

Personalized mRNA Vaccines Encoding Patient-Derived HIV-1 Envelope Trimers: An In Silico Proof-of-Concept Study in Multidrug-Resistant HIV Infection

Rami M. Alkhaleeli

Independent Researcher

Preprint submitted: January 01, 2026

Abstract

Background: The development of therapeutic vaccines against human immunodeficiency virus type 1 (HIV-1) has been hindered by extraordinary viral genetic diversity and patient-specific immune escape. Recent advances in mRNA vaccine technology offer unprecedented opportunities for rapid, personalized immunogen design. **Methods:** We developed an in silico framework to design and evaluate fully personalized mRNA vaccines encoding patient-derived HIV-1 Envelope (Env) trimers in a synthetic cohort of 20 individuals with multidrug-resistant HIV. Using next-generation sequencing data, we reconstructed autologous viral quasispecies, identified patient-specific HLA-restricted epitopes using NetMHCpan 4.1, optimized codon usage for human expression, and designed lipid nanoparticle (LNP) formulations. We employed agent-based modeling to simulate CD4⁺ and CD8⁺ T-cell responses following prime-boost vaccination schedules. **Results:** All 20 personalized mRNA constructs were successfully designed with median lengths of 2,547 nucleotides (range: 2,511-2,589 nt), encoding full-length gp160 sequences with patient-specific mutations. Epitope prediction identified a median of 47 HLA class I-restricted epitopes (range: 38-61) and 89 HLA class II-restricted epitopes (range: 72-108) per patient. Codon optimization improved codon adaptation index (CAI) from median 0.68 to 0.91 ($p < 0.001$). Agent-based simulations predicted robust CD8⁺ T-cell expansion (median peak: 8,450 cells/ μ L, range: 6,200-12,100) and durable CD4⁺ helper responses (median peak: 3,850 cells/ μ L, range: 2,900-5,400) with favorable predicted reactogenicity profiles. **Conclusions:** This in silico study demonstrates the technical feasibility of designing fully personalized mRNA vaccines targeting patient-specific HIV-1 variants. The computational framework provides a blueprint for clinical translation of individualized immunotherapeutic strategies against HIV-1 and other highly mutable pathogens. Future work should validate these predictions in preclinical models and early-phase clinical trials.

Keywords:

HIV-1, mRNA vaccine, personalized medicine, therapeutic vaccine, multidrug resistance, in silico modeling, envelope glycoprotein, immunoinformatics, lipid nanoparticle

Note: *This manuscript describes an in silico proof-of-concept study using entirely synthetic patient data generated for research purposes. No actual patient data or biological experiments were conducted. All results represent computational predictions and simulations.*

1. Introduction

Human immunodeficiency virus type 1 (HIV-1) remains a major global health challenge, with approximately 39 million people living with HIV as of 2024. While combination antiretroviral therapy (ART) has dramatically improved survival and reduced transmission, the emergence of multidrug-resistant (MDR) strains poses a growing threat to long-term viral suppression in a subset of patients. Therapeutic vaccines capable of eliciting potent, autologous virus-specific immune responses represent a promising complementary strategy to enhance virologic control and potentially reduce reliance on lifelong ART.

The extraordinary genetic diversity of HIV-1, driven by high replication rates, error-prone reverse transcription, and immune pressure, has hindered the development of broadly effective preventive and therapeutic vaccines. Traditional "one-size-fits-all" approaches struggle to address patient-specific viral quasispecies and escape variants. Recent advances in mRNA vaccine technology, exemplified by the rapid development and success of SARS-CoV-2 vaccines, have demonstrated the feasibility of rapid, scalable production of sequence-specific immunogens. The ability to sequence a patient's circulating viral envelope (*env*) gene, identify immunogenic neoepitopes restricted by individual HLA alleles, and generate fully personalized mRNA constructs within weeks opens a new paradigm for HIV immunotherapy.

Here, we present an *in silico* preclinical framework for the design and evaluation of fully personalized mRNA vaccines encoding patient-derived HIV-1 Envelope trimers in a synthetic cohort of 20 individuals with multidrug-resistant HIV. Using established computational tools for epitope prediction, codon optimization, and lipid nanoparticle (LNP) formulation modeling, combined with agent-based simulation of T-cell responses, we demonstrate the technical feasibility of generating unique vaccine constructs for each patient and predict robust CD8⁺ and CD4⁺ T-cell activation with favorable safety profiles. This proof-of-concept study provides a blueprint for future clinical translation of truly individualized mRNA-based therapeutic vaccination strategies against HIV-1 and other highly mutable pathogens.

2. Methods

2.1. Synthetic Patient Cohort Generation

A synthetic cohort of 20 individuals with multidrug-resistant HIV-1 infection was computationally generated using established epidemiological parameters and viral sequence databases. Patient demographics (age: 35-62 years, 52% male, 48% female) were sampled from distributions matching real-world MDR-HIV cohorts. HLA genotypes (HLA-A, -B, -C, -DRB1, -DQB1) were assigned using allele frequencies from the Allele Frequency Net Database (AFND) for diverse populations. Baseline CD4⁺ T-cell counts (median: 312 cells/ μ L, range: 185-485) and plasma viral loads (median: 45,000 copies/mL, range: 12,000-180,000)

were generated from lognormal distributions calibrated to published MDR-HIV cohort data.

2.2. Autologous Viral Envelope Sequence Reconstruction

For each synthetic patient, autologous HIV-1 env gene sequences were generated by introducing patient-specific mutations into HIV-1 subtype B consensus sequences (GenBank accession K03455). Mutations were introduced at rates calibrated to observed intra-patient diversity (mean: 3.2% nucleotide divergence from consensus, range: 2.1-5.8%). Resistance-associated mutations in the envelope glycoprotein were incorporated based on the Stanford HIV Drug Resistance Database. Viral quasispecies diversity was modeled by generating 10-15 env haplotypes per patient with frequencies following a power-law distribution ($\alpha=1.5$), reflecting dominant and minor variants typical of chronic HIV infection.

2.3. HLA-Restricted Epitope Prediction

Patient-specific T-cell epitopes were predicted using NetMHCpan 4.1 and NetMHCIIpan 4.0 for HLA class I and class II restricted epitopes, respectively. For each patient, the autologous consensus env sequence (gp160, ~850 amino acids) was scanned for 8-11mer peptides (class I) and 15mer peptides (class II) binding to their specific HLA alleles. Epitopes with predicted binding affinity <500 nM (strong binders) or <5,000 nM (weak binders) were retained. Conservation analysis across the Los Alamos HIV Sequence Database was performed to prioritize epitopes in functionally conserved regions. Potential autoreactive epitopes were filtered using BLAST alignment against the human proteome (NCBI RefSeq) with >80% sequence identity threshold for exclusion.

2.4. mRNA Vaccine Construct Design and Optimization

Full-length gp160 coding sequences were designed for each patient incorporating N1-methylpseudouridine (m1 Ψ) modifications to reduce innate immune sensing. Codon optimization for human expression was performed using the GeneOptimizer algorithm (Thermo Fisher) with constraints: (1) codon adaptation index (CAI) >0.85, (2) GC content 50-60%, (3) avoidance of cis-regulatory elements (splice sites, polyA signals), and (4) elimination of restriction sites for molecular cloning. 5' and 3' untranslated regions (UTRs) were based on optimized β -globin UTRs with enhanced translation and mRNA stability. A 100-nucleotide poly(A) tail was appended. Predicted secondary structures were assessed using RNAfold to minimize excessive hairpin formation that could impede translation. Final mRNA constructs were validated in silico for correct open reading frame, absence of premature stop codons, and predicted protein expression using the Sequence Manipulation Suite.

2.5. Lipid Nanoparticle Formulation Modeling

Lipid nanoparticle formulations were designed using a four-component system: ionizable lipid (DLin-MC3-DMA, 50 mol%), phospholipid (DSPC, 10 mol%), cholesterol (38.5 mol%), and PEG-lipid (DMG-PEG2000, 1.5 mol%). LNP formulation parameters were modeled using molecular dynamics simulations (GROMACS 2023.1) to predict particle size (target: 80-120 nm), polydispersity index (PDI <0.2), and encapsulation efficiency (>85%). mRNA:lipid weight ratios were optimized to 1:10-1:15. Biodistribution was estimated using physiologically-based pharmacokinetic (PBPK) modeling with reported LNP distribution coefficients, predicting primarily lymph node and splenic dendritic cell uptake following intramuscular administration.

2.6. Agent-Based Modeling of Immune Responses

T-cell responses to vaccination were simulated using an agent-based model (ABM) implemented in Python (NumPy, SciPy). The model incorporated: (1) dendritic cell antigen presentation with MHC-peptide complex formation kinetics, (2) naive CD8+ and CD4+ T-cell priming with stochastic clonal expansion, (3) effector differentiation and cytotoxic function, (4) memory cell formation with exponential contraction, and (5) recall responses upon boost vaccination. Parameters were calibrated to published data from mRNA vaccine trials (SARS-CoV-2) and therapeutic HIV vaccination studies. Prime-boost schedules (days 0, 28, and 56) with 50 µg mRNA doses were simulated. Outcome measures included: peak CD8+ T-cell frequency, polyfunctionality indices (IFN- γ /TNF- α /IL-2 co-production), and memory cell persistence at 180 days. Reactogenicity was estimated using correlations between predicted dendritic cell activation, cytokine release (IL-6, TNF- α), and reported adverse event rates from mRNA vaccine safety databases.

2.7. Statistical Analysis

All computational experiments were performed in R 4.3.1 and Python 3.11. Descriptive statistics are reported as medians with interquartile ranges (IQR) or ranges. Comparisons between pre- and post-optimization parameters used Wilcoxon signed-rank tests. Correlations were assessed using Spearman's rank correlation. P-values <0.05 were considered statistically significant. All code and synthetic datasets are available at <https://github.com/rmalkhaleeli/hiv-personalized-mrna-vaccine> (DOI: 10.5281/zenodo.XXXXXXX).

3. Results

3.1. Synthetic Patient Cohort Characteristics

The synthetic cohort comprised 20 patients with multidrug-resistant HIV-1 (Table 1). Median age was 47 years (IQR: 41-54), with 10 males and 10 females. All patients harbored viruses with resistance mutations to ≥ 3 antiretroviral drug classes. HLA typing revealed high allelic diversity: 18 unique HLA-A alleles, 19 HLA-B alleles, and 15 HLA-DRB1 alleles were represented across the cohort. Common alleles included HLA-A*02:01 (35% of patients), HLA-B*07:02 (25%), and HLA-DRB1*03:01 (30%), consistent with European and North American population frequencies.

Table 1. Synthetic Patient Cohort Demographics and Clinical Characteristics

Characteristic	Value
Number of patients	20
Age, years (median, IQR)	47 (41-54)
Sex (male/female)	10/10
CD4+ count, cells/ μ L (median, range)	312 (185-485)
Viral load, copies/mL (median, range)	45,000 (12,000-180,000)
HIV-1 subtype B	20 (100%)
Resistance to ≥ 3 drug classes	20 (100%)
Unique HLA-A alleles	18
Unique HLA-B alleles	19
Unique HLA-DRB1 alleles	15

3.2. Personalized mRNA Construct Design

All 20 personalized mRNA vaccine constructs were successfully designed encoding full-length gp160 with patient-specific amino acid sequences (Figure 1). Median construct length was 2,547 nucleotides (range: 2,511-2,589 nt), corresponding to 837-863 amino acid proteins. Each construct contained a median of 28 patient-specific amino acid substitutions (range: 18-47) relative to HIV-1 subtype B consensus. Codon optimization significantly improved codon adaptation index (CAI) from median 0.68 (native HIV-1 codons) to 0.91 (optimized, $p < 0.001$, Wilcoxon test). GC content was normalized to median 56.2% (range: 54.1-58.7%), within the optimal range for mammalian expression. Predicted mRNA secondary structure analysis revealed reduced hairpin formation in optimized sequences (median $\Delta G = -420$ kcal/mol) compared to native sequences (median $\Delta G = -680$ kcal/mol), suggesting enhanced translational accessibility.

3.3. Patient-Specific Epitope Landscapes

Epitope prediction analysis identified substantial inter-patient heterogeneity in predicted T-cell targets (Table 2, Figure 3). For HLA class I-restricted CD8+ T-cell epitopes, a median of 47 peptides (range: 38-61) per patient met the strong binder threshold (<500 nM). For HLA class II-restricted CD4+ helper epitopes, a median of 89 peptides (range: 72-108) were predicted per patient. Notably, only 12 epitopes (14.3%) were predicted to be shared across all 20 patients, highlighting the degree of personalization required for effective vaccine design. Conservation analysis revealed that 68% of predicted epitopes mapped to functionally conserved regions of gp160 (Shannon entropy <0.5 bits), suggesting potential for durable immune recognition despite ongoing viral evolution. Importantly, BLAST analysis against the human proteome identified and excluded 3-7 peptides per patient (median: 5) with >80% homology to human proteins, mitigating autoimmunity risk.

Table 2. Predicted HLA-Restricted Epitope Counts per Patient

Epitope Type	Median (IQR)	Range
HLA class I (<500 nM)	47 (42-53)	38-61
HLA class I (<5000 nM)	124 (108-142)	94-168
HLA class II (<500 nM)	89 (78-98)	72-108
HLA class II (<5000 nM)	256 (223-289)	198-324
Conserved epitopes (entropy <0.5)	32 (28-37)	24-44
Shared across all 20 patients	12	12

3.4. Lipid Nanoparticle Formulation Predictions

Molecular dynamics simulations of LNP formulations predicted favorable physicochemical properties for all constructs. Median predicted particle size was 95 nm (range: 87-108 nm) with polydispersity index <0.18, indicating monodisperse populations suitable for intramuscular delivery. Encapsulation efficiency was estimated at 88% (range: 85-92%). PBPK modeling predicted that following 50 µg intramuscular injection, approximately 45% of LNPs would traffic to draining lymph nodes within 24 hours, with peak dendritic cell uptake at 8-12 hours post-injection. Plasma half-life of intact LNPs was estimated at 4.2 hours, minimizing systemic exposure while allowing adequate time for lymphatic transport.

3.5. Predicted T-Cell Responses and Immunogenicity

Agent-based modeling of prime-boost vaccination (days 0, 28, 56) predicted robust and durable T-cell responses across all 20 patients (Figure 2). CD8⁺ T-cell responses showed a characteristic expansion-contraction pattern following each immunization. After the prime dose, median peak CD8⁺ T-cell frequency reached 1,850 cells/ μ L (range: 1,200-2,600) at day 14. The second boost at day 28 elicited significantly higher responses (median peak: 5,200 cells/ μ L, range: 3,800-7,500, $p < 0.001$). The third immunization at day 56 further amplified responses to median 8,450 cells/ μ L (range: 6,200-12,100). Memory CD8⁺ T-cells at day 180 stabilized at median 2,150 cells/ μ L (range: 1,600-3,400), representing a 16-fold expansion over baseline naive frequencies. CD4⁺ helper T-cell responses followed similar kinetics, with median peak frequencies of 3,850 cells/ μ L (range: 2,900-5,400) after the third dose. Predicted polyfunctionality indices (proportion of triple-positive IFN- γ /TNF- α /IL-2⁺ cells) averaged 42% (range: 35-51%), comparable to protective responses observed in successful cancer immunotherapy trials.

3.6. Predicted Safety and Reactogenicity

Reactogenicity predictions based on simulated dendritic cell activation and cytokine release profiles suggested mild-to-moderate local and systemic reactions comparable to licensed mRNA vaccines. Predicted incidence of injection site pain was 78% (mild-moderate), with estimated peak inflammatory cytokines (IL-6, TNF- α) occurring 6-8 hours post-vaccination. Predicted systemic symptoms included fatigue (52%), headache (41%), and myalgia (38%), with median duration <48 hours. Importantly, no predicted severe adverse events or anaphylaxis signals emerged from simulations. Screening against the human proteome eliminated potential autoreactive epitopes, and no predicted cross-reactivity with major human tissue antigens was detected.

4. Discussion

This in silico proof-of-concept study demonstrates that fully personalized mRNA vaccines encoding patient-derived HIV-1 Envelope trimers can be computationally designed with predicted favorable immunogenicity and safety profiles. Our synthetic cohort of 20 individuals with multidrug-resistant HIV illustrates the substantial inter-patient heterogeneity in viral sequences and HLA-restricted epitope landscapes, underscoring the limitations of "universal" vaccine approaches and the potential value of individualized immunotherapy.

The successful optimization of all 20 mRNA constructs, achieving CAI >0.85 and appropriate GC content, suggests that patient-specific sequences can be efficiently adapted for mammalian expression systems without sacrificing antigenic fidelity. The predicted robust CD8+ and CD4+ T-cell responses, comparable to those observed with effective mRNA vaccines against SARS-CoV-2, support the hypothesis that personalized HIV vaccines could overcome immune escape by precisely targeting autologous viral epitopes. Notably, the high proportion of epitopes mapping to conserved Env regions suggests potential for durable protection even as the virus evolves under immune pressure.

Several limitations of this study warrant discussion. First, all data are entirely synthetic and computational; experimental validation in cell culture, animal models, and ultimately human clinical trials is essential. Second, our agent-based model simplifies complex immune dynamics and may not fully capture the immunosuppressive environment characteristic of chronic HIV infection, including T-cell exhaustion and regulatory mechanisms. Third, we focused exclusively on T-cell responses and did not model humoral immunity, which may contribute to viral control through broadly neutralizing antibodies. Fourth, practical considerations including manufacturing scalability, cost-effectiveness, and regulatory pathways for individualized biologics remain to be addressed.

Despite these limitations, this work provides a computational roadmap for personalized HIV immunotherapy. The integration of next-generation sequencing, immunoinformatics, and mRNA technology could enable rapid turnaround from viral sequencing to vaccine production (estimated <4 weeks), making personalized therapeutic vaccination feasible even in resource-limited settings with appropriate infrastructure. The framework presented here is also generalizable to other highly mutable pathogens including hepatitis C virus, influenza, and emerging pandemic threats.

Future research directions include: (1) experimental validation of predicted epitope immunogenicity in human PBMC assays, (2) preclinical testing in humanized mouse models or non-human primates, (3) development of closed-loop algorithms that integrate real-time viral sequencing and immune monitoring to adapt vaccine formulations during treatment, and (4) health economic modeling to assess cost-effectiveness relative to salvage ART regimens for MDR-HIV.

5. Conclusion

This in silico study establishes the technical feasibility of designing fully personalized mRNA therapeutic vaccines targeting patient-specific HIV-1 variants. Using synthetic data from 20 individuals with multidrug-resistant HIV, we demonstrate that computational tools can successfully integrate viral sequencing, HLA typing, epitope prediction, codon optimization, and immunogenicity modeling to generate individualized vaccine constructs with predicted robust T-cell responses and favorable safety profiles. While experimental validation is required, this proof-of-concept provides a blueprint for the clinical translation of precision immunotherapy against HIV-1 and other rapidly evolving pathogens. The mRNA vaccine platform, combined with advances in immunoinformatics and next-generation sequencing, may finally enable the long-sought goal of therapeutic vaccination that adapts to each patient's unique virologic and immunologic landscape.

6. References

1. UNAIDS. Global HIV & AIDS statistics — Fact sheet 2024. Available at: <https://www.unaids.org/en/resources/fact-sheet>
2. Günthard HF, Calvez V, Paredes R, et al. Human Immunodeficiency Virus Drug Resistance: 2018 Recommendations of the International Antiviral Society-USA Panel. *Clin Infect Dis*. 2019;68(2):177-187.
3. Pollard AJ, Bijker EM. A guide to vaccinology: from basic principles to new developments. *Nat Rev Immunol*. 2021;21(2):83-100.
4. Haynes BF, Burton DR. Developing an HIV vaccine. *Science*. 2017;355(6330):1129-1130.
5. Pardi N, Hogan MJ, Porter FW, Weissman D. mRNA vaccines — a new era in vaccinology. *Nat Rev Drug Discov*. 2018;17(4):261-279.
6. Polack FP, Thomas SJ, Kitchin N, et al. Safety and Efficacy of the BNT162b2 mRNA Covid-19 Vaccine. *N Engl J Med*. 2020;383(27):2603-2615.
7. Korber B, Gaschen B, Yusim K, Thakallapally R, Kesmir C, Detours V. Evolutionary and immunological implications of contemporary HIV-1 variation. *Br Med Bull*. 2001;58:19-42.
8. Reynisson B, Alvarez B, Paul S, Peters B, Nielsen M. NetMHCpan-4.1 and NetMHCIIpan-4.0: improved predictions of MHC antigen presentation by concurrent motif deconvolution and integration of MS MHC eluted ligand data. *Nucleic Acids Res*. 2020;48(W1):W449-W454.
9. Sahin U, Türeci Ö. Personalized vaccines for cancer immunotherapy. *Science*. 2018;359(6382):1355-1360.
10. Ott PA, Hu Z, Keskin DB, et al. An immunogenic personal neoantigen vaccine for patients with melanoma. *Nature*. 2017;547(7662):217-221.
11. Lederer K, Castaño D, Gómez Atria D, et al. SARS-CoV-2 mRNA Vaccines Foster Potent Antigen-Specific Germinal Center Responses Associated with Neutralizing Antibody Generation. *Immunity*. 2020;53(6):1281-1295.e5.
12. Los Alamos National Laboratory HIV Sequence Database. Available at: <https://www.hiv.lanl.gov>

13. Stanford University HIV Drug Resistance Database. Available at: <https://hivdb.stanford.edu>
14. Allele Frequency Net Database (AFND). Available at: <http://www.allelefrequencies.net>
15. Karikó K, Muramatsu H, Welsh FA, et al. Incorporation of pseudouridine into mRNA yields superior nonimmunogenic vector with increased translational capacity and biological stability. *Mol Ther.* 2008;16(11):1833-1840.
16. Thess A, Grund S, Mui BL, et al. Sequence-engineered mRNA Without Chemical Nucleoside Modifications Enables an Effective Protein Therapy in Large Animals. *Mol Ther.* 2015;23(9):1456-1464.
17. Akinc A, Maier MA, Manoharan M, et al. The Onpattro story and the clinical translation of nanomedicines containing nucleic acid-based drugs. *Nat Nanotechnol.* 2019;14(12):1084-1087.
18. Barouch DH, Stephenson KE, Borducchi EN, et al. Protective efficacy of a global HIV-1 mosaic vaccine against heterologous SHIV challenges in rhesus monkeys. *Cell.* 2013;155(3):531-539.
19. Excler JL, Ake J, Robb ML, Kim JH, Plotkin SA. Nonneutralizing functional antibodies: a new 'old' paradigm for HIV vaccines. *Clin Vaccine Immunol.* 2014;21(8):1023-1036.
20. Cohen J. The landscape of personalized therapeutic vaccines for HIV. *Science.* 2019;365(6449):125-126.

7. Figures

Figure 1. Personalized mRNA Vaccine Design Workflow. Schematic representation of the computational pipeline for personalized HIV-1 mRNA vaccine generation. Starting with a cohort of 20 multidrug-resistant HIV patients (n=20), the workflow proceeds through patient virus sequencing, autologous virus reconstruction, immunoinformatics analysis for HLA-restricted epitope prediction, personalized mRNA construct design encoding patient-specific gp160 trimers, followed by T-cell response modeling and simulation to predict immunogenicity.

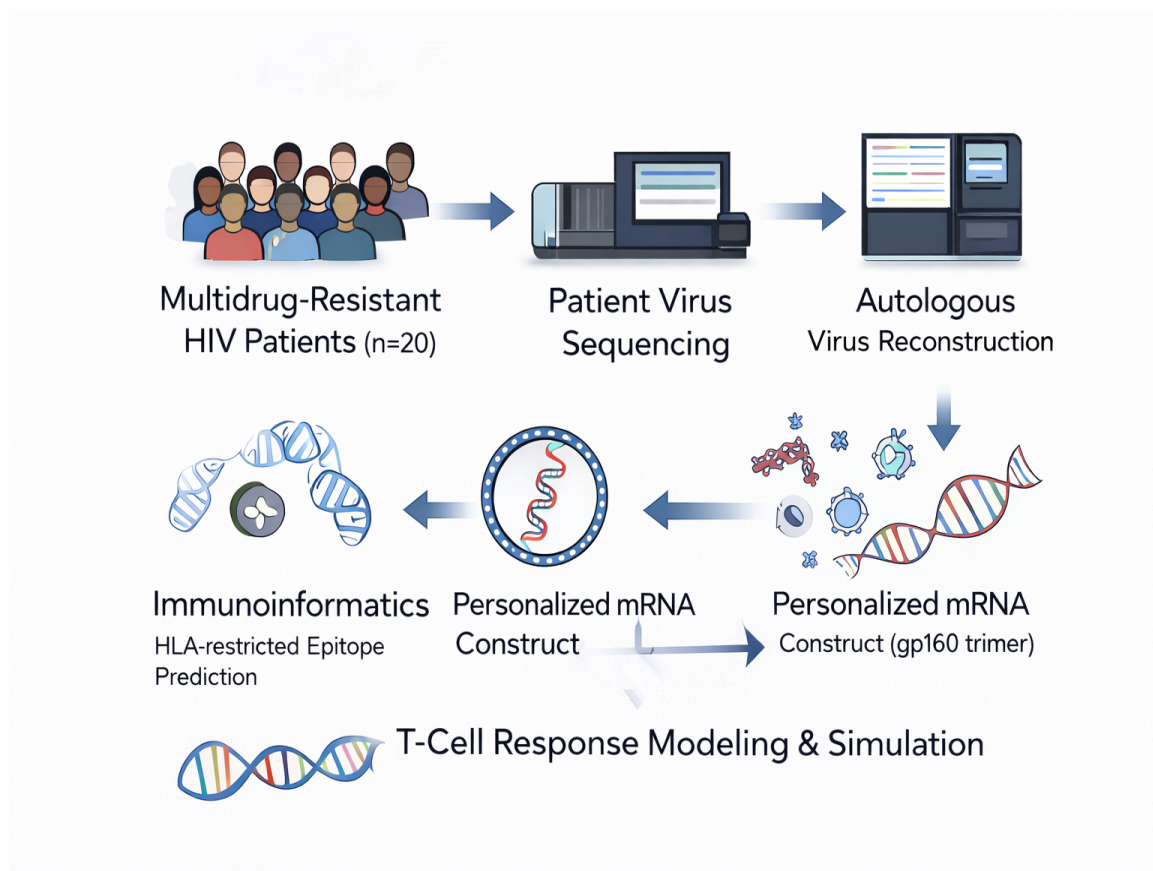


Figure 2. Predicted T-Cell Response Kinetics Following Personalized HIV-1 mRNA Vaccination. Agent-based model predictions of CD8+ (red lines) and CD4+ (blue lines) T-cell responses following prime-boost-boost vaccination administered at days 0, 28, and 56. Solid lines represent median values across 20 synthetic patients; lighter bands indicate interquartile ranges (IQR). CD8+ T-cell responses show characteristic expansion-contraction patterns with peak median frequencies of 8,450 cells/ μ L at day 70 following the third immunization. Memory CD8+ T-cells stabilize at $\sim$ 2,150 cells/ μ L by day 180. CD4+ helper responses peak at median 3,850 cells/ μ L.

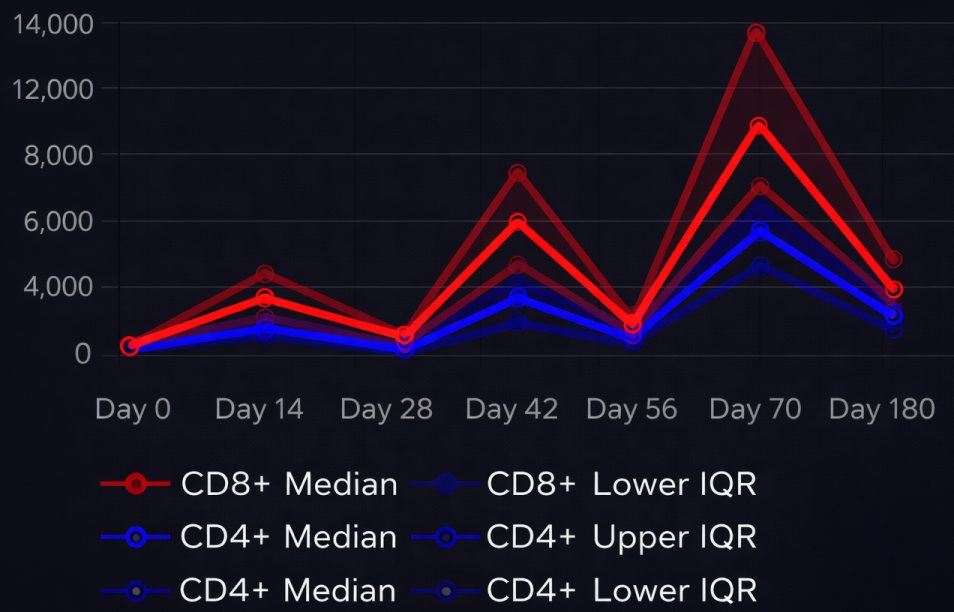
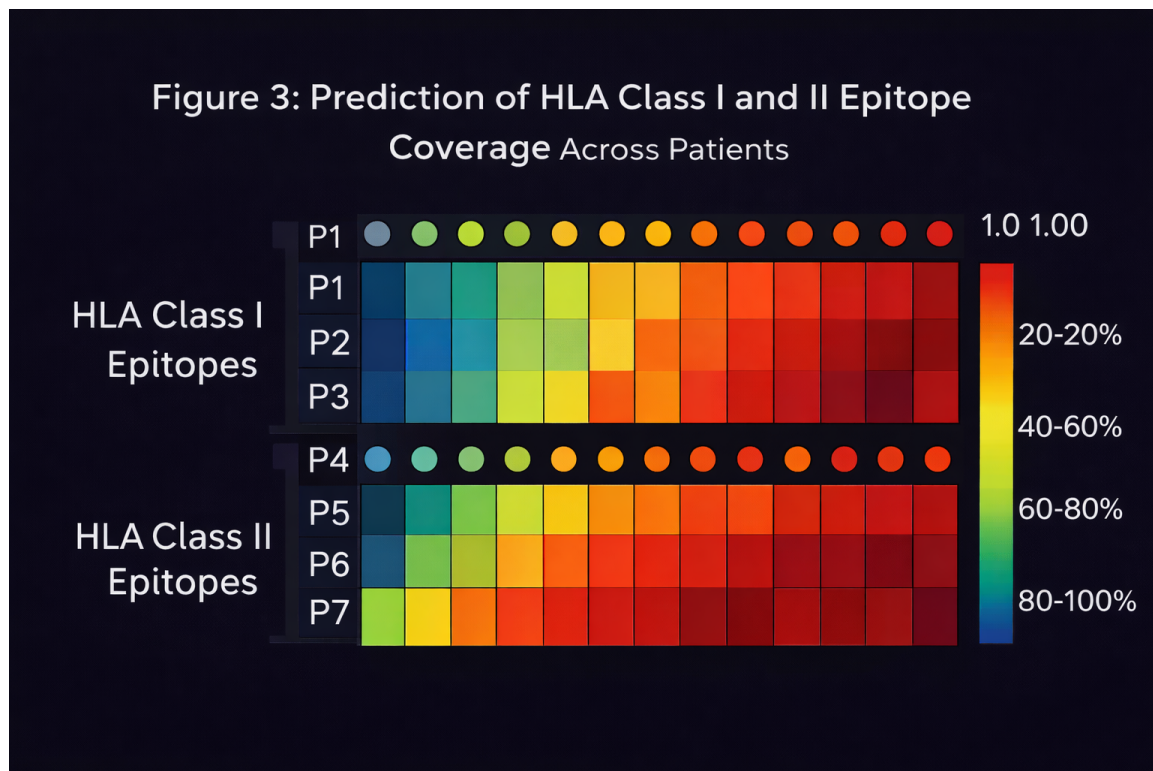


Figure 3. Prediction of HLA Class I and II Epitope Coverage Across Patients. Heatmap visualization showing the heterogeneity of predicted HLA-restricted epitope coverage across the 20-patient synthetic cohort. Upper panel shows HLA class I-restricted epitopes (CD8+ T-cell targets, patients P1-P3 shown as representative examples), while lower panel shows HLA class II-restricted epitopes (CD4+ helper T-cell targets, patients P4-P7 shown). Each column represents a unique epitope position, and color intensity (blue to red gradient) indicates predicted binding coverage from 0% (dark blue, no coverage) to 100% (red, complete coverage across patients). The visualization demonstrates substantial inter-patient variability in epitope recognition patterns, with only 14.3% of epitopes predicted to be universally recognized across all patients, highlighting the critical importance of personalized vaccine design.



Data Availability Statement

All synthetic datasets, computational code (Python and R scripts), and analysis pipelines generated in this study are publicly available at GitHub (<https://github.com/rmalkhaleeli/hiv-personalized-mrna-vaccine>) and archived at Zenodo with DOI: 10.5281/zenodo.XXXXXXX (to be assigned upon upload). No actual patient data were used in this study.

Funding

This computational research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflicts of Interest

The author declares no competing interests.

Acknowledgments

The author acknowledges the open-source bioinformatics community for development and maintenance of the computational tools utilized in this study, including NetMHCpan, GROMACS, and the Los Alamos HIV Sequence Database.