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Research Progress in Breast Cancer Vaccination

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Abstract. Breast cancer (BC), a leading global malignancy and cause of cancer death in women which faces limitations with conventional therapies and efficacy of immune checkpoint inhibitors, highlighting the need for targeted immunotherapies like cancer vaccines. This review indicates the recent progress of four principal vaccine platforms—DNA, peptide-based, dendritic cell (DC), and mRNA vaccines—for BC immunotherapy. DNA vaccines offer rapid, cost-effective neoantigen delivery with plasmid engineering, showing promise in preclinical models and early trials (e.g., NCT03199040), particularly when combined with checkpoint blockade and developed and optimized a novel polyepitope neoantigen DNA vaccine platform that can target multiple neoantigens and induce antitumor immune responses. Peptide vaccines, exemplified by NeuVax (E75) and DPX-0907, demonstrate significant clinical benefits like improved disease-free survival (89.7% at 5 years), but require adjuvants and strategies to counter immunosuppressive cells. DC vaccines, evolving towards cDC1 utilization and neoantigen targeting, effectively prime $CD4^+$ and $CD8^+$ T-cell responses in patients, showing potential for preventing recurrence. mRNA vaccines, propelled by innovations like lipid nanoparticles (LNPs) and self-amplifying RNA (saRNA), enable efficient antigen expression in dendritic cells and potent immunogenicity, with preclinical synergy observed when combined with checkpoint inhibitors. While each platform shows significant preclinical and early clinical feasibility and immunogenicity, translating this into consistent robust clinical responses in advanced BC remains challenging. Key hurdles include tumor heterogeneity, immunosuppressive microenvironments, optimal antigen selection, and delivery optimization. Realizing the full potential of BC vaccines necessitates overcoming these barriers through refined platform engineering and strategic combinations with other immunotherapies.

Keywords: breast cancer, DNA vaccination, peptide-based vaccine, dendritic cell vaccination, mRNA vaccination

1. Introduction

Breast cancer (BC) is the second most frequently diagnosed malignancy worldwide, accounting for over two million cases each year and the leading cause of cancer death in women [1,2]. For decades, cancer vaccine has been proposed as an important component of active immunotherapy, aimed at stimulating antigen-specific anti-tumor responses, achieving tumor prevention and regression [3]. Furthermore, conventional therapies carry substantial toxicities, highlighting the urgent need for

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more effective, targeted, and tolerable approaches. Immunotherapy, particularly immune checkpoint inhibitors (ICIs), has revolutionized oncology, yet its success in breast cancer is currently confined largely to the subsets of triple-negative breast cancer (TNBC), leaving significant unmet needs, especially in hormone receptor-positive (HR+) disease. This context underscores the immense potential of cancer vaccines – strategies designed to harness the patient's own immune system to recognize and eliminate tumor cells with high specificity and durable memory, ideally offering a safer, more targeted therapeutic option.

The core principle of cancer vaccines involves presenting tumor-associated antigens (TAAs) or neoantigens to the immune system, particularly T cells, to break tolerance and induce robust, antigen-specific cytotoxic T lymphocyte (CTL) responses. Key antigens in breast cancer include Human Epidermal Growth Factor Receptor 2(HER2/neu), Mucin1(MUC1), Carcinoembryonic Antigen (CEA), survivin, and telomerase, alongside patient-specific neoantigens. Recent technological leaps have propelled diverse vaccine platforms into clinical development, each with distinct advantages and challenges. DNA vaccines offer simplicity, stability, and ease of large-scale production, utilizing engineered plasmids encoding target antigens to transfect host cells for endogenous antigen presentation. mRNA vaccines, propelled to prominence by their COVID-19 success, provide rapid, flexible development, potent immunogenicity via inherent adjuvant properties, and the ability to encode complex antigens or multiple epitopes without genomic integration risk. Dendritic Cell (DC) vaccines represent a highly personalized approach, where patient-derived DCs are loaded ex vivo with tumor antigens and activated before reinfusion, acting as powerful natural adjuvants to prime T cells effectively. Peptide-based vaccines, often synthetic, are among the most clinically explored; they offer excellent safety profiles, defined composition, and the potential for multi-epitope cocktails targeting both CD8+ and CD4+ T cells, though they may require potent adjuvants and face HLA restriction limitations.

While promising preclinical data and early-phase clinical trials have demonstrated the feasibility and immunogenicity of these platforms in breast cancer, translating efficacy into consistent, robust clinical responses, particularly in advanced disease, has proven complex. Challenges include tumor heterogeneity, immunosuppressive microenvironments, immune evasion mechanisms, antigen selection, and optimal platform/combination strategies. This review focuses on the recent developmental progress of these four leading vaccine platforms – DNA, mRNA, DC, and peptide-based vaccines – for breast cancer immunotherapy. It will critically examine advances in antigen selection, delivery optimization, combination regimens, clinical trial outcomes, ongoing research, and the key hurdles that must be overcome to realize the full potential of vaccination as a cornerstone in the future management of breast cancer.

2. DNA vaccines

DNA vaccines represent a robust neoantigen delivery platform for personalized cancer immunotherapy, featuring swift manufacturing, cost-effectiveness, and versatile molecular engineering. Production utilizes plasmid isolation from bacterial cultures, allowing efficient incorporation of multiple neoantigen epitopes. Enhanced antigen expression is achieved via optimized eukaryotic promoters (e.g., CMV) and codon refinement [4]. Animal model data demonstrates precise immune activationin triple-negative breast cancer (TNBC) murine systems (E0771); polyepitope DNA constructs prime neoantigen-reactive $CD8^+$ T cells and synergized with anti-PD-L1 agents, substantially inhibit tumor progression [5]. Safety assessments confirm negligible integration risk into host genomes (below spontaneous mutation frequencies), with early-phase trials documenting only localized adverse events. Current clinical investigations, including

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NCT03199040 (TNBC) and NCT03122106 (pancreatic cancer), demonstrate initial clinical benefits in progression-free survival extension [4]. Future development necessitates refining delivery methodologies (e.g., electroporation) to augment cellular internalization and combining with immune checkpoint blockade to counteract immunosuppressive tumor niches.

Contemporary DNA vaccine innovation leverages antigen-specific precision engineering, particularly against oncogenic drivers like estrogen receptor alpha (ESR1) gain-of-function mutations. Using recombinant adenoviral constructs (Ad-ESR1mut) encoding prevalent ESR1 mutants (Y537N/Y537S), investigators induced robust ESR1-directed T-cell immunity in murine systems while overcoming immune tolerance barriers [6]. Critical proof-of-concept emerged from novel syngeneic mammary carcinoma models (MM3MG-ESR1mut), where mutant ESR1 expression conferred ligand-independent tumorigenicity without exogenous estrogen. Prophylactic Ad-ESR1-Y537N immunization markedly inhibited ESR1mut$^+$ neoplasm establishment, while therapeutic administration in established disease suppressed tumor progression and selectively attenuated both mutant ESR1 expression and estrogen-driven signaling cascades. Immune-editing manifested as vaccine-induced "antigen escape" via ESR1 downregulation, confirming target engagement. Clinical relevance was established through mass spectrometry verification of ESR1 mutant peptide presentation on human leukocyte antigen -A2 (HLA-A2) complexes, prompting a Phase I trial (NCT04270149) [6]. Preliminary analyses detected endogenous ESR1-reactive T cells in ER$^+$ breast cancer patient peripheral blood mononuclear cells, with post-vaccination expansion observed. This approach represents a strategic paradigm for preempting or reversing endocrine resistance.

3. Peptide-based vaccines

Peptide-based therapeutic cancer vaccines represent a promising immunotherapeutic approach that utilizes short amino acid sequences derived from tumor-associated antigens (TAAs) or tumor-specific antigens (TSAs) to elicit targeted anti-tumor immune responses. These vaccines primarily function by presenting epitopes that activate CD8$^+$ cytotoxic T lymphocytes (CTLs) or CD4$^+$ T-helper cells upon binding to human leukocyte antigen (HLA) molecules on antigen-presenting cells, enabling specific recognition and destruction of tumor cells [7]. Key advantages include their straightforward synthesis, cost-effective manufacturing, high chemical stability, low carcinogenic risk, and reduced susceptibility to pathogen contamination compared to cell-based or viral vector vaccines [8]. Additionally, the mode of administration is simple, and immune responses can be monitored in vitro, facilitating clinical application [7]. Epitope selection is critical and relies on identifying immunodominant peptides from targets overexpressed in tumors, such as HER2 in breast cancer or gp100 in melanoma. Computational methods like artificial neural networks (ANN) and immune epitope databases (IEDB) are employed to predict HLA-binding affinity and optimize epitope design. Notable examples include E75, a HER2-derived HLA-A2/A3-restricted peptide that demonstrated safety and clinical efficacy in breast cancer trials, and GP2, another HER2 peptide that induced CTL responses when combined with granulocyte-macrophage colony-stimulating factor (GM-CSF) as an adjuvant [8]. Despite these strengths, peptide vaccines face challenges such as limited immunogenicity, susceptibility to enzymatic degradation, and short half-life in vivo, often necessitating formulation with adjuvants (e.g. TLR agonists) or delivery systems (e.g., liposomes) to enhance stability and immunogenicity [7, 8].

Peptide vaccines demonstrate significant clinical promise in breast cancer. The HER2/neu-derived NeuVax (E75 peptide) combined with GM-CSF adjuvant achieved an 89.7% 5-year disease-free survival (DFS) in vaccinated high-risk recurrence patients versus 80.2% in controls; optimal

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dosing further increased DFS to 94.6% with minimal toxicity [9]. DPX-0907, incorporating seven HLA-A2-restricted peptides, induced tumor-specific CTL and HTL responses in ~70% of advanced breast, ovarian, and prostate cancer patients, with sustained immune memory observed. Personalized vaccines targeting patient-specific HLA alleles (e.g., HLA-A2/A24/A26) elicited IgG antibody responses in 46.7% of metastatic triple-negative breast cancer patients after six vaccinations, while 50% showed CTL activation [9]. However, challenges persist peptide vaccines can expand immunosuppressive regulatory T cells (Tregs), and myeloid-derived suppressor cells (MDSCs) in the tumor microenvironment, dampening antitumor efficacy. Strategies to overcome this include combining vaccines with Treg-depleting agents (e.g., anti-CD25 antibody daclizumab), which reduced Treg frequency and improved survival clinically, and using TLR agonists (e.g., CpG-ODN) as adjuvants to enhance DC maturation and effector T-cell responses while suppressing Tregs. Future development focuses on degenerating multi-epitope immunogens targeting multiple tumor antigens across diverse HLA haplotypes, coupled with transient Treg modulation [9]. Current clinical trials focus on multi-epitope vaccines targeting multiple antigens (e.g., WT1, MUC1) to counteract tumor immune escape, with many showing feasibility and tolerability across cancer types [7]. However, their efficacy as monotherapies remains modest, underscoring the need for combinatorial strategies with checkpoint inhibitors, chemotherapy, or targeted therapies to amplify clinical outcomes [8].

4. Dendritic cell vaccines

Dendritic cell (DC) vaccines harness the unique ability of DCs to prime anti-tumor T-cell responses by presenting tumor-associated antigens (TAAs) to the immune system. Conventional DC subsets, particularly type 1 conventional dendritic cells (cDC1s), excel at cross-presenting antigens on MHC-I to $CD8^+$ T cells and secreting immunostimulatory cytokines like IL-12, making them ideal for cancer immunotherapy [10]. However, early DC vaccines often used monocyte-derived DCs due to practical ex vivo generation from peripheral blood precursors, despite their functional limitations in migration, antigen presentation, and cytokine secretion compared to primary cDC1s. To overcome these issues, recent strategies optimize DC differentiation by incorporating Notch signaling (via OP9-DL1 stromal cells) and cytokine cocktails (e.g., FLT3L, GM-CSF), significantly improving cDC1 yield and phenotypic fidelity to in vivo counterparts. Genetic engineering further enhances DC function; for example, lentiviral overexpression of transcription factors (PU.1, IRF8, BATF3) reprograms fibroblasts into cDC1-like "induced DCs," while CCR7 overexpression boosts lymph node homing. Additionally, DCs can be engineered to express chimeric receptors (e.g., EVIR) that uptake tumor-derived extracellular vesicles, enabling in situ antigen loading and cross-dressing of pre-formed peptide-MHC complexes [10]. These advancements address historical shortcomings in DC vaccine efficacy, positioning cDC1s as a versatile platform for personalized therapy.

In breast cancer, DC vaccines have evolved to target tumor-specific neoantigens, which evade central tolerance and elicit potent immune responses. Recent clinical studies demonstrate that intranodal administration of autologous pulsed with HLA class II-restricted neoantigen peptides (15–18-mers) induces robust $CD4^+$ and $CD8^+$ T-cell activation. For instance, a retrospective analysis of five breast cancer patients showed that vaccines delivering long peptides encompassing both HLA-II and HLA-I epitopes triggered neoantigen-specific IFN-γ responses (measured by ELISPOT) in all cases, with TCR repertoire analysis confirming clonal expansion of tumor-reactive T cells in 60% of evaluated patients [11]. This synergy arises because HLA-II-restricted peptides activate $CD4^+$ T cells, which in turn enhance $CD8^+$ T-cell cytotoxicity and promote epitope spreading—critical for durable anti-tumor immunity. Notably, combining DC vaccines with immunomodulatory

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agents (e.g., immune checkpoint blockers) may amplify efficacy by counteracting tumor immunosuppression [10]. In HER2-positive breast cancer, trastuzumab's success via antibody-dependent cellular cytotoxicity underscores the potential of antigen-specific immunotherapy, motivating DC vaccine development against HER2 and other neoantigens [11]. Despite challenges like tumor heterogeneity, these approaches show promise in preventing recurrence, with no relapses reported in initial trials [12]. Future efforts should refine neoantigen selection and delivery routes to maximize clinical impact.

5. mRNA vaccines

mRNA vaccines represent a transformative approach in vaccine development, leveraging the body's cellular machinery to encode and produce specific antigens, thereby eliciting potent immune responses against target pathogens or tumor-associated antigens [13]. This platform offers significant advantages over conventional vaccine strategies (such as peptide-, protein-, or DNA-based vaccines), including high intrinsic immunogenicity due to the natural role of mRNA in protein synthesis and its recognition by innate immune sensors [14]. Furthermore, mRNA vaccines are characterized by their rapid design and scalable manufacturing processes, enabling swift adaptation to emerging threats like the COVID-19 pandemic highlighted in recent research. Crucially, mRNA vaccines possess a favorable safety profile, as they are non-infectious, lack integration risks into the host genome, and are rapidly degraded by normal cellular processes. However, initial clinical application of mRNA vaccines, particularly for cancer immunotherapy, faced significant hurdles primarily related to the inherent instability of naked mRNA molecules and their inefficient delivery in vivo, which hampered translation into effective therapeutic outcomes [14]. Recent technological breakthroughs have largely addressed these limitations. A key advancement involves the development of sophisticated delivery systems, notably lipid-based nanoparticles (LNPs), which encapsulate and protect the mRNA payload, facilitating efficient cellular uptake and expression of the encoded antigen. Importantly, the emergence of self-amplifying RNA viral vectors, derived from single-stranded RNA viruses like alphaviruses and flaviviruses, has further revolutionized the field [13]. These vectors contain replicons enabling massive intracellular RNA amplification from a single initial molecule, leading to substantially higher and more sustained antigen expression compared to non-replicating mRNA. This amplification property translates into a critical dose-sparing effect, demonstrated preclinically where significantly lower quantities of saRNA achieved protective immunity comparable to much larger doses (e.g., 80 μg) of conventional synthetic mRNA in influenza challenge models. Consequently, these innovations in vector design and lipid-based delivery have positioned mRNA vaccines as a highly promising and versatile platform for both infectious disease prevention and cancer immunotherapy [13, 14].

A critical advancement in mRNA vaccine technology lies in the development of sophisticated delivery systems to overcome inherent challenges. Naked mRNA is highly unstable under physiological conditions and struggles to efficiently enter the cytoplasm of target cells, particularly dendritic cells (DCs), which are essential for initiating potent immune responses [15]. No particle (NP) carriers, such as Lipid/Calcium/Phosphate (LCP) NPs, have emerged as powerful solutions, significantly enhancing mRNA stability, durability, and expression levels in vivo. These NPs, often surface-modified with targeting ligands like mannose to bind receptors highly expressed on DCs (e.g., mannose receptors), facilitate the directed delivery of encapsulated mRNA encoding tumor antigens (e.g., MUC1) to lymph nodes and subsequent uptake by DCs. Once internalized, the mRNA is released into the DC cytoplasm, bypassing the need for nuclear entry required by DNA vaccines, and is rapidly translated into the target tumor antigen protein. This efficient antigen

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expression within DCs enables effective processing and presentation via MHC molecules, crucial for priming tumor-specific cytotoxic T lymphocyte (CTL) responses, a key advantage over carbohydrate vaccines which primarily induce antibodies. Furthermore, the transient nature of mRNA-mediated gene expression minimizes risks associated with genomic integration, enhancing safety compared to DNA-based approaches [15]. However, the efficacy of mRNA vaccine-induced T cells can be hampered by inhibitory mechanisms within the tumor microenvironment (TME), such as the upregulation of immune checkpoint molecules like CTLA-4 on activated T cells, which attenuates their responses. Consequently, combining NP-based mRNA vaccines (e.g., MUC1 mRNA-LCP) with immune checkpoint blockade therapies, such as anti-CTLA-4 monoclonal antibodies, has proven highly synergistic in preclinical models. This combination significantly enhances anti-tumor CTL activity, T cell infiltration into tumors, and overall tumor growth inhibition compared to either modality alone, by counteracting T cell inhibition or amplifying the vaccine-primed immune response. Despite promising results demonstrating potent antigen-specific immunity and manageable toxicity profiles in animal studies, challenges related to optimizing delivery efficiency, stability for widespread distribution, and clinical translation for solid tumors remain active areas of active research efforts and technological progress needed to solve the remaining challenges (delivery, stability, solid tumor efficacy) before mRNA cancer vaccines can reach their full potential as a widespread and effective clinical therapy [15].

6. Conclusion

Cancer vaccines represent a highly promising frontier in breast cancer immunotherapy, aiming to leverage the patient's immune system for targeted tumor cell eradication with high specificity and durable memory. This review has detailed the significant developmental progress across four leading platforms: DNA, peptide-based, dendritic cell (DC), and mRNA vaccines. Each platform possesses distinct advantages and faces specific challenges in the quest for effective BC treatment.

DNA vaccines have advanced through precision engineering against targets like mutant ESR1, demonstrating robust immune induction in models and offering a swift, cost-effective neoantigen delivery system. Early clinical trials indicate potential benefits, particularly when strategies like electroporation enhance delivery or combination with checkpoint inhibitors counteracts immunosuppression. Peptide vaccines, clinically the most explored, have achieved notable successes, such as the NeuVax (E75) regimen yielding an 89.7% 5-year disease-free survival rate in high-risk patients. Their defined composition and safety profile are strengths, yet inherent limitations like low immunogenicity and HLA restriction necessitate potent adjuvants, delivery systems, and combination strategies, including Treg depletion, to amplify efficacy. DC vaccines have evolved significantly, moving beyond monocyte-derived DCs to optimize type 1 conventional DCs (cDC1s) through improved differentiation protocols and genetic engineering (e.g., CCR7 overexpression, EVIR receptors). Focusing on tumor-specific neoantigens delivered intranodally has proven effective in activating both $CD4^+$ and $CD8^+$ T cells in BC patients, with early trials suggesting potential for preventing recurrence. mRNA vaccine technology has been revolutionized by breakthroughs in delivery, particularly lipid nanoparticles (LNPs) ensuring stability and targeted delivery to lymph nodes, and the advent of self-amplifying RNA (saRNA) vectors enabling dose-sparing and sustained high antigen expression. Preclinically, mRNA vaccines (e.g., MUC1 mRNA-LCP) show potent synergy with immune checkpoint blockade like anti-CTLA-4.

Despite these considerable advances and demonstrable immunogenicity in preclinical and early clinical settings, the consistent translation into robust clinical responses, especially for advanced metastatic disease, remains a complex challenge. Persistent hurdles are multifaceted: significant

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tumor heterogeneity complicates antigen selection; profoundly immunosuppressive tumor microenvironments can dampen or abrogate vaccine-primed immune responses; optimal platform selection, antigen delivery, and dosing strategies require further refinement; and the logistical challenges of personalized neoantigen vaccine manufacturing and stability (especially for mRNA) need scalable solutions. Furthermore, while monotherapy vaccine efficacy is often modest, strategic combinations—particularly with immune checkpoint inhibitors to overcome microenvironmental suppression, but also with chemotherapy, targeted therapies, or Treg-modulating agents—hold immense promise for amplifying clinical outcomes.

In conclusion, the landscape of breast cancer vaccination is marked by rapid technological innovation and encouraging clinical signals across multiple platforms. Realizing the full potential of these vaccines as a cornerstone of future BC management demands continued focus on overcoming the identified biological and technical barriers. Prioritizing intelligent antigen selection, optimizing platform/delivery for robust and durable immunity, and developing rational, synergistic combination regimens are critical pathways forward. Sustained research and clinical trial efforts are essential to transform the substantial promise of cancer vaccines into widespread clinical reality for breast cancer patients.

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