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A COMPREHENSIVE REVIEW OF CONTEMPORARY AND CONVENTIONAL TREATMENTS FOR PROSTATE CANCER AND OTHER ALTERNATIVE APPROACHES

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ABSTRACT: This review synthesizes contemporary advances in the diagnosis and management of prostate cancer, with a focus on therapeutic developments that have substantially improved outcomes in both localized and metastatic disease. The scope of the review encompasses curative-intent treatment modalities, advances in systemic therapy, and improvements in diagnostic imaging and staging that collectively inform modern clinical decision-making. Key therapeutic advances discussed include the widespread adoption of robotic-assisted radical prostatectomy, stereotactic body radiotherapy, and brachytherapy, which have expanded curative options for patients with localized disease. In the metastatic setting, the integration of novel androgen receptor–targeted agents, next-generation androgen deprivation strategies, and life-prolonging chemotherapeutic regimens has markedly improved survival and disease control. The review also highlights the role of advanced imaging techniques in more accurate staging, risk stratification, and treatment selection. Major themes include the transition from predominantly palliative approaches to individualized, multimodal treatment strategies with curative or life-extending intent, as well as the increasing importance of precision in diagnosis and therapy selection. Emphasis is placed on clinically relevant developments rather than historical evolution. In conclusion, recent advances in prostate cancer management have transformed the therapeutic landscape, enabling improved prognosis across disease stages. Ongoing innovation in imaging, systemic therapies, and localized treatments continues to refine patient outcomes, underscoring the need for integrated, evidence-based approaches in contemporary prostate cancer care.

INTRODUCTION: Prostate cancer management has evolved substantially with the expansion of effective local and systemic therapies; however, advanced disease remains associated with treatment resistance and limited long-term survival. While surgery and radiotherapy remain central to the management of localized disease, systemic approaches including androgen deprivation therapy, androgen receptor signalling inhibitors, and chemotherapy form the foundation of treatment for recurrent and metastatic prostate cancer.

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Despite these advances, progression to castration-resistant disease is common and represents a major therapeutic challenge. Recent years have seen the incorporation of novel systemic strategies, such as radioligand therapy and poly (ADP-ribose) polymerase inhibitors, which have improved outcomes in selected patient populations. In contrast, immune checkpoint inhibitors have demonstrated limited efficacy in prostate cancer, largely due to an immunologically "cold" tumour microenvironment.

Nonetheless, clinical activity has been observed in specific molecular subgroups, underscoring the need for more effective immunotherapeutic approaches. Emerging T-cell–based therapies offer a potential strategy to overcome immune resistance in prostate cancer. Building on the clinical proof-

of-concept established by sipuleucel-T, bispecific T-cell engagers represent a promising novel class of immunotherapy designed to redirect T cells toward tumour-specific antigens such as prostate-specific membrane antigen. By facilitating targeted immune activation within the tumour microenvironment, these agents may address key limitations of existing immunotherapies.

This review aims to summarize the current standard of care for metastatic hormone-sensitive and castration-resistant prostate cancer, and to critically examine the therapeutic rationale, mechanisms, and clinical potential of bispecific T-cell engagers in this evolving treatment landscape.

Key Historical Milestones in Prostate Cancer Treatment:

Early Surgical Management: The development of radical prostatectomy in the early 20th century established surgery as a definitive treatment for localized prostate cancer and remains foundational to current surgical approaches, including minimally invasive techniques.

Introduction of Radiation Therapy: The early application of radiation therapy in the early 1900s, including the emergence of brachytherapy, laid the groundwork for modern external beam radiotherapy and contemporary brachytherapy techniques used in curative treatment.

Biomarker Development: The identification of prostate-specific acid phosphatase (PAP) in 1938 represented the first use of a tumour biomarker in prostate cancer, paving the way for biomarker-driven diagnosis and monitoring, later refined by prostate-specific antigen (PSA) [1-3].

Key Therapeutic Developments (1940s–1980s):

Hormonal Manipulation: The demonstration in the 1940s that prostate cancer growth is androgen-dependent established the biological basis for androgen deprivation therapy, which remains a cornerstone of treatment for advanced and metastatic disease [4].

Advances in Surgical Technique: The introduction of the retropubic approach to radical prostatectomy improved surgical access and outcomes, influencing contemporary open and minimally invasive prostatectomy techniques [5].

Evolution of Radiation Therapy: The transition to external beam radiation therapy using megavoltage technology enabled deeper tumour penetration and improved dose delivery, forming the foundation of modern EBRT. The use of cobalt teletherapy further expanded radiotherapeutic options for patients with inoperable disease [6].

Key Advances in Local Therapy and Androgen Deprivation (1970s–1980s):

Modern Radiotherapy Foundations: The introduction of iodine-125–based brachytherapy and the clinical adoption of external beam radiation therapy established radiation as a curative option. Subsequent development of intensity-modulated radiation therapy and TRUS-guided brachytherapy implantation enabled precise dose delivery and remain integral to contemporary practice [7].

Hormonal Therapy Evolution: The synthesis and clinical application of luteinizing hormone–releasing hormone (LHRH) analogues defined medical castration as a standard alternative to surgical orchiectomy. The commercial availability of agents such as leuprolide and buserelin, along with early antiandrogens, formed the backbone of modern androgen deprivation therapy [8].

Key Diagnostic Advances (1960s–1980s):

Risk Stratification and Disease Monitoring: The introduction of the Gleason grading system remains central to prognosis and treatment selection. The identification and clinical adoption of prostate-specific antigen transformed prostate cancer diagnosis, treatment monitoring, and recurrence detection [9, 10].

Imaging and Biopsy Techniques: The use of bone scintigraphy for metastatic detection and the development of systematic prostate biopsy enabled accurate staging and histopathological confirmation, both essential to modern diagnostic workflows [11].

Transformative Advances of the Past 30 Years

Chemotherapy and Systemic Therapy: Docetaxel demonstrated a survival benefit in metastatic castration-resistant and later metastatic hormone-sensitive prostate cancer, establishing chemotherapy as a life-prolonging modality. Cabazitaxel further expanded second-line treatment options [12, 13].

Androgen Receptor Pathway Inhibitors: The emergence of abiraterone, enzalutamide, and newer agents such as darolutamide significantly improved survival across multiple disease states, redefining systemic therapy paradigms [14-16].

Combination Strategies: Recent evidence supporting triplet therapy, including androgen receptor pathway inhibition with docetaxel in metastatic hormone-sensitive disease, represents a major advance in contemporary prostate cancer management [17].

Practice-Defining Clinical Trials in Contemporary Prostate Cancer Management:

Chemotherapy as Standard of Care: The TAX-327 and SWOG 99-16 trials established docetaxel as the first therapy to confer an overall survival benefit in metastatic castration-resistant prostate cancer (mCRPC), forming the backbone of subsequent systemic treatment strategies [12]. Cabazitaxel later expanded second-line chemotherapy options following docetaxel failure [13].

Androgen Receptor Pathway Inhibition: Landmark trials including COU-AA-301/302, AFFIRM, and PREVAIL demonstrated survival benefits with abiraterone and enzalutamide in both pre- and post-docetaxel mCRPC, leading to their widespread adoption across disease stages [14-16]. Subsequent studies (LATITUDE, TITAN, ARCHES) confirmed the efficacy of androgen receptor pathway inhibitors in metastatic hormone-sensitive prostate cancer (mHSPC) [17-19].

Nonmetastatic Castration-Resistant Disease: The PROSPER, SPARTAN, and ARAMIS trials showed that enzalutamide, apalutamide, and darolutamide significantly delay metastasis and improve outcomes in nonmetastatic castration-resistant prostate cancer, establishing a new treatment paradigm [20, 22].

Targeted and Radioligand Therapies: The Profound trial introduced biomarker-driven therapy with PARP inhibition for patients harboring DNA repair gene alterations. The ALSYMPCA and VISION trials validated the role of radionuclide therapies, including radium-223 and lutetium-177–PSMA-617, in selected patients with advanced mCRPC [23, 25].

Combination and Intensification Strategies: The CHAARTED and ARASENS trials demonstrated survival benefits with treatment intensification using docetaxel and triplet therapy in mHSPC, shaping current combination treatment approaches [26, 27].

PSMA and Nuclear Medicine:

PSMA-targeted Imaging and Therapy: The clinical adoption of PSMA PET imaging has markedly improved staging accuracy compared with conventional imaging and is now integral to disease localization and treatment planning [28]. The success of radionuclide therapies, including radium-223 and lutetium-177–PSMA-617, established theranostics as a standard option for selected patients with metastatic castration-resistant prostate cancer, with expanding clinical use in recent years [24, 25].

Precision Medicine and Immuno-Oncology:

Targeted and Cellular Therapies: Sipuleucel-T provided proof of principle for immunotherapy in prostate cancer, while the Profound trial established PARP inhibition as a biomarker-driven treatment for patients with homologous recombination repair gene alterations, integrating genomic testing into routine clinical decision-making [23, 29].

Advances in Curative Treatment and Surveillance:

Surgical Innovation: The transition from open to laparoscopic and robot-assisted radical prostatectomy improved perioperative outcomes and functional recovery, making robotic surgery the predominant approach in many centers [30].

Radiotherapy Optimization: Evidence supporting moderate hypofractionation demonstrated noninferior oncologic and toxicity outcomes compared with conventional fractionation. This approach is now recommended as the standard for external beam radiotherapy due to equivalent efficacy and improved resource efficiency [31].

Modern Diagnostic Refinement:

Pathological Classification: Refinements to Gleason grading and the introduction of ISUP grade groups standardized risk stratification and reporting, improving prognostication and treatment selection [32].

Imaging-guided Diagnosis: Pre-biopsy multiparametric MRI and MRI-targeted biopsy techniques have become the diagnostic standard, reducing unnecessary biopsies and enhancing detection of clinically significant disease. The PI-RADS system further standardized MRI interpretation across clinical practice [33].

Metastatic Hormone-Sensitive Prostate Cancer (mHSPC): Prognostic Stratification: Risk stratification is central to therapeutic decision-making in metastatic hormone-sensitive prostate cancer. The most widely used prognostic frameworks are derived from the CHAARTED and LATITUDE trials, which classify patients based on disease burden and adverse clinical features [34, 35].

Disease Volume and Risk: High-volume or high-risk disease is defined by the presence of extensive bone metastases and/or visceral involvement, with additional consideration of tumour grade in some models. Patients who do not meet these criteria are classified as low-volume or low-risk.

Timing of Metastasis: The presence of de novo metastatic disease at initial diagnosis is an established adverse prognostic factor. Patients presenting with de novo metastases experience significantly poorer overall survival compared with those who develop metastatic disease following prior local therapy (metachronous metastases).

Clinical Implications: High-volume or high-risk disease, particularly in the de novo metastatic setting, is associated with inferior survival outcomes and supports the use of treatment intensification strategies, including combination systemic therapy. In contrast, patients with low-volume or metachronous disease generally demonstrate more favourable outcomes and may benefit from individualized treatment approaches. In summary, contemporary prognostic models in mHSPC emphasize metastatic burden, disease biology, and timing of metastatic presentation. These factors guide risk-adapted treatment selection and underpin current recommendations for therapy intensification in advanced prostate cancer.

Androgen Deprivation Therapy (ADT) in mHSPC: Androgen deprivation therapy, achieved through surgical castration or medical castration using LHRH agonists or antagonists, remains the therapeutic backbone for metastatic hormone-sensitive prostate cancer.

However, ADT monotherapy is no longer considered standard of care, as survival outcomes are inferior compared with intensified regimens. ADT alone should be reserved for patients who are very elderly, frail, or unable to tolerate combination therapy [36].

Combination Therapies in mHSPC:

ADT plus Chemotherapy: The addition of docetaxel to ADT significantly improves overall survival, particularly in patients with high-volume disease. This benefit established chemotherapy as an important component of treatment intensification, although its utility is more limited in low-volume disease [37].

ADT plus Androgen Receptor Signalling Inhibitors (ARSIs): Multiple randomized trials have demonstrated that combining ADT with ARSIs including abiraterone, enzalutamide, and apalutamide provides a consistent survival benefit over ADT alone. These benefits are observed across both high- and low-volume disease, making ADT plus ARSI a preferred first-line strategy for most patients with mHSPC [38, 39].

Triple Therapy (ADT + ARSI + Docetaxel): Recent evidence supports further treatment intensification with triplet therapy in selected patients. The combination of ADT, docetaxel, and an ARSI particularly darolutamide or abiraterone has demonstrated additional survival benefits, especially in patients with high tumour burden and good performance status. Triplet therapy now represents an important option for fit patients with aggressive disease biology. In summary, modern management of mHSPC emphasizes early treatment intensification. ADT serves as the foundation, while combination and triplet strategies are now central to improving survival outcomes and are tailored according to disease burden, patient fitness, and treatment goals [39].

Metastatic Castration-Resistant Prostate Cancer (mCRPC):

First-Line Systemic Therapy: First-line treatment of mCRPC is guided by prior therapy exposure, disease burden, and patient fitness. Docetaxel

chemotherapy and androgen receptor signalling inhibitors (ARSIs), including abiraterone and enzalutamide, are established first-line options with proven survival benefit. ARSIs are commonly preferred in asymptomatic or minimally symptomatic patients, whereas docetaxel is favoured in patients with rapidly progressive or high-burden disease. Treatment selection is individualized, as cross-resistance between ARSIs may limit sequential efficacy [40, 41].

Treatment after Progression: Following progression on first-line therapy, multiple life-prolonging options are available. Cabazitaxel is the preferred chemotherapy after docetaxel and has demonstrated superiority over switching to an alternate ARSI in previously treated patients. Abiraterone and enzalutamide remain options in selected patients depending on prior exposure and treatment response.

Radionuclide therapy with radium-223 is indicated for patients with symptomatic bone-predominant disease without visceral metastases. This approach improves survival while providing symptomatic benefit [24, 25].

Molecular and Targeted Therapies: The integration of molecular profiling has introduced precision medicine into mCRPC management. PARP inhibitors, particularly olaparib, provide significant benefit in patients with homologous recombination repair gene alterations, such as BRCA1/2 or ATM mutations. PSMA-targeted radioligand therapy with lutetium-177–PSMA has demonstrated improved progression-free and overall survival in heavily pretreated patients and is now an important therapeutic option.

AKT pathway inhibition represents an emerging strategy in molecularly selected populations, although its role remains under investigation.

Safety Considerations: Molecular and radionuclide therapies are associated with distinct toxicity profiles, including hematologic toxicity with PARP inhibitors, gastrointestinal and metabolic effects with AKT inhibitors, and fatigue, xerostomia, and anaemia with PSMA-targeted radioligand therapy. Careful patient selection and toxicity monitoring are essential. In summary, mCRPC management relies on rational sequencing of chemotherapy, ARSIs, radionuclide therapy, and molecularly targeted treatments, guided by prior therapy, molecular alterations, disease distribution, and patient-related factors [23, 25].

Immunotherapy and Immune-Based Approaches in Prostate Cancer: Critical Appraisal: Despite transformative success in other solid tumours, immunotherapy has shown limited clinical benefit in prostate cancer. Large randomized trials evaluating immune checkpoint inhibitors have failed to demonstrate consistent improvements in overall survival or progression-free survival in unselected patient populations. Ongoing studies are exploring immunotherapy in molecularly defined subgroups; however, results to date suggest that broad applicability remains unlikely.

The underperformance of immunotherapy in prostate cancer is largely attributable to tumour-intrinsic and microenvironmental factors. Prostate cancer is characterized by a low tumour mutational burden, limited neoantigen expression, and a predominantly immunologically “cold” tumour microenvironment. This is further compounded by poor T-cell infiltration, impaired antigen presentation, and an immunosuppressive milieu enriched with regulatory T cells and myeloid-derived suppressor cells, collectively limiting responsiveness to immune checkpoint blockade [29].

Cell-Based Immunotherapy: Cell-based immunotherapies have not secured a durable role in metastatic castration-resistant prostate cancer. Sipuleucel-T provided early proof of principle for immune activation and demonstrated a modest survival benefit in selected patients. However, its lack of impact on disease progression, logistical complexity, high cost, and limited clinical applicability have restricted widespread adoption, and it is no longer routinely used in practice.

Therapeutic Vaccines: Cancer vaccines have similarly failed to translate early signals of activity into meaningful clinical benefit. Although agents such as PROSTVAC demonstrated promising survival signals in early-phase studies, confirmatory phase III trials did not show an overall survival advantage. These findings highlight the challenge of generating a sustained

and effective antitumor immune response in prostate cancer [29, 42].

Summary Perspective: Overall, immune-based therapies have underperformed in prostate cancer due to unfavourable tumour biology and a suppressive immune microenvironment. Future progress is likely to depend on rational combination strategies, improved patient selection based on molecular or immune biomarkers, and approaches designed to convert immunologically "cold" tumours into "hot" tumours capable of mounting effective immune responses.

Bispecific T-Cell Engagers in Prostate Cancer: Bispecific T-cell engagers (BiTEs) represent an investigational immunotherapeutic strategy designed to redirect cytotoxic T cells toward tumour-specific antigens. In prostate cancer, PSMA-directed BiTEs consist of bispecific antibody constructs that bind CD3 on T cells and prostate-specific membrane antigen on tumour cells, thereby promoting localized T-cell activation and tumour cell lysis. The clinical feasibility of this approach has been established in hematologic malignancies, but its applicability to solid tumours remains under active investigation.

Early-phase clinical studies of PSMA-targeted BiTEs in metastatic castration-resistant prostate cancer have primarily evaluated safety and tolerability. These studies have demonstrated manageable toxicity profiles, with common adverse events including fever, chills, fatigue, and injection-site reactions. Preliminary signals of antitumor activity have been reported; however, these findings are derived from small patient cohorts and are insufficient to establish clinical efficacy. Several biological and practical challenges may limit the effectiveness of BiTEs in prostate cancer, including heterogeneous PSMA expression, limited T-cell infiltration within the tumour microenvironment, and the risk of systemic immune-related toxicities. Additional bispecific constructs targeting antigens such as STEAP-1, ADAM-17, and glypican-1 are in early stages of development, but clinical data remain limited. In summary, bispecific T-cell engagers in prostate cancer should currently be regarded as experimental therapies. While early-phase studies support further investigation, their role in routine clinical practice remains undefined and will depend on results from larger trials demonstrating meaningful efficacy, durability of response, and acceptable safety profiles [43].

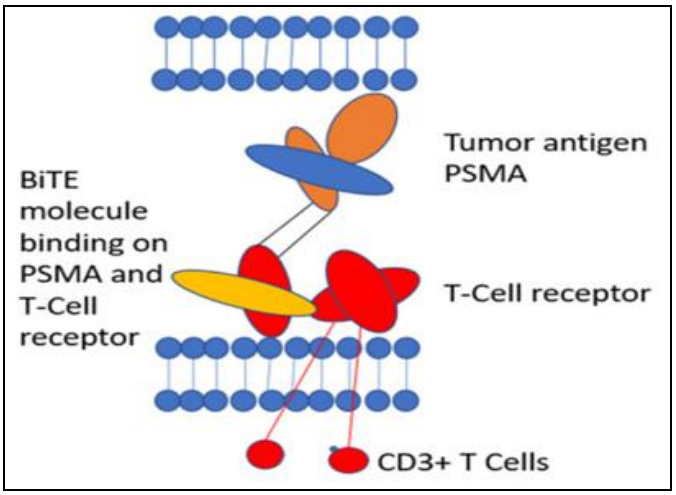

FIG. 1: BISPECIFIC T-CELL ENGAGER. MODIFIED ACCORDING TO STROHL *ET AL* [43]

Discussion and Future Directions: Substantial progress in prostate cancer management has translated into excellent outcomes for localized disease; however, high-risk and metastatic prostate cancer continue to drive disease-specific mortality. Future advances must therefore prioritize durable disease control and survival improvement in these populations rather than further refinement of already effective low-risk interventions.

In local therapy, technological advances such as stereotactic body radiotherapy and dose-escalated brachytherapy have improved treatment precision, but long-term survival advantages over established standards remain unproven. Current evidence supports careful patient selection, as improvements in convenience or short-term toxicity have not consistently translated into superior oncologic outcomes. Similarly, the role of definitive local treatment in metastatic disease, while supported by selected trial data, requires further clarification regarding patient selection and optimal integration with systemic therapy. Systemic treatment advances have reshaped outcomes in advanced disease, particularly through early treatment intensification with androgen receptor pathway inhibitors, chemotherapy, and combination regimens. Future progress is likely to depend less on introducing additional agents within the same therapeutic classes and more on optimizing

sequencing, combination strategies, and biomarker-driven selection to minimize resistance and cumulative toxicity. Diagnostic innovation remains central to progress. PSMA PET imaging has redefined staging and disease localization, while MRI-based diagnostic pathways have reduced overtreatment. However, screening strategies based on prostate-specific antigen alone have shown limited impact on overall mortality, underscoring the need for risk-adapted, multimodal screening approaches incorporating imaging and molecular markers. Emerging fields including precision oncology, theranostics, and immune-based therapies offer incremental but still investigational advances. Meaningful clinical impact will require robust validation in well-defined patient subsets and clear demonstration of survival benefit. In conclusion, the future of prostate cancer care lies in integrating advanced imaging, molecular stratification, and rational treatment intensification, with a focus on high-risk and metastatic disease rather than further expansion of low-risk interventions.

Limitations: This review is intended as a broad, narrative overview of the evolution of prostate cancer diagnosis and treatment rather than an in-depth analysis of individual therapeutic modalities or trials. As such, the scope prioritizes synthesis over exhaustive evaluation, and detailed methodological critique of primary studies was not undertaken. The narrative and historical nature of the review inherently introduces a degree of selection bias in the inclusion and emphasis of milestones, and the interpretation of developments may reflect prevailing clinical perspectives rather than a strictly historiographical framework. In addition, the review was authored by medical professionals without formal collaboration with a professional historian. While this approach ensures clinical relevance and contextual interpretation for contemporary practice, it may limit the depth of historical analysis and the consideration of broader sociocultural or scientific contexts. Finally, as with all narrative reviews, the conclusions drawn are dependent on the available literature and do not follow a systematic review methodology, which may restrict reproducibility and completeness.

CONCLUSIONS: Prostate cancer remains the most common solid malignancy in men, and major therapeutic advances have substantially improved outcomes, particularly in advanced disease. The integration of treatment intensification strategies including chemotherapy and androgen receptor pathway inhibitors has redefined survival expectations in metastatic hormone-sensitive prostate cancer, while molecularly targeted agents and radioligand therapies have expanded options for selected patients with castration-resistant disease. Despite these advances, metastatic castration-resistant prostate cancer remains incurable, underscoring the need for more effective and durable treatment strategies. Therapeutic progress is increasingly constrained by treatment resistance, cumulative toxicity, and the limited benefit of sequential therapies within the same drug classes.

Advances in diagnostic imaging have become central to modern clinical practice. PSMA PET/CT and standardized MRI-based assessment systems such as PI-RADS® have improved disease staging, risk stratification, and treatment selection, enabling more personalized management approaches. Targeted therapies, including PARP inhibitors for patients with homologous recombination repair gene alterations and PSMA-directed radioligand therapy for PSMA-expressing tumors, represent clinically meaningful steps toward precision oncology. However, their benefits remain limited to defined subgroups, and further innovation is required. In summary, contemporary prostate cancer care is characterized by improved survival through treatment intensification and precision diagnostics, but continued progress will depend on overcoming resistance mechanisms and developing effective therapies for advanced, treatment-refractory disease.

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