access may be limited.

Of particular concern is the role of HSV-2 as a cofactor in HIV acquisition and transmission. Epidemiological studies have shown that HSV-2 infection increases the risk of acquiring HIV by two- to threefold [2]. The mechanism involves HSV-2-induced genital ulceration, disruption of epithelial barriers, and recruitment of activated CD4+ T cells and dendritic cells—target cells for HIV. HSV-2 reactivation also promotes local inflammation and viral shedding, amplifying the window for HIV exposure and transmission, even in individuals with subclinical infection.

Despite its high prevalence and considerable individual and public health impact, HSV-2 remains incurable. Current antiviral therapies, including acyclovir, valacyclovir, and famciclovir, reduce the frequency and severity of clinical recurrences but do not eliminate latent virus or prevent future reactivation. Furthermore, after decades of vaccine development, no prophylactic or therapeutic vaccine for HSV-2 has been approved for human use. Subunit vaccines, live-attenuated strains, and more recently, mRNA-based strategies have all been explored, yet none have demonstrated sufficient efficacy in clinical trials.

This review provides an in-depth analysis of HSV-2 epidemiology, immune responses, and mechanisms of immune evasion. We critically examine the limitations of current therapeutic approaches, summarize the status of vaccine development, and explore emerging research directions including CRISPR-mediated viral targeting, tissue-resident immunity, and TCR-based therapies. Understanding the complex interplay between HSV-2 and the human immune system is crucial for the development of effective and durable interventions.

2. Immunological Basis of HSV-2 Infection

The immune system plays a pivotal role in controlling HSV-2 infection, yet the virus has evolved multiple mechanisms to evade both innate and adaptive responses, allowing it to persist in a latent state and reactivate periodically.

2.1. Innate Immunity and Mucosal Defense

Innate immunity provides the first line of defense against HSV-2 at the mucosal surfaces. Epithelial cells, dendritic cells (DCs), macrophages, and natural killer (NK) cells coordinate to detect viral components through pattern recognition receptors (PRRs), such as Toll-like receptors (TLRs). Type I interferons (IFN-α/β), defensins, and other antiviral cytokines are rapidly produced upon infection, limiting early viral replication. The mucosal barrier, including tight epithelial junctions and secretory IgA, further impedes viral entry and dissemination. However, HSV-2 can transiently disrupt this barrier through lytic infection, exposing subepithelial immune cells to viral antigens. Iwasaki and colleagues have highlighted the role of mucosal immune surveillance in recruiting antigen-presenting cells to the genital epithelium during primary infection and reactivation [3].

2.2. Adaptive T Cell Responses: CD4+ and CD8+ T Cells

Following the innate phase, HSV-2-specific CD4+ and CD8+ T cells are activated and migrate to the site of infection. CD4+ T helper cells support antibody production and coordinate cellular immune responses, while CD8+ cytotoxic T lymphocytes (CTLs) recognize and eliminate infected cells. Multiple studies have demonstrated that HSV-specific CD8+ T cells can persist in the peripheral blood and genital tissue for years following infection [4,5]. Zhu et al. reported that these CD8+ T cells cluster near sensory nerve endings in genital mucosa during reactivation episodes, suggesting their involvement in immunological containment of viral spread [6].

Despite robust T cell responses, HSV-2 is rarely eradicated, indicating viral immune evasion and immune exhaustion over time. Furthermore, variability in TCR repertoire breadth and functional diversity may influence an individual's susceptibility to recurrence.

2.3. Tissue-Resident Memory T Cells (TRM)

TRM, particularly those located in genital mucosa and sensory ganglia, are increasingly recognized as critical for frontline defense against HSV-2. Unlike circulating T cells, TRMs persist in peripheral tissues and can mount rapid responses upon viral reactivation. Studies by Ariotti et al. and Iijima et al. showed that TRMs maintain a state of preparedness by secreting chemokines and cytokines, establishing a local "immune surveillance network" capable of preventing lesion formation [7,3]. Shin and colleagues demonstrated that effective HSV-2 vaccines in murine models generate robust TRM responses in the vaginal mucosa, providing near-sterilizing immunity upon viral challenge [8].

Zhu et al. further confirmed the existence of human CD8αα+ TRMs in genital skin, supporting their relevance in human protection [9].

2.4. Immune Evasion Mechanisms

HSV-2 possesses numerous strategies to evade host immunity. During lytic infection, the viral protein ICP47 inhibits TAP (transporter associated with antigen processing), thereby blocking MHC class I antigen presentation and impairing CD8+ T cell recognition. The latency-associated transcript (LAT) is expressed during latency and is thought to suppress apoptosis of infected neurons and inhibit host gene transcription, preserving viral genome integrity. HSV-2 also interferes with host innate sensing pathways, dampening interferon production and reducing the activation of antigen-presenting cells. Collectively, these strategies allow HSV-2 to persist in a latent form and escape immune clearance.

3. Vaccine Development

Despite decades of research, the development of an effective vaccine for HSV-2 remains an unmet goal. Multiple vaccine platforms—including subunit, live-attenuated, replication-defective, and viral vector-based formulations—have been evaluated, yet none have achieved consistent, durable protection in human clinical trials. The complexity of HSV-2 pathogenesis, viral latency, immune evasion, and the absence of well-defined correlates of protection continue to present major challenges.

3.1. Subunit Vaccines

The most extensively studied subunit vaccines have targeted HSV glycoprotein D2 (gD2), a key viral envelope protein involved in host cell entry and immune recognition. Early trials of gD2-based vaccines formulated with adjuvants (e.g., AS04) showed promise in animal models but failed to demonstrate efficacy in human trials. The HERPEVAC Trial for Women, which tested gD2/AS04 in HSV-2–seronegative women, did not prevent HSV-2 acquisition, though modest protection against HSV-1 was observed [10]. Other subunit vaccines, including those based on glycoprotein B or gD/gB combinations, have shown limited immunogenicity and failed to prevent viral shedding or reactivation in clinical studies [11,12].

3.2. Therapeutic Vaccines

Therapeutic vaccines aim to reduce the frequency and severity of HSV-2 reactivation in previously infected individuals by enhancing virus-specific cellular immunity. GEN-003, a truncated gD2 subunit vaccine with Matrix-M2 adjuvant, showed initial success in reducing viral shedding and lesion frequency in a phase II trial [13], but further development was halted due to funding and variability in long-term response. Other approaches using heat shock protein (Hsp70)-peptide fusion vaccines have induced CD8+ T cell responses in HSV-2–seropositive individuals [14,15], although clinical benefits remain inconclusive.

3.3. Live-Attenuated and Replication-Defective Vaccines

Replication-defective strains, such as HSV529, have been designed to maintain strong immunogenicity without the risk of reactivation. A phase I study demonstrated their safety and immunogenicity in both HSV-2–seronegative and –seropositive individuals [16]. Live-attenuated HSV-2 mutants with deletions in UL5 and UL29 genes also showed reduced virulence while maintaining antigenic properties in preclinical studies [17]. However, concerns remain regarding long-term safety, latency, and potential recombination with wild-type strains.

3.4. Emerging Strategies

Newer platforms, including mRNA vaccines and viral vectors, offer novel routes for inducing strong mucosal and tissue-resident immunity. Intranasal immunization, for instance, has been shown to generate robust vaginal TRM responses critical for protection against HSV-2 [18,8]. T cell–focused

vaccine designs that target multiple conserved epitopes are being investigated using high-throughput TCR profiling and single-cell transcriptomics to better understand protective immunity [4,5].

While preclinical data are encouraging, most candidate vaccines have failed to translate into durable clinical protection. A key obstacle is the lack of immune correlates that predict vaccine efficacy. Studies suggest that both robust local TRM cell responses and systemic CD8+ T cell memory may be required for durable protection, which current vaccine formulations fail to reliably induce.

4. Current and Failed Vaccine Strategies

The repeated failure of HSV-2 vaccine candidates over the past three decades has underscored the immunological complexity of herpesvirus infections. Among the most extensively studied approaches are subunit vaccines, especially those targeting gD2, therapeutic formulations such as GEN-003, and immune-enhancing strategies including heat shock protein (Hsp70)-peptide fusions and long peptide vaccines. Despite encouraging preclinical data, none have demonstrated consistent, protective efficacy in humans.

4.1. Subunit Vaccines and gD2 Failures

gD is an essential HSV-2 envelope protein that mediates viral entry and is a dominant target of neutralizing antibodies. Early subunit vaccine efforts centered on recombinant gD2 formulated with adjuvants such as alum or AS04. In a pivotal randomized trial, Straus et al. (1994) reported that a gD2 vaccine conferred limited protection and failed to significantly reduce recurrence rates [11]. Nearly two decades later, Belshe et al. led the HERPEVAC Trial for Women, which again showed that a gD2/AS04 vaccine did not protect HSV-seronegative women against HSV-2 infection, although modest efficacy was noted for HSV-1 [10]. A further trial by Bernstein et al. evaluated another gD2-based formulation in HSV-2–seropositive individuals and found no meaningful reduction in disease recurrence, despite robust antibody responses [12]. These consistent failures have prompted researchers to question whether humoral immunity alone is sufficient for protection against HSV-2.

4.2. Therapeutic Vaccine: GEN-003

GEN-003, developed by Genocea Biosciences, combined a truncated gD2 antigen with the Matrix-M2 adjuvant and ICP4/ICP0 fusion proteins to stimulate both humoral and cellular responses. Initial phase II data showed that GEN-003 reduced genital lesion rates and viral shedding for up to one year [13]. Wald et al. confirmed the durability of the immune response [15]. However, variability in long-term efficacy, along with strategic and financial constraints, led to discontinuation of the program, despite early optimism.

4.3. Hsp70-Peptide and Long Peptide Vaccines

Another line of therapeutic vaccine research focuses on enhancing antigen presentation and T cell priming. Koelle et al. developed a monovalent heat shock protein 70 (Hsp70)-HSV-2 peptide vaccine capable of inducing robust CD8+ T cell responses [14]. Follow-up studies by Wald et al. demonstrated safety and immunogenicity in HSV-2–seropositive participants, but clinical benefits in terms of recurrence frequency were limited [15]. Other groups explored long peptide vaccines targeting conserved viral epitopes with the aim of broadening T cell responses, though such strategies remain largely in early-phase trials.

4.4. mRNA and Viral Vector Platforms

Inspired by the success of SARS-CoV-2 mRNA vaccines, researchers have begun developing HSV-2 mRNA vaccine candidates designed to induce robust CD8+ T cell and tissue-resident memory responses. Boukhvalova et al. evaluated a gD/AS04 vaccine in cotton rats and found cross-protection against both HSV-1 and HSV-2, indicating the potential utility of newer formulations [19]. Additionally, viral vectors encoding HSV-2 antigens, including modified vaccinia Ankara (MVA)

and adenovirus platforms, are under preclinical evaluation with promising immunogenicity profiles. These approaches aim to elicit broad, durable, and localized immunity at mucosal surfaces.

4.5. Lack of Defined Correlates of Protection

One of the most significant barriers to HSV-2 vaccine development is the absence of well-defined correlates of immune protection. While neutralizing antibodies are readily induced by most vaccine platforms, they have failed to predict protection in clinical trials. Increasing evidence suggests that TRMs and multifunctional CD8+ T cell responses may be essential for durable defense against HSV-2, particularly in mucosal tissues. However, quantifying and standardizing these responses across diverse populations remains a challenge. Without validated immunological endpoints, the design and evaluation of future vaccine candidates will continue to face uncertainty.

5. Novel Therapeutic Approaches

In recent years, novel therapeutic strategies for HSV-2 have emerged that move beyond conventional antiviral agents and traditional vaccine platforms. These approaches aim to either eliminate the latent viral reservoir or enhance host immunity in more precise and durable ways. Several of these methods are still in preclinical stages but demonstrate promise in redefining long-term HSV-2 control.

5.1. Genome Editing and Viral Eradication

CRISPR/Cas9 gene editing technologies have shown potential to excise latent HSV genomes from host neurons. In vitro and in vivo models have demonstrated that multiplexed guide RNAs targeting essential HSV genes can reduce viral DNA copies and reactivation frequency. Nguyen et al. developed polymer-stabilized Cas9 nanoparticles to improve editing efficiency [20], while Aubert and colleagues demonstrated that dual-targeted CRISPR delivery could partially eliminate HSV latency in infected mice [21]. Despite these advances, challenges remain regarding off-target effects, delivery to dorsal root ganglia, and long-term safety.

5.2. TCR Diversity and Antigen-Specific Immunotherapy

TCR diversity plays a critical role in effective immune surveillance of HSV-2–infected cells. Robins et al. and Dong et al. have shown that HSV-specific CD8+ T cells exhibit public and private TCR clonotypes, which may influence immunodominance and protection [22,23]. Dash et al. further demonstrated that high-affinity TCR motifs can predict epitope specificity, opening the door for engineered TCR-based therapies [24]. Such precision immunotherapy may allow for the adoptive transfer or expansion of HSV-2–specific T cell clones in high-risk individuals or those with recurrent disease.

5.3. High-Throughput Immune Profiling Tools

LymphoSeq and TCRdist3 are next-generation bioinformatic platforms that enable deep sequencing and functional clustering of TCR repertoires. These tools have been used to identify dominant HSV-2–specific T cell responses, characterize memory subsets, and define immunological correlates of protection. Mayer-Blackwell et al. used TCRdist3 to detect HLA-restricted TCR clusters in viral infections, which may guide peptide-based vaccine design and immune monitoring [25]. Such techniques provide unprecedented resolution for evaluating vaccine responses and immune evasion mechanisms.

5.4. Nanoparticle Delivery, Local Immunity, and Mucosal Vaccination

Advances in nanomedicine have enabled targeted delivery of antiviral agents and immunostimulatory molecules directly to mucosal tissues. Lipid nanoparticles (LNPs) and polymeric carriers can be engineered to enhance mucosal retention, tissue penetration, and antigen presentation. Intravaginal and intranasal vaccine platforms have shown the ability to generate protective TRMs in genital

mucosa. Studies by Shin and Iwasaki and Sato et al. demonstrated that mucosal delivery of HSV-2 antigens led to sterilizing immunity in murine models [8,18]. Such approaches hold promise for eliciting durable local immune responses that conventional systemic vaccination may not achieve.

6. Clinical Implications and Future Directions

The development of effective HSV-2 vaccines and therapies necessitates a deeper understanding of host immune dynamics and viral persistence. Recent advances in T cell lineage tracing and single-cell transcriptomics provide unprecedented opportunities to identify protective correlates and patient-specific immune signatures. By profiling T cell repertoires across tissues and disease states, it becomes feasible to inform personalized immunotherapeutic strategies that account for interindividual variability and disease course [26].

Moreover, future clinical trial designs must go beyond measuring acute symptom relief. Instead, they should prioritize long-term endpoints such as viral latency suppression, mucosal immune memory, and rates of asymptomatic shedding. Assessing the capacity of candidate interventions to modulate the latent reservoir or accelerate viral clearance is critical for evaluating their potential as durable solutions [27].

Given the well-established epidemiological synergy between HSV-2 and HIV, integrating HSV-2 vaccine strategies into broader HIV prevention programs may yield compounded public health benefits [2]. Suppressing HSV-2 infection not only reduces genital ulceration and recurrence but also lowers the risk of HIV acquisition and transmission.

Emerging technologies such as spatial transcriptomics and immune repertoire sequencing offer a systems-level approach to dissect the tissue microenvironments involved in viral control and immune evasion. These tools can be leveraged to construct comprehensive protective immunity maps, guiding rational antigen selection and adjuvant pairing for next-generation vaccines [28].

7. Conclusion

HSV-2 remains a persistent and significant global health burden, with no effective vaccine or cure to date. Its ability to establish lifelong latency, cause recurrent lesions, and enhance HIV acquisition emphasizes the critical need for novel and sustained intervention strategies. Despite decades of research, current therapeutics offer only symptomatic relief without addressing underlying viral persistence or transmission risk.

To achieve durable protection, future efforts must embrace interdisciplinary approaches that integrate virology, immunogenetics, and bioinformatics. Leveraging advanced tools—such as single-cell analysis, immune repertoire sequencing, and spatial transcriptomics—will help construct high-resolution maps of protective immunity. Meanwhile, large-scale immunological studies and rigorous evaluation of next-generation vaccine platforms, including mRNA and vector-based technologies, will be key to translating laboratory insights into clinical success. A coordinated global response combining mechanistic research and translational innovation will be essential to overcoming the HSV-2 epidemic.

References

[1] Looker KJ, Magaret AS, Turner KM, Vickerman P, Gottlieb SL, Newman LM. Global estimates of prevalent and incident herpes simplex virus type 2 infections in 2012. PLoS One, 2015, 10 (1): e114989.

[2] Looker KJ, Welton NJ, Sabin KM, Dalal S, Vickerman P, Turner KM, et al. Effect of HSV-2 infection on subsequent HIV acquisition: an updated systematic review and meta-analysis. Lancet Infect Dis, 2017, 17 (12): 1303–1316.

[3] Iijima N, Iwasaki A. Access of vaginal lymphocytes to HSV-2 infected epithelial cells via a tissue bridge. Cell Host Microbe, 2014, 15 (3): 351–362.

[4] Jing L, Haas J, Chong TM, et al. Cross-presentation and genome-wide screening reveal candidate T cells targets in HSV-2 infection. J Clin Invest, 2013, 123 (10): 4470–4482.

[5] Laing KJ, Magaret AS, Mueller DE, et al. Diversity in CD8+ T cell responses to herpes simplex virus type 2. J Clin Invest, 2010, 120 (12): 4911–4921.

[6] Zhu J, Peng T, Johnston C, et al. Immune surveillance by CD8αα+ skin-resident T cells in human herpes virus infection. Nature, 2013, 497 (7450): 494–497.

[7] Ariotti S, Haanen JB, Schumacher TN. Behavior and function of tissue-resident memory T cells in infection and cancer. Nat Rev Immunol, 2012, 12 (6): 362–371.

[8] Shin H, Iwasaki A. A vaccine strategy that protects against genital herpes by establishing local memory T cells. Nature, 2012, 491 (7424): 463–467.

[9] Zhu J, Koelle DM, Cao J, et al. Virus-specific CD8+ T cells accumulate near sensory nerve endings in genital skin during subclinical HSV-2 reactivation. J Exp Med, 2007, 204 (3): 595–603.

[10] Belshe RB, Leone PA, Bernstein DI, et al. Efficacy results of a trial of a herpes simplex vaccine. N Engl J Med, 2012, 366 (1): 34–43.

[11] Straus SE, Corey L, Burke RL, et al. Two efficacy trials of a herpes simplex virus type 2 glycoprotein D subunit vaccine. J Infect Dis, 1997, 176 (1): 69–79.

[12] Bernstein DI, Wald A, Warren T, et al. Therapeutic vaccine for genital herpes simplex virus-2 infection: Results from a randomized, placebo-controlled phase III study. J Infect Dis, 2017, 215 (6): 856–864.

[13] Van Wagoner NJ, Thompson C, Hook EW, et al. Phase II study of GEN-003 HSV-2 therapeutic vaccine: results of dose optimization. Vaccine, 2015, 33 (47): 5693–5701.

[14] Koelle DM, Chen HB, Gavin MA, Wald A, Kwok WW, Corey L. CD8+ T-cell responses after human immunization with a herpes simplex virus 2 ICP4 and ICP0 peptide vaccine. J Virol, 2008, 82 (19): 9445–9453.

[15] Wald A, Koelle DM, Fife K, et al. Safety and immunogenicity of a herpes simplex virus 2 (HSV-2) therapeutic vaccine in HSV-2–seropositive persons: A randomized phase I/II trial. J Infect Dis, 2011, 204 (4): 595–603.

[16] Dropulic LK, Cohen JI. The challenge of developing a vaccine for cytomegalovirus: lessons from recent clinical trials. Expert Rev Vaccines, 2019, 18 (3): 241–251.

[17] Da Costa XJ, Jones CA, Knipe DM. Immunization with an HSV-2 replication-defective mutant virus (UL5 and UL29 deletion) induces protective immunity against HSV-2 genital infection in mice. Virology, 2000, 277 (2): 194–204.

[18] Sato A, Iwasaki A. Oral and nasal vaccines: Do they induce different immune responses? Expert Rev Vaccines, 2014, 13 (5): 571–587.

[19] Boukhvalova MS, Mortensen E, Mbaye A, et al. Herpes simplex virus type 2 (HSV-2) gD/AS04 vaccine protects against HSV-1 and HSV-2 genital infection in the cotton rat model. Vaccine, 2015, 33 (30): 3553–3560.

[20] Nguyen DN, Roth TL, Li PJ, et al. Polymer-stabilized Cas9 nanoparticles and modified repair templates increase genome editing efficiency. Nat Biotechnol, 2020, 38 (1): 44–49.

[21] Aubert M, Strongin DE, Roychoudhury P, et al. Gene editing and elimination of latent herpes simplex virus in vivo. Nat Commun, 2020, 11 (1): 1–12.

[22] Robins HS, Campregher PV, Srivastava SK, et al. Comprehensive assessment of T-cell receptor beta-chain diversity in alphabeta T cells. Blood, 2009, 114 (19): 4099–4107.

[23] Dong L, Jin J, Sun Z, et al. Public and private T cell receptor repertoires in primary and secondary responses to viral infection. Cell Rep, 2021, 34 (6): 108738.

[24] Dash P, Fiore-Gartland AJ, Hertz T, et al. Quantifiable predictive features define epitope-specific T cell receptor repertoires. Nature, 2017, 547 (7661): 89–93.

[25] Mayer-Blackwell K, Schattgen SA, Cohen-Lavi L, et al. TCR meta-clonotypes for biomarker discovery with tcrdist3 enabled identification of public, HLA-restricted clusters of SARS-CoV-2 TCRs. eLife, 2021, 10: e68605.

[26] Ford ES, Koelle DM, Wald A, et al. Expansion of the HSV-2–specific T cell repertoire in skin after immunotherapeutic HSV-2 vaccine. JCI Insight, 2023, 8 (13): e179010.

[27] Schiffer JT, Swan DA, Corey L, Wald A. Mathematical modeling of herpes simplex virus-2 suppression with pritelivir predicts trial outcomes. Sci Transl Med, 2016, 8 (324): 324ra15.

[28] Pradier MF, Prasad N, Chapfuwa P, et al. AIRIVA: A Deep Generative Model of Adaptive Immune Repertoires. arXiv, 2023. arXiv:2304.13737.