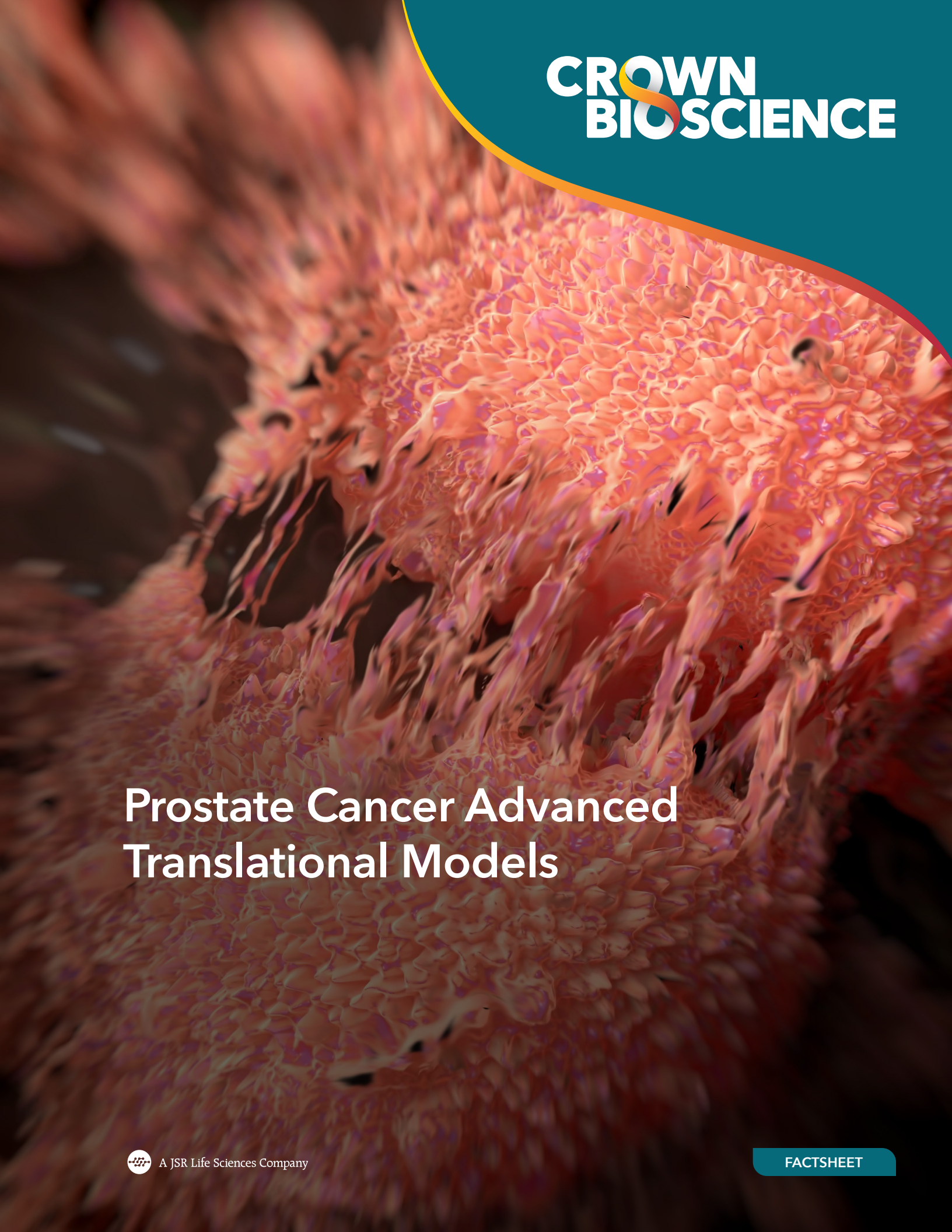


CROWN BIOSCIENCE

Prostate Cancer Advanced Translational Models

A JSR Life Sciences Company

FACTSHEET

Preclinical Prostate Cancer Models

At Crown Bioscience, we provide a comprehensive suite of **Prostate Cancer** models to advance targeted therapy and immuno-oncology research. From standard cell lines to patient-derived xenografts (PDX) and organoids, our platform accurately captures the heterogeneity of the disease, offering a physiologically relevant system to study androgen receptor (AR) signaling, target expression (PSMA, STEAP1), and drug efficacy.

Clinically Relevant Tumor Modeling – Access a diverse collection of models ranging from androgen-sensitive to castration-resistant prostate cancer (CRPC) and neuroendocrine types.

Enhanced Translational Insights – Leverage deep genomic and proteomic characterization, including RNAseq and IHC, to identify responders for Antibody-Drug Conjugates (ADCs) and bispecifics.

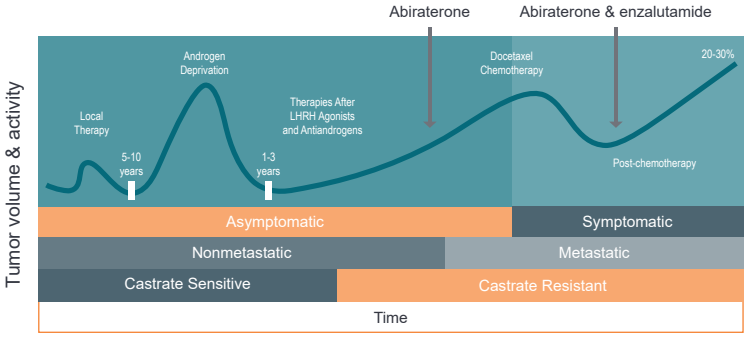
Accelerated and Scalable Drug Testing – Utilize our matched patient-derived organoids (PDO) for high-throughput *in vitro* screening before moving to *in vivo* validation.

Expanded Capabilities for Target Validation – Select models based on specific antigen density, including high, moderate, and negative expression of PSMA (FOLH1) and STEAP1.

Access to High-Quality Patient-Derived Material – Utilize well-characterized PDX models that retain the histological and genetic features of the original patient tumors.

Overview

Prostate cancer is a multistage disease with different treatment options (e.g., androgen deprivation therapy (ADT) for hormone responsive tumors or docetaxel for CRPC. Targeting AR signaling with novel therapeutics, such as abiraterone acetate and enzalutamide, has also been successful in treating CRPC. However, resistance and metastatic disease eventually emerges, presenting an urgent focus for current drug discovery efforts in prostate cancer research.



Note : This diagram represents typical disease progression.
Some patients are metastatic at diagnosis and are therefore still castration sensitive.

Catalyze Your Research with the Right Target Profile

Breakthrough discoveries start with the right model selection. Our prostate cancer platform is fully characterized for key surface antigens to give you more reliable and predictive results for targeted therapies.

High-Fidelity Target Expression

Validated H-scores for **PSMA** and **STEAP1** allow for precise evaluation of ADCs and radioligands.

Dual-Antigen Profiling

Unique "Double Positive" PDX models that express both FOLH1 and STEAP1, ideal for testing bispecific antibodies and combination therapies.

Metastatic and CRPC Phenotypes

Models derived from distinct metastatic sites, including bone, brain, and lymph nodes, to study CRPC mechanisms.

Androgen Receptor Diversity

A full spectrum of AR status, ranging from sensitive to splice variant AR-V7 expressers and completely androgen-independent phenotypes.

Matched Patient-Derived Organoids

Paired organoid and PDX models derived from the same patient tumor enable seamless translation from *in vitro* screening to *in vivo* validation

Distinct Molecular Subtypes

Representation of critical genetic drivers and rare subtypes, including TMPRSS2-ERG fusion positive and neuroendocrine prostate cancer.

Patient-Derived Xenograft (PDX) Models

Our prostate cancer PDX models retain clinically relevant treatment histories and biomarker profiles, enabling evaluation of standard-of-care therapies, resistance mechanisms, and biomarker-driven therapeutic strategies *in vivo*.

Model Name	Disease State / Origin	Patient Pretreatment	Castration Response	Docetaxel	AR-Targeted Therapy*	Key Biomarker Features
PR6511	CRPC, Primary	Hormone, Chemo	Partial sensitivity	Sensitive	Resistant (Enza, Abi)	PSA$^+$, PSMA$^+$, AR$^+$, PTEN loss
PR6512	CRPC, Primary	Hormone	Sensitive	Variable	Resistant (Enza, Abi)	PSA$^+$, PSMA$^+$, AR$^+$
PR6513	HR / mCSPC, Primary	Hormone	Sensitive	Partial response	Sensitive (Enza)	PSA$^+$, PSMA$^+$, AR$^+$, AR-V7$^+$
PR9582	mCRPC, LN Met	Hormone, Diethylstilbestrol	Partial sensitivity	Poor response	Sensitive (low-dose Enza)	PSA$^+$, AR$^+$, PTEN loss, PSMA low, KLK2 protein$^+$
PR9583	mCRPC, Bone Met	Diethylstilbestrol, Mitoxantrone	Sensitive	Moderate response	Sensitive (Enza)	PSA$^+$, PSMA$^+$, AR$^+$, KLK2 protein high
PR9585	mCRPC, Bladder Met	None	Resistant	Resistant	Resistant	PSA$^+$, PSMA$^+$, AR$^+$, PTEN loss
PR9586	mCRPC, Ascites	Hormone, Diethylstilbestrol	Partial sensitivity	Partial response	Partial response	PSA$^+$/-, AR heterogeneous, PTEN loss, KLK2 protein$^+$
PR9587	mCRPC, Rib/Bone Met	Ketoconazole, corticosteroids, Docetaxel, Enza	Partial sensitivity	Resistant	Resistant	PSA$^+$, AR$^+$, PTEN low, KLK2 protein high
PR9588	mCRPC, Rib Met	Lupron, Casodex	Resistant	Resistant	Resistant	PSA low, PSMA$^-$, AR$^-$
PR9675	Metastatic Prostate cancer	NA	NA	NA	NA	PSA status available

* AR-targeted therapy includes enzalutamide (Enza) and/or abiraterone (Abi)
† Castration sensitivity observed under specific experimental conditions

Cell Line Xenografts

Our standard cell line models serve as robust benchmarks for efficacy studies. We provide detailed IHC scoring for PSMA to ensure the correct control selection.

Model Name	Type	PSMA Status (IHC)	Key Characteristics
LNCaP clone FGC	Adenocarcinoma	Score 3+	Androgen-sensitive, metastatic lymph node derivative.
C4-2	LNCaP Derivative	Score 3+	Castration-resistant, bone metastatic model.
22RV-1	Carcinoma	Score 2.5+	Castration-resistant, expresses AR-V7 splice variant.
PC-3	Adenocarcinoma	Negative	Androgen-independent, high metastatic potential (bone).
DU-145	Carcinoma	Negative	Androgen-independent, brain metastasis derivative.
VCap	Carcinoma	Negative	Vertebral metastasis, TMPRSS2-ERG fusion positive.
NCI-H660	Neuroendocrine	Negative	Rare neuroendocrine prostate cancer model.

Patient-Derived Organoid Characterization

Our 3D organoid models are developed directly from patient tissue, preserving the original tumor architecture, receptor status, and drug response profiles.

Model Name	Subtype & Origin	AR Status (IHC / Mut)	PSMA (FOLH1) Status	SoC Drug Sensitivity	Key Features
PR6513B	Primary Adenocarcinoma	IHC 3+ Mutation: p.Q78_Q80del	High (IHC 3+) Gene Exp: 8.188	Resistant: Abiraterone, Enzalutamide, Cabazitaxel Sensitive: Docetaxel	High AR and PSMA expression; ideal for ADC testing.
PR9582B	mCRPC (Lymph Node Met)	IHC 3+ Mutation: p.Q77_Q80dup	Low (IHC 1+) Gene Exp: ~4.93	Resistant: Abiraterone, Enzalutamide Sensitive: Docetaxel, Cabazitaxel	Castration-resistant model with multi-drug resistance history.
PR9585B	mCRPC (Bladder Met)	Gene Exp: 1.882 Mutation: p.Q80dup	Moderate Gene Exp: 5.349	*Validation data available upon request*	Experimentally derived castration-resistant line.
PR9588B	Double Negative mCRPC	IHC 0+ No known AR mutation	Negative (IHC 0)	Resistant: Presumed to AR-targeted agents Sensitivity to chemotherapy pending validation	Double negative for PSMA and STEAP1

(Note: Gene expression values are Log2 transformed. IHC scoring performed on matched samples.)

Syngeneic Models

Our syngeneic prostate cancer models use host-matched tumor lines in immunocompetent mice, preserving native tumor–immune interactions and enabling accurate evaluation of immuno-oncology therapies.

Treatment	Dose / Route / Schedule	Observed Tumor Response	Responder Status
Anti-PD-1 (RMP1-14)	10 mg/kg, BIW	Minimal inhibition vs PBS	Non-Responder
Anti-mPD-1	10 mg/kg, i.p., BIW × 3 wks	Minimal inhibition vs PBS	Non-Responder
Anti-PD-L1 (10F.9G2)	5 mg/kg, BIW	Minimal inhibition vs PBS	Non-Responder
aCTLA-4 (9D9)	10 mg/kg, BIW	Minimal inhibition vs PBS	Non-Responder
Radiotherapy (RT)	3 Gy/animal, QD × 4 days (1 day off/on cycles)	Clear reduction in tumor growth	Responder
Anti-PD-1 + Radiotherapy	PD-1: 10 mg/kg BIW; RT: 3 Gy schedule	Enhanced inhibition vs RT or PD-1 alone	Responder
Cisplatin	2 mg/kg, Q4D	Clear reduction in tumor growth	Responder
Anti-PD-1 + Cisplatin	PD-1: 10 mg/kg BIW; Cisplatin: 2 mg/kg Q4D	Slightly greater inhibition vs Cisplatin alone	Responder
Gemcitabine	50 mg/kg, i.p., BIW × 2 doses (D0,D3)	Minor inhibition	Partial Responder
Gemcitabine + Anti-PD-1	Same schedule as above	Slight synergistic inhibition	Responder
Doxycycline (PO)	5 mg/mL, 200 µL/mouse, QD	Minimal inhibition	Non-Responder
Castration (post-inoculation)	Surgical	Variable inhibition depending on timing	Partial Responder

Note: Responder status is qualitative, inferred from tumor volume trends. Quantitative classification would require raw tumor volume data.

Genetically Engineered / Carcinogen-Induced Murine Prostate Models

Homografts of spontaneous murine tumors from GEMM or carcinogen-induced systems, fully studied in immunocompetent mice.

Model ID	Mutations	Pathology
mPR6003	TRAMP (Pbsn-SV40TTG)	Moderately differentiated adenocarcinoma (Pa), prostate cancer (P0)
mPR6065	PTEN (Flox/Flox); P53-/-	LipoSA – Sarcoma (P2)
mPR6066	PTEN (Flox/Flox); P53-/-	LipoSA – Sarcoma (P1)
mPR6135	KRAS (G12D); PTEN (Flox/Flox); Probasin-cre	Adenocarcinoma
mPR6189	Probasin-cre; PTEN (Flox/Flox); KRAS (G12D)	Adenocarcinoma
mPR6190	Probasin-cre; PTEN (Flox/Flox); KRAS (G12D)	Poorly differentiated adenocarcinoma
mPR6194	Probasin-cre; PTEN (Flox/Flox)	Adenocarcinoma
mPR6195	Probasin-cre; PTEN (Flox/Flox)	Adenocarcinoma
mPR6197	Probasin-cre; PTEN (Flox/Flox)	Adenocarcinoma

Comprehensive Model Selection: Validated for PSMA, STEAP1, and AR Status

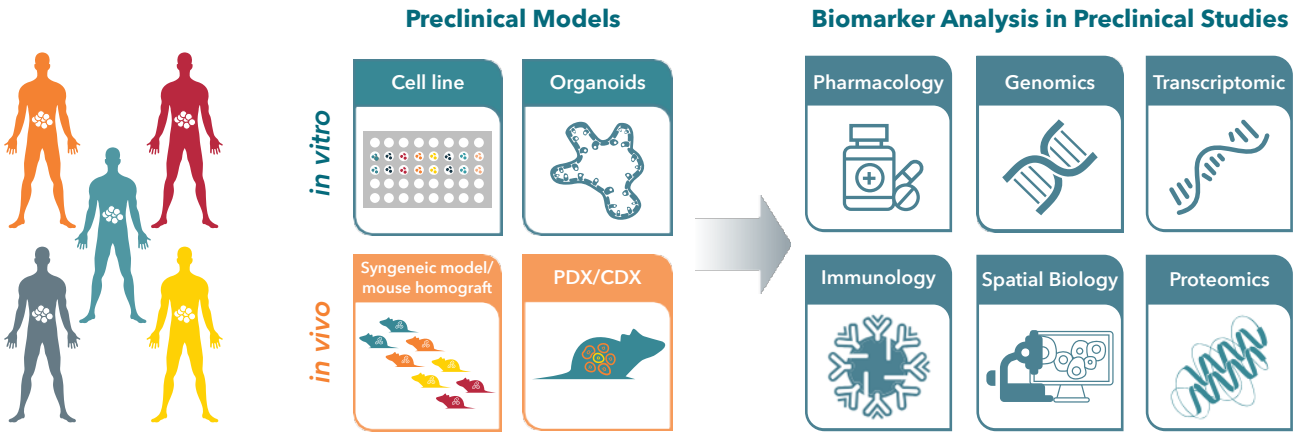
Accelerate your drug discovery with a platform fully characterized for key prostate cancer targets. From AR signaling to surface antigens like FOLH1 (PSMA) and STEAP1, we provide the specific phenotypes needed to validate your therapeutic candidates.

Platform	Description
Patient-Derived Biobanks	Well-characterized patient tissue containing patient demographics, genomics, and treatment history forms the foundation for meaningful studies.
3D Prostate Organoids	*In vitro* platforms that mimic the native tumor environment, preserving receptor status for robust drug screening.
CDX Models	Standard cell line models are fully characterized for PSMA and AR status to serve as robust benchmarks for efficacy studies.
PDX Models	Patient-derived xenograft models provide a predictive *in vivo* system to validate therapeutic efficacy.
Biomarker Analysis	In-depth characterization of drug response and resistance mechanisms through multi-omics and high-content imaging.

Seamless Transition from *In Vitro* to *In Vivo*

We offer a fully integrated platform that enables a smooth progression of research from early discovery to translational studies:

- **Patient-Derived Biobanks** – Well-characterized patient tissue containing patient demographics, genomics and treatment history forms the foundation for meaningful studies.
- **PDX Models** – Patient-derived xenograft models provide a predictive *in vivo* system to validate therapeutic efficacy.
- **Comprehensive Biomarker Analysis** – In-depth characterization of drug response and resistance mechanisms through multi-omics and advanced analytics.



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