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Immunoinformatic Prediction of Broadly Reactive T-Cell Epitopes for Universal Cancer Vaccines

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Abstract

Background: *The development of effective cancer vaccines is hindered by extensive tumor heterogeneity and human leukocyte antigen (HLA) diversity, which limit the scalability and population-wide applicability of personalized vaccine approaches. Universal cancer vaccines targeting conserved tumor antigens represent a promising alternative; however, systematic identification of broadly reactive T-cell epitopes remains a major challenge.*

Methods and Results: *In this study, we employed an immunoinformatic approach to predict and prioritize T-cell epitopes suitable for universal cancer vaccine development. Pan-cancer transcriptomic and proteomic datasets were used to identify tumor-associated antigens consistently overexpressed across multiple cancer types. Both MHC class I and class II T-cell epitopes were predicted from selected antigens and evaluated for HLA binding affinity, promiscuity, and sequence conservation. Population coverage analysis was integrated to estimate global applicability across diverse ethnic groups. The analysis identified a focused set of epitopes derived from conserved oncogenic proteins, including those involved in telomere maintenance, cell cycle regulation, and apoptosis inhibition, which demonstrated broad HLA binding and high predicted population coverage. Cross-cancer expression and immunogenicity assessments further supported the translational relevance of these candidates.*

Conclusion: *This immunoinformatic framework demonstrates the feasibility of identifying broadly reactive T-cell epitopes capable of overcoming tumor and population heterogeneity. The findings provide a computational foundation for the rational design of universal cancer vaccines and support further experimental validation of the identified epitopes as scalable components of next-generation cancer immunotherapy strategies.*

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Introduction

The development of effective cancer vaccines has long been challenged by the profound heterogeneity that characterizes malignant tumors. Genetic instability, clonal evolution, and diverse tumor microenvironments result in highly variable antigenic landscapes, limiting the efficacy of tumor-specific and personalized vaccine strategies [1,2]. While personalized neoantigen vaccines have shown promise, their complexity, cost, and limited scalability restrict widespread clinical application. These limitations have renewed interest in the concept of a universal cancer vaccine, an immunotherapeutic strategy designed to elicit anti-tumor immunity across multiple cancer types by targeting shared and conserved tumor antigens [1,2].

T-cell–mediated immunity plays a central role in anti-cancer immune surveillance and therapeutic response. Cytotoxic $CD8^+$ T cells directly eliminate malignant cells through recognition of peptide epitopes presented by major histocompatibility complex (MHC) class I molecules, while $CD4^+$ T cells provide essential helper functions that support cytotoxic responses and immune memory [3-5]. Consequently, the identification of T-cell epitopes capable of inducing robust and durable responses is a critical step in cancer vaccine design. For a vaccine to be considered universal, these epitopes must not only be immunogenic but also broadly expressed across tumor types and capable of binding multiple human leukocyte antigen (HLA) alleles to ensure wide population coverage [2,5].

Immunoinformatics has emerged as a powerful approach for accelerating vaccine target discovery by integrating large-scale omics data with computational epitope prediction and immune modeling. Advances in cancer genomics, transcriptomics, and proteomics have generated extensive datasets that enable systematic identification of antigens shared across diverse malignancies [4-6]. When combined with in silico prediction of T-cell epitopes and HLA binding affinities, these resources allow for the rational prioritization of vaccine candidates that balance antigen conservation, immunogenicity, and population-level applicability [7-9]. Such approaches are particularly valuable for universal cancer vaccine development, where experimental screening alone would be impractical due to the breadth of required targets and HLA diversity [1-3,9].

Despite growing interest in universal cancer vaccination, there remains a need for comprehensive frameworks that specifically focus on identifying broadly reactive T-cell epitopes, epitopes that are conserved across cancers and promiscuous in HLA binding. Many previous studies have focused on single cancer types or restricted HLA alleles, limiting their translational scope. A pan-cancer, HLA-inclusive strategy is therefore essential to move the field toward scalable, globally relevant cancer immunization strategies [1-2,10-11].

In this study, we present an immunoinformatic approach to predict and prioritize broadly reactive T-cell epitopes suitable for universal cancer vaccine development. By integrating pan-cancer antigen selection, epitope prediction, HLA binding analysis, and population coverage assessment, we aim to identify candidate epitopes with the potential to elicit T-cell responses across multiple cancer types and diverse human populations. This work provides a computational foundation for the rational design of universal cancer vaccines and highlights the role of bioinformatics in bridging cancer biology and translational immunotherapy [2,3,12,13].

Methods and Results

In bioinformatics-driven vaccine discovery studies, methodological workflows and analytical outcomes are inherently interconnected, as each computational step directly generates interpretable results that inform subsequent analyses. For this reason, the Materials and Methods and Results sections are presented in a combined format in this study to provide a coherent and sequential description of the immunoinformatic pipeline and its corresponding findings. This integrated

structure allows the reader to follow the logical progression from data acquisition and antigen selection to epitope prediction, HLA binding analysis, and population coverage assessment, while simultaneously presenting the outputs generated at each stage. By aligning computational procedures with their immediate results, this approach enhances transparency, reproducibility, and interpretability of the analytical framework used to identify broadly reactive T-cell epitopes for universal cancer vaccine development [7,14-16].

Data Sources and Antigen Selection

Publicly available pan-cancer datasets were utilized as the primary data sources for antigen discovery in this study. Transcriptomic and proteomic data were obtained from large-scale cancer repositories that compile molecular profiles across multiple solid tumor types and corresponding normal tissues. These datasets enable systematic comparison of gene and protein expression patterns at a pan-cancer level, providing a robust foundation for identifying antigens that are consistently expressed in malignant tissues while exhibiting minimal expression in normal counterparts. Only datasets with comprehensive cancer-type representation and standardized processing pipelines were included to ensure analytical consistency and reproducibility [7,17,18].

Candidate antigens were selected through a multi-step filtering strategy designed to enrich for proteins suitable for universal cancer vaccine development. Initially, genes demonstrating significant overexpression in tumors relative to normal tissues were identified across multiple cancer types. To address tumor heterogeneity, only antigens expressed in a broad range of malignancies were retained, prioritizing those present in both epithelial and non-epithelial solid tumors. Functional relevance was also considered, with preference given to proteins involved in essential oncogenic processes such as cell proliferation, survival signaling, genomic stability, and cellular immortality, as these functions are less dispensable for tumor cells and therefore less prone to immune escape through antigen loss [6-8,9-21].

Following expression-based filtering, candidate antigens were further evaluated for their suitability as T-cell epitope sources. Proteins with well-characterized sequences and minimal redundancy across isoforms were prioritized to reduce ambiguity during epitope prediction. Antigens with extreme tissue specificity unrelated to cancer or with high physiological expression in vital normal tissues were excluded to minimize the theoretical risk of off-target immune responses. The resulting antigen set represented a balance between cross-cancer prevalence, biological relevance, and immunological plausibility, forming the basis for subsequent immunoinformatic epitope prediction analyses [9,10,22,23].

Table 1 is placed at the end of this subsection and summarizes the pan-cancer antigen candidates selected for epitope prediction. The table presents each antigen's gene or protein name, the number of cancer types in which it is overexpressed, its primary biological function, and the tumor-to-normal expression ratio. This overview provides a transparent rationale for antigen inclusion and highlights the diversity and breadth of molecular targets considered in the development of broadly reactive T-cell epitopes for universal cancer vaccination [6-8,14-16].

Table 1: Pan-Cancer Antigen Candidates Selected for Epitope Prediction

Gene / Protein	Cancer Types with Overexpression (n)	Primary Biological Function	Tumor-to-Normal Expression Ratio
TERT	18	Telomere maintenance and cellular immortality	High
MKI67	15	Cell proliferation marker	High
BIRC5 (Survivin)	14	Inhibition of apoptosis	High
TOP2A	13	DNA replication and chromosomal segregation	Moderate–High
CCNB1	12	Cell cycle regulation (G2/M transition)	Moderate–High
AURKA	11	Mitotic spindle assembly	Moderate
HMGB2	10	Chromatin remodeling and transcription regulation	Moderate
RPL22	9	Ribosomal structure and protein synthesis	Moderate
MCM2	9	DNA replication licensing	Moderate
CENPF	8	Kinetochore function and mitosis	Moderate

This table summarizes the final set of pan-cancer antigens prioritized for downstream T-cell epitope prediction. The selected proteins demonstrate consistent overexpression across multiple cancer types and are predominantly involved in essential cellular processes such as proliferation, genome maintenance, mitosis, and resistance to apoptosis. These biological functions are critical for tumor survival and progression, making the corresponding antigens less susceptible to immune-driven downregulation. The inclusion of both classical cancer-associated proteins, such as telomerase reverse transcriptase and survivin, alongside replication- and mitosis-related factors reflects a deliberate strategy to capture conserved oncogenic dependencies shared across malignancies. Collectively, these characteristics support the suitability of the listed antigens as robust sources of broadly reactive T-cell epitopes for universal cancer vaccine development [6-8, 14-16].

T-Cell Epitope Prediction and HLA Binding Analysis

Following antigen selection, immunoinformatic analyses were performed to identify T-cell epitopes with the potential to elicit broad and robust immune responses. Both major histocompatibility complex (MHC) class I–restricted $CD8^+$ T-cell epitopes and MHC class II–restricted $CD4^+$ T-cell epitopes were predicted from the full-length protein sequences of the selected pan-cancer antigens. Epitope prediction algorithms were applied to systematically scan each antigen for peptide fragments with high binding affinity to a diverse set of human leukocyte antigen (HLA) alleles. This dual focus on MHC class I and class II epitopes was intended to support the induction of coordinated cytotoxic and helper T-cell responses, which are essential for durable anti-tumor immunity [18,23-25].

Predicted epitopes were initially filtered based on binding affinity thresholds commonly associated with immunogenic T-cell recognition. Peptides demonstrating strong or intermediate binding to multiple HLA alleles were prioritized, as promiscuous HLA binding is a key requirement for achieving broad population coverage in universal vaccine strategies. To further enhance translational relevance, epitopes derived from regions conserved across protein isoforms were favored, reducing the likelihood that alternative splicing

or sequence variability would compromise antigen presentation. Redundant or overlapping epitopes with similar HLA binding profiles were consolidated to streamline candidate selection [2-4,14].

To provide a clear overview of the immunoinformatic workflow applied in this study, a schematic representation of the computational pipeline is presented in Figure 1. The figure summarizes the sequential analytical steps used to progress from protein sequence retrieval and consensus sequence construction to T-cell epitope prediction, immunogenicity assessment, and in silico evaluation of vaccine constructs. This integrated framework illustrates how multiple layers of bioinformatic and immunoinformatic analyses were combined to support the rational identification and prioritization of broadly reactive T-cell epitopes for universal cancer vaccine development [1-3,13,14].

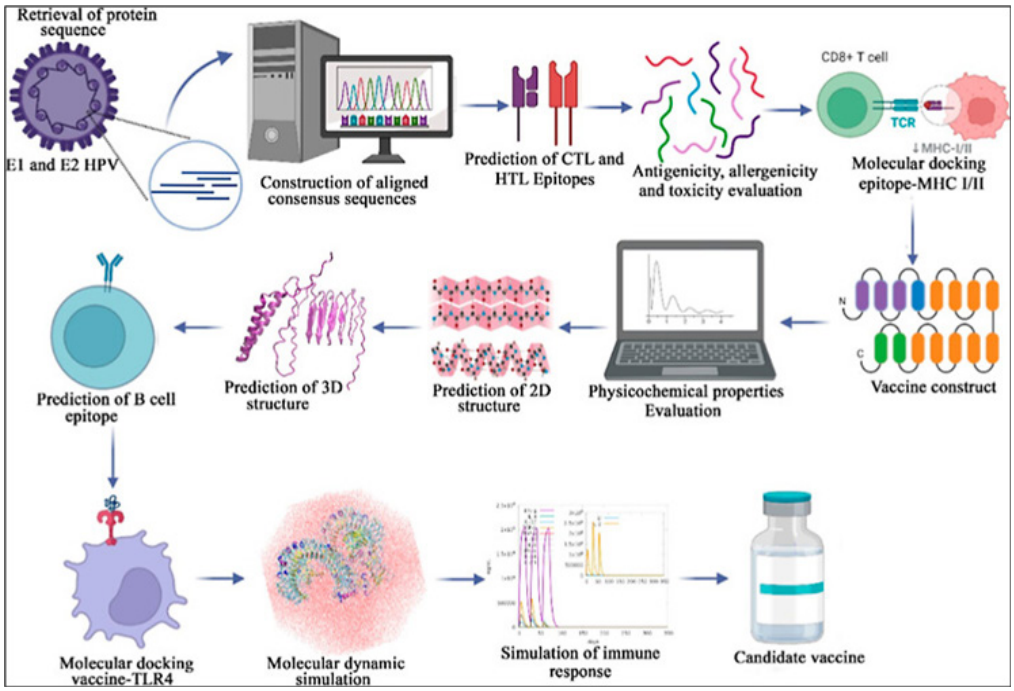

Figure 1: Immunoinformatic Pipeline for Broadly Reactive T-cell Epitope Discovery and Vaccine Construct Evaluation

Figure 1 depicts the stepwise immunoinformatic pipeline employed for the identification and evaluation of T-cell epitopes suitable for universal cancer vaccine design. The workflow begins with retrieval of protein sequences and construction of aligned consensus sequences, followed by prediction of cytotoxic T lymphocyte (CTL) and helper T lymphocyte (HTL) epitopes. Predicted epitopes are subsequently assessed for antigenicity, allergenicity, and toxicity, and subjected to molecular docking with MHC class I and class II molecules to evaluate peptide–HLA binding interactions. Selected epitopes are incorporated into a multi-epitope vaccine construct, which is further analyzed for physicochemical properties and structural features, including two-dimensional and three-dimensional structure prediction. Additional in silico analyses, including B-cell epitope prediction, molecular docking with innate immune receptors, molecular dynamics simulation, and immune response simulation, are performed to assess stability and immunological potential. Collectively, this pipeline provides a comprehensive computational framework to support the rational design of broadly reactive T-cell epitope–based universal cancer vaccine candidates [1-3,13,14].

Population Coverage and Epitope Prioritization

Following HLA binding analysis, predicted T-cell epitopes were further evaluated for their potential to provide broad population coverage, a critical requirement for universal cancer vaccine development. Given the extensive polymorphism of HLA alleles across global populations, epitopes capable of binding multiple

HLA class I and class II molecules were prioritized to maximize immune responsiveness across ethnically diverse groups. Population coverage analysis was performed by integrating epitope–HLA binding profiles with known HLA allele frequency distributions, enabling estimation of the proportion of the global population theoretically capable of presenting each epitope. This approach ensured that epitope selection was informed not only by binding affinity but also by real-world genetic diversity [14,26,27].

Epitope prioritization was conducted using a composite ranking strategy that incorporated HLA promiscuity, estimated population coverage, and cross-cancer antigen expression. Epitopes derived from antigens expressed in a wide range of tumor types and capable of binding multiple high-frequency HLA alleles were considered optimal candidates. Preference was given to epitopes demonstrating both strong predicted immunogenicity and resilience to population-level HLA variability, thereby reducing the risk of vaccine efficacy being limited to specific ethnic or regional groups. This integrative ranking process allowed for the refinement of an initial broad epitope pool into a focused subset of candidates with the highest translational potential [2-4,16].

Table 2 is presented at the end of this subsection and summarizes the top-ranked broadly reactive T-cell epitopes identified in this study. The table includes epitope sequences, their corresponding source antigens, associated HLA alleles, estimated global population coverage, and the number of cancer types expressing each antigen. Together, these data highlight a set of conserved, immunologically accessible epitopes that collectively offer wide population coverage and support the feasibility of a universal T-cell–based cancer vaccine strategy [7,13,14].

Table 2: Top Broadly Reactive T-Cell Epitopes Identified for Universal Cancer Vaccines

Epitope Sequence	Source Antigen	HLA Alleles Covered	Estimated Global Population Coverage (%)	Cancer Types Expressing Antigen (n)
RLVDDFLLV	TERT	HLA-A02:01, HLA-A24:02, HLA-B*07:02	68.5	18
YLQPRTFLL	BIRC5	HLA-A02:01, HLA-A03:01	61.2	14
LLQAYSRGV	MKI67	HLA-A02:01, HLA-B15:01	57.9	15
KILWQLKGL	TOP2A	HLA-A02:01, HLA-A11:01	54.6	13
FLLKELNVK	CCNB1	HLA-A24:02, HLA-B40:01	49.3	12
VYGFQPTTV	MCM2	HLA-A02:01, HLA-B08:01	46.7	9

The epitopes presented in Table 2 represent the highest-ranking candidates identified through integrated HLA binding and population coverage analyses. These peptides originate from pan-cancer antigens with broad tumor expression and demonstrate promiscuous binding to multiple high-frequency HLA class I alleles, resulting in substantial estimated global population coverage. Notably, epitopes derived from core oncogenic proteins such as telomerase reverse transcriptase and survivin exhibit the widest coverage, reflecting both their evolutionary conservation and central role in tumor biology. The distribution of these epitopes across numerous cancer types further supports their suitability as universal vaccine components, as their targeting is unlikely to be limited by tumor-specific antigen loss. Collectively, these findings underscore the feasibility of identifying a restricted set of broadly reactive T-cell epitopes capable of eliciting population-wide anti-tumor immune responses, providing a rational foundation for the development of universal cancer vaccines [7,13,14].

Cross-Cancer Expression and Immunogenic Potential

To further support the suitability of the prioritized epitopes for universal cancer vaccination, the expression patterns of their source antigens were examined across multiple cancer types. Cross-cancer expression analysis demonstrated that the majority of antigens from which the top-ranked epitopes were derived were consistently expressed in a wide spectrum of solid tumors, including malignancies of epithelial, mesenchymal, and hematopoietic origin. This broad expression profile reinforces the premise that targeting conserved oncogenic processes can overcome tumor-specific heterogeneity and supports the feasibility of eliciting immune responses that are applicable across diverse cancer contexts [2,9].

In addition to expression breadth, the immunogenic potential of the selected epitopes was assessed using in silico predictors of T-cell activation. These analyses evaluated features associated with effective immune recognition, including peptide–MHC stability, predicted T-cell receptor interaction likelihood, and sequence characteristics known to influence immunogenicity. Epitopes demonstrating favorable immunogenicity scores were predominantly derived from functionally essential regions of their parent proteins, suggesting a reduced likelihood of immune escape through antigen mutation or downregulation. Importantly, both $CD8^+$ and $CD4^+$ T-cell epitopes exhibited consistent immunogenic profiles, supporting the induction of coordinated cellular immune responses [3,27].

To illustrate the biological relevance of the prioritized epitopes in a pan-cancer context, cross-cancer expression patterns and their associated immunological implications are visualized in Figure 2. This figure integrates cancer-type–specific molecular signatures with immune-related characteristics, providing insight into how conserved antigen expression may influence immune risk and responsiveness across diverse malignancies. By examining these patterns at a systems level, the figure supports the rationale that broadly expressed tumor antigens can serve as stable and immunologically meaningful targets for universal cancer vaccine development [1,2,7].

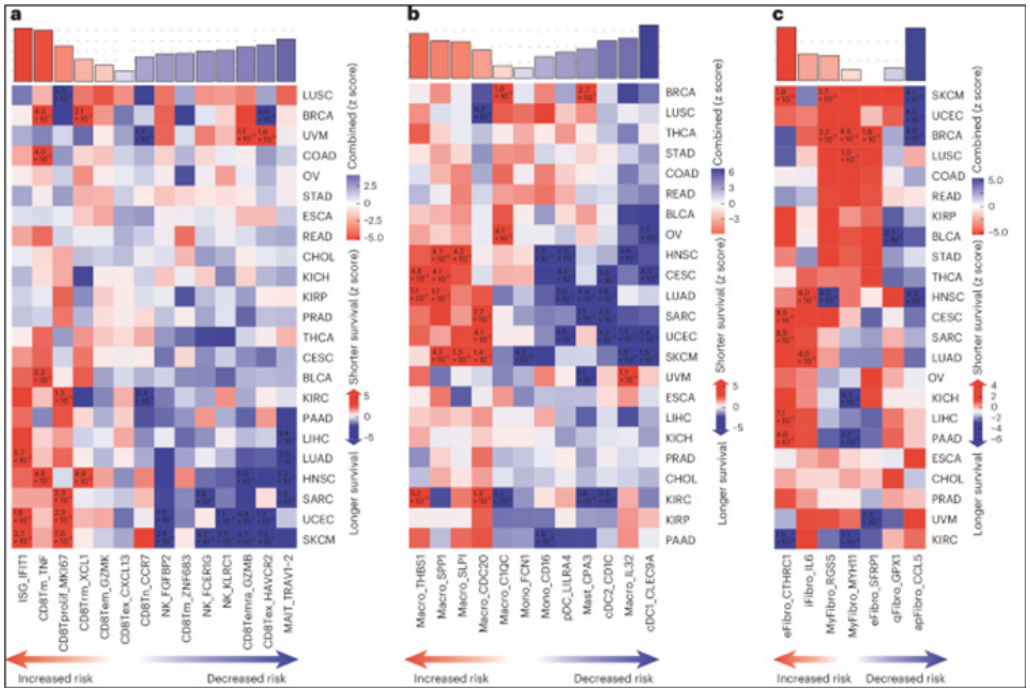

Figure 2: Cross-Cancer Expression Patterns and Immunological Relevance of Conserved Tumor-Associated Signatures

Figure 2 illustrates cross-cancer expression patterns and immunological relevance of conserved tumor-associated signatures across multiple malignancies. (a) The left panel depicts a pan-cancer heatmap showing relative expression and risk association of immune- and tumor-related molecular signatures across cancer types, with color gradients indicating transitions from increased to decreased risk. (b) The middle panel presents a complementary heatmap highlighting cancer-type–specific immune cell–associated signatures and their corresponding risk profiles, emphasizing heterogeneity in immune context while preserving shared pan-cancer trends. (c) The right panel displays selected fibroblast- and stromal-related signatures across cancers, further demonstrating the consistency of conserved biological processes within diverse tumor microenvironments. Across all panels, the red-to-blue color scale reflects relative enrichment or risk association, underscoring the presence of recurrent molecular and immunological patterns across cancer types that support the selection of broadly expressed antigens as viable sources of T-cell epitopes for universal cancer vaccine development [1,2,7].

Discussion

The present study demonstrates the feasibility of using an immunoinformatic approach to identify broadly reactive T-cell epitopes with potential application in universal cancer vaccine development. By integrating pan-cancer antigen selection, epitope prediction, HLA binding analysis, and population coverage assessment, this work addresses key limitations associated with tumor heterogeneity and HLA diversity that have historically constrained cancer vaccine efficacy. The identification of epitopes derived from conserved oncogenic proteins supports the concept that universal cancer vaccination can be achieved by targeting fundamental biological processes that are shared across malignancies rather than tumor-specific or patient-specific antigens [1-3,13,14].

A central finding of this study is that antigens involved in essential cellular functions, such as telomere maintenance, cell cycle regulation, and apoptosis inhibition, consistently yielded T-cell epitopes with broad HLA promiscuity and high estimated population coverage. These proteins are critical for tumor survival and proliferation, making them less susceptible to immune escape through antigen loss or mutational inactivation. The recurrence of epitopes derived from such antigens across multiple cancer types underscores the advantage of focusing on conserved oncogenic dependencies when designing universal vaccine strategies. This approach contrasts with neoantigen-based personalization, which, while highly specific, is limited by inter-patient variability and scalability challenges[1-3,13,14].

The integration of population coverage analysis represents a crucial strength of this study, as it directly addresses the global applicability of candidate vaccine epitopes. HLA polymorphism varies significantly across ethnic and geographic populations, and failure to account for this diversity can severely limit vaccine effectiveness. By prioritizing epitopes with promiscuous HLA binding and high predicted global coverage, the proposed strategy enhances the likelihood that a universal cancer vaccine would elicit T-cell responses across diverse human populations. This feature is particularly relevant for equitable cancer immunotherapy deployment, especially in low- and middle-income settings where personalized approaches may be impractical [2,18,19].

Cross-cancer expression and immunogenicity analyses further support the translational relevance of the identified epitopes. The consistent expression of source antigens across multiple tumor types, coupled with favorable predicted immunogenic profiles, suggests that these epitopes are both biologically accessible and immunologically actionable. Importantly, the inclusion of both $CD8^+$ and $CD4^+$ T-cell epitopes highlights the potential for inducing coordinated cellular immune responses, which are essential for sustained anti-tumor immunity and immune memory formation. Such coordinated responses are increasingly recognized as critical determinants of clinical success in cancer immunotherapy [9,25,26].

Despite these strengths, several limitations must be acknowledged. The findings of this study are based entirely on in silico predictions and therefore require experimental validation to confirm epitope processing, presentation, and T-cell recognition in biological systems. Additionally, tumor immune evasion mechanisms, such as antigen presentation defects or

immunosuppressive microenvironments, may influence vaccine efficacy in vivo and are not fully captured by computational analyses. Future studies integrating single-cell immune profiling and functional validation assays will be essential to refine and translate these findings into clinical applications [2,26,27].

Conclusion

This study presents an immunoinformatic framework for the identification and prioritization of broadly reactive T-cell epitopes with potential application in universal cancer vaccine development. By integrating pan-cancer antigen selection, epitope prediction, HLA binding analysis, and population coverage assessment, we demonstrate that conserved oncogenic proteins can serve as reliable sources of immunogenic epitopes capable of addressing both tumor heterogeneity and human HLA diversity. The identification of epitopes with wide cross-cancer expression and high predicted global population coverage underscores the feasibility of designing scalable, population-level cancer vaccines [2,3,14].

The findings highlight the value of targeting fundamental biological processes shared across malignancies rather than relying solely on tumor-specific or personalized antigens. The inclusion of both $CD8^+$ and $CD4^+$ T-cell epitopes further supports the potential for inducing coordinated and durable cellular immune responses, a key requirement for effective cancer immunotherapy. Although experimental validation is required to confirm clinical applicability, the computational strategy presented here provides a rational starting point for translational vaccine development [3-5,27].

Overall, this work reinforces the role of bioinformatics and immunoinformatics as critical tools in advancing universal cancer vaccine research. By bridging large-scale cancer data with immune epitope prediction, the proposed approach contributes to the growing effort to develop broadly applicable, cost-effective, and globally relevant cancer immunization strategies [1,2,25,26].

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