

Original Article

Movember GAP1 PDX Project: An international collection of serially transplantable Prostate Cancer Patient-Derived Xenograft (PDX) models:¹

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Abstract

Background: While it has been challenging to establish prostate cancer patient-derived xenografts (PDXs), with a take rate of 10-40 percent and long latency time, multiple groups throughout the world have developed methods for the successful establishment of serially transplantable human prostate cancer PDXs using a variety of immune deficient mice. In 2014, the Movember Foundation launched a Global Action Plan 1 (GAP1) project to support an international collaborative prostate cancer PDX program involving eleven groups. Between these Movember consortium members, a total of 98 authenticated human prostate cancer PDXs were available for characterization. Eighty three of these were derived directly from patient material, and 15 were derived as variants of patient-derived material via serial passage in androgen deprived hosts. A major goal of the Movember GAP1 PDX project was to provide the prostate cancer research community with a summary of both the basic characteristics of the 98 available authenticated serially transplantable human prostate cancer PDX models and the appropriate contact information for collaborations. Herein we report a summary of these PDX models.

Methods: PDX models were established in immunocompromised mice via subcutaneous or subrenal-capsule implantation. Dual-label species (*i.e.* human vs. mouse) specific centromere and telomere Fluorescence In Situ Hybridization (FISH) and immuno-histochemical (IHC) staining of tissue microarrays containing replicates of the PDX models were used for characterization of expression of a number of phenotypic markers important for prostate cancer including AR (assessed by IHC and FISH), Ki67,

vimentin, RB1, P-Akt, chromogranin A (ChgA), p53, ERG, PTEN, PSMA and epithelial cytokeratins.

Results: Within this series of PDX models, the full spectrum of clinical disease stages is represented, including androgen-sensitive and castration-resistant primary and metastatic prostate adenocarcinomas as well as prostate carcinomas with neuroendocrine differentiation. The annotated clinical characteristics of these PDXs were correlated with their marker expression profile.

Conclusion: Our results demonstrate the clinical relevance of this series of PDXs as a platform for both basic science studies and therapeutic discovery/drug development. The present report provides the prostate cancer community with a summary of the basic characteristics and a contact information for collaborations using these models.

INTRODUCTION

The rapid advancement of technologies for assessing genomes and gene expression has identified the underlying genetic drivers of prostate cancer behavior, allowing these to be integrated with assessments of response to drugs and to develop predictive biomarkers of treatment outcome (1, 2). To accelerate the translational potential of these molecular discoveries, patient-derived xenografts (PDXs) are an important platform for drug development, enabling investigators to generate preclinical results that more accurately reflect the clinical responses occurring in patients. This is due to recognition that PDXs growing in immunocompromised mice retain key molecular aberrations present in patient specimens. These aberrations include mutations,

structural genomic events, epigenetic features, and gene expression programs driving their 3-dimensional growth, which is dependent on cancer cell autonomous, as well as infiltrating host cell-dependent processes needed within the tumor microenvironment (3-5). In addition, in contrast to *in vitro* experiments using prostate cancer cell lines, the use of prostate cancer PDXs *in vivo* allows evaluation of both, the anti-cancer efficacy and toxicity, thus defining the therapeutic index of new approaches to be evaluated simultaneously.

Over the last 50 years, many investigators developed and reported methods for establishing PDXs with good take rates for one generation (3). In contrast, the rate of generating and establishing enduring serially transplantable human prostate cancer PDXs using a variety of immune deficient mice (4) has been much lower. Consequently, the use of these prostate cancer PDXs has been limited with few serially transplantable lines distributed throughout the prostate cancer research community. There are very few prostate cancer PDX models in international PDX repositories (e.g. NCI, Novartis, Jackson Laboratories, Charles River and Champion) and thus the molecular diversity of human patient prostate cancer tumors is not well represented. In 2014, the Movember Foundation launched a Global Action Plan 1 (GAP1) project to support an international collaborative prostate cancer PDX program involving multiple groups throughout the world. A goal of this international collaborative program was to provide the prostate cancer research community with a summary of the basic characteristics of the existing serially transplantable prostate cancer PDX lines, together with the contact information

for the investigator who established each of these serially transplantable human prostate cancer PDXs (Figure 1). This information is presented in this publication.

MATERIALS AND METHODS

Acquisition of prostate cancer patient tissues and PDXs establishment

Numerous PDX models were established by multiple groups and contributed to the Movember GAP1 PDX project consortium for characterization. The initial establishment of the PDXs was performed according to several published protocols (3-16). An overview of the methodologies is provided below.

Patient samples and clinical data abstraction

Tissue collection for research was approved by the IRB of each institution: University of Washington, University of Texas, MD Anderson Cancer Center, Johns Hopkins University, Erasmus Medical Center, Monash University, Vancouver Prostate Cancer Centre, St. Vincent's Hospital Melbourne, Queensland University of Technology, Metro South Health, Innsbruck Medical University, University Hospital, University Hospital Bern Inselspital Basel, and University of York. Tumor specimens were acquired from patients who signed informed consent, and were obtained from palliative TURP, radical prostatectomy, cystoprostatectomy, resection of distant metastases at rapid autopsy in a manner which limited warm ischemic time as much as possible (aiming for 4–8 hours after death) or other procedures (e.g. bone marrow aspirate, or biopsy). Tumor tissues obtained between 1977 and 1992 for the Erasmus MC Rotterdam collection were a surplus surgical material, and were anonymized for research purposes, which according

to Dutch law did not require informed consent. Pertinent clinical information was extracted from the patients' charts, including age, PSA levels, treatments, and treatment responses when available.

PDX establishment

Harvested tumor tissues were evaluated by pathologists, viable tumor tissue was macro-dissected when appropriate, and tumor pieces were then prepared for implantation. All animal procedures were approved by individual Institutional Animal Care and Use Committee and their specific countries' animal welfare and handling guidelines of vertebrate animals used for experimental purposes. Various adult male immunodeficient mice were used for tissue implantation including athymic Nu/Nu (NU-Foxn1^{nu}), CB-17 SCID (CB17/lcr-Prkdcscid/lcrCrl), NOD-SCID, triple immune-deficient NOG, NSG (NOD.Cg-PrkdcScidIl2rgtm1Wji/Szj), and athymic NMRI (NMRI-Foxn1^{nu}) mice. Grafts were implanted at the subcutaneous site, or as subrenal grafts. At the time of grafting, mice were intact, castrated, or had androgen supplementation using silastic implants of testosterone pellet. Mice were monitored for up to 18 months post implantation for initial growth. Tumors that grew were serially passaged into new male mice under the same conditions and by the same method as the original implants. A line was considered established if there was active growth after at least five passages. Tumor samples were harvested from later passages (>3) for characterization.

Tissue microarray construction

Formalin-fixed paraffin-embedded PDX tissues were used for tissue microarray (TMA) construction. Three one-millimeter tissue cores from 1-3 different PDX tumors of each PDX line were punched and embedded into TMAs. Five- μ m sections were used for IHC and Fluorescence In Situ Hybridization (FISH) evaluation.

FISH analysis

Dual-label centromere and telomere FISH was performed on TMA tissue sections to identify human and mouse cell components according to published procedures (17). FISH was also performed to evaluate androgen receptor amplification as previously described (18). AR amplification was defined as a ratio of AR/centromere ≥ 2 with at least 4 AR signals; or as the presence of dense clusters of AR signals.

Immunohistochemistry

Immunohistochemistry was performed on TMA tissue sections for a series of markers known to be important in prostate cancer progression. Staining was performed by multiple investigators at multiple institutions. Conditions for each stain, including antibody details and staining methods, are provided in Table 1.

Pathological analysis and quantification of IHC

Immunohistochemical stains and FISH were visually examined. Immunoreactivity was assessed using criteria described in Table 2.

Statistical analysis

Association of selected markers expression with anatomical description and other disease parameters was evaluated using Pearson association test. Correlation of IHC markers expression was evaluated using Pearson correlation test. Unsupervised hierarchical clustering was generated using uncentered Pearson correlation and average linkage, with Cluster 3.0 software and JavaTreeview for visualization.

RESULTS and DISCUSSION

A total of 108 PDX models were available for characterization within the Movember consortium. Ninety three of these were derived directly from patient tumors and 15 were derived as a variant of the primary PDXs via serial passage in castrated male mice. Ten PDXs derived directly from patient material were excluded from further analysis based on negative cytokeratin immunoreactivity. These models represent Epstein-Barr Virus (EBV) associated human lymphomas that can develop from tumor infiltrating EBV-transformed B cells present in prostate cancer tissue when initially xenografted into immunodeficient mice (19, 20). The human origin of the epithelial cytokeratins AE1/AE3/PCK26-positive prostate cancer PDXs was confirmed by FISH analysis using human vs. mouse specific centromere and telomere probes; all 83 parental and 15 variant PDXs were shown to be of human origin. The 15 derived lines were established from the original PDXs that regrew after castration or removal of T supplementation, and are considered to be castration-resistant sublines. Within the 83 PDXs derived directly from patient material, the full spectrum of clinical disease stages is represented: primary and metastatic tumors, androgen-sensitive and castration-resistant adenocarcinomas, as well adenocarcinoma with neuroendocrine differentiation and neuroendocrine prostate cancer carcinomas (Table 3). Association of the annotated

clinical characteristics of the 83 parental PDX models with their expression of a number of phenotypic markers including AR (assessed by FISH and IHC), Ki67, vimentin, RB1, P-AKT, chromogranin A (ChgA), p53, ERG, FOXA1, PTEN, and PSMA were evaluated. Figure 2 and 3 show results that reached significance ($P < 0.05$) or trend ($P < 0.1$). Importantly, the overall proportion of PDXs that are positive or negative for these various phenotypic markers mimics distribution of these markers in prostate cancer clinical specimens (2) (Table 4). Moreover, in concordance with what is known about the biology of prostate cancer, the expression of AR was positively correlated with PSMA, ERG, FOXA1, PTEN and RB1 expression while negatively correlated with ChgA, Ki67, p53, vimentin and P-AKT (Supplemental Table 1). Clustering analysis separated the PDXs in two major clusters, which are mainly defined by high AR, FOXA1 and PSMA expression vs AR and PSMA negative PDXs with high vimentin and p53. In addition, AR negative tumors show enrichment for neuroendocrine differentiation. Clustering analysis of primary PDX lines with their castration-resistant sublines showed that PDX lines derived from the same tumor tended to cluster together and there was no clear separation based on castration response or hormonal status of the host. Two main clusters, mainly defined by high AR, FOXA1 and PSMA expression (left cluster) versus weak/ heterogeneous AR/ FOXA1/ PSMA, with absence of PTEN and high P-AKT (right cluster), were observed. This, suggests activation of the PI3K/AKT pathway (right cluster) (Figure 4). These results together clearly indicate the clinical relevance of these PDX models as a platform for therapeutic discovery/drug development and studies of prostate cancer biology.

In conclusion, while it has been challenging to establish serially transplantable prostate cancer PDX models with a take rate that is at best in the 10-40% range with a long latency time (21), there are now multiple prostate cancer PDX models available that can be used to advance our understanding of prostate cancer biology and help to identify new long-term effective treatment strategies to conquer this disease. Detailed information about all models is provided in Table 5 and 6, and the investigator contact information for additional details and/or requests for collaborations is presented in Table 7.

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Table 1

Antibodies Used for IHC

Catalogue #	Concentration/ dilution used	Antigen retrieval	IHC protocol/detection
760-2135	neat	Ventana Ultra Discovery	ChromoMap DAB kit
M3562	1:50	Target Retrieval Solution (Dako-CAT#: S1699)	Dako AutoStainer Plus
Ab8204	1:20 (4.5 µg/µL)	Boil for 7 minutes in 0.01M Na citrate	Dako Autostainer Plus
EPR3864	1:400	CC1: 950-124 - Ultra cell conditioning sol ultra CC1,60 minutes	Ventana Ultra Discovery - DISCOVERY® ChromoMap DAB Kit (RUO)
HNF3α (c-20)	0.2ug/ml	ER2 (Epitope Retrieval EDTA based pH 9.0) 30min	Leica BOND –MAX™ autostainer (Leica)
NCL-L-MM1	0.2ug/ml	ER2 (Epitope Retrieval EDTA based pH 9.0) 30min	Leica BOND –MAX™ autostainer (Leica)
DO-7	1:400	CC1: 950-124 - Ultra cell conditioning sol ultra CC1, 32 minutes	Ventana Ultra Discovery - DISCOVERY® ChromoMap DAB Kit (RUO)
#4060	1:50	Ventana Benchmark Ultra, Cell conditioning reagent 1	VentanaUltra View DAB IHC Detection Kit
M3620	1:75	Na citrate pH 6.0, 30 minutes in pressure cooker	ABC reagent (Vector Laboratories)
#9188	1:100	Na citrate pH 6.0, 30 minutes in pressure cooker	ABC reagent (Vector Laboratories)
OP66	1/50	Target Retrieval Solution (Dako-CAT#: S1699)	Dako AutoStainer Plus
M0725	1.025ug/ml	CC1 36 mins	Ventana Ultra

Table 2

Categorization of IHC Stains

Marker	Negative	Weak or heterogeneous immunoreactivity	Strong homogenous immunoreactivity
AE1/AE3/PCK26	only positive PDX were included in the analyses		
AR	0: negative	1: heterogeneous stain 50-98% cells	2: intensive homogeneous staining in 100 % cells
AR fish	0: not evaluable	1: Not amplified	2: amplified
ChgA	0: negative	1: >10% cells positive	
ERG	0: negative	1: weak or heterogeneous	2: >90% positive
FOXA1	0: negative	1: heterogeneous stain 50-98% cells	2 >90% positive, intensity 3
Ki67	0: negative	1: 5-50%	2: >50%
p53	0: negative	1: weak or heterogeneous	2: >90% intensive staining
P-AKT	0: negative	1: <50%	2: >50%
PSMA	0: negative	1: score ,20-99	2: score >99
PTEN	0: negative	1: IHC score >20	
RB1	0: negative	1: >10%	
vimentin	0: negative	1: weak stain <50%	2: intense stain >50% cells

Table 3

Summary of Characteristics of Donor Tumor

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	#	%
Anatomic Description		
Primary PC/Local Extension	38	46%
Metastatic PC	45	54%
Diagnosis		
Adenocarcinoma	35	42%
Metastatic Adenocarcinoma	32	39%
Neuroendocrine Differentiation	16	19%
Clinical State		
Androgen Naive/Sensitive	32	39%
Castration Resistant	51	61%
Procedure Type		
Radical Prostatectomy/Cytoprostatectomy	22	27%
Biopsy Core/Excisional Biopsy	16	19%
Bone Marrow Aspirate	3	4%
Autopsy	16	19%
Other	26	31%
Metastatic Site		
Bone	13	30%
Liver	7	16%
Lymph Nodes	12	27%
Other	12	27%

Table 4

Expression of Selected Markers in the Primary PDXs in the Movember PDX Collection

	negative		positive	
	#	%	#	%
AR	21	26%	62	74%
ChgA	67	81%	16	19%
ERG	60	72%	23	28%
FOXA1	2	2%	80	98%
ki67	16	20%	66	80%
p53	31	37%	52	63%
P-AKT	42	50%	41	49%
PSMA	33	40%	50	60%
PTEN	48	58%	35	42%
RB1	19	23%	63	77%
vimentin	54	68%	26	32%
	NOT AMPLIFIED		AMPLIFIED	
AR fish	53	83%	11	17%

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Table 5

Anatomic Description, Source, Diagnosis, Clinical State and Procedure Information

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PDX	Year Established	Anatomic Description	Source	Diagnosis	Clinical State	Gleason Score	Procedure
BM18	2001	Metastatic	Bone	Metastatic Adenocarcinoma	Androgen naïve	NA	Surgical reconstruction (pathological fracture)
LTL311	2008	Primary	Primary	Adenocarcinoma	Androgen naïve	NA	Biopsy - Core
LTL313A	2010	Primary	Prostate	Adenocarcinoma	Androgen naïve	5+3	Biopsy - Core
LTL313C	2010	Primary	Prostate	Adenocarcinoma	Androgen naïve	5+3	Biopsy - Core
LTL313D	2010	Primary	Prostate	Adenocarcinoma	Androgen naïve	5+3	Biopsy - Core
LTL313H	2009	Primary	Prostate	Adenocarcinoma	Androgen naïve	3+5	Biopsy - Core
LTL313HR	2013	primary	LTH313H	Adenocarcinoma	CRPC	NA	PDX
LTL331	2010	Primary	Prostate	Adenocarcinoma	Androgen naïve	5+4	Radical Prostatectomy
LTL331R	2010	Primary	LTL331	Adenocarcinoma	CRPC	NA	Biopsy - Core
LTL352	2009	Metastatic	Ureter	Metastatic Small Cell Carcinoma	CRPC	NA	Other
LTL370	2010	Metastatic	Soft Tissue	Metastatic Small Cell Carcinoma	CRPC	NA	Other
LTL412	2010	Metastatic	Lymph Node	Metastatic Adenocarcinoma	Androgen responsive	NA	Biopsy - Excisional
LTL418	2011	Primary	Prostate	Adenocarcinoma	Androgen naïve	4+3	Radical Prostatectomy
LTL467	2011	Primary	Prostate	Adenocarcinoma	Androgen responsive	4+5	Radical Prostatectomy
LTL471	2013	Primary	Prostate	Adenocarcinoma	Androgen naïve	4+4	Radical Prostatectomy
LTL484	2013	Primary	Prostate	Adenocarcinoma	CRPC	4+5	Radical Prostatectomy
LTL508	2013	Primary	Prostate	Adenocarcinoma	Androgen naïve	3+5	Radical Prostatectomy
LTL539	2014	Metastatic	Soft Tissue	Adenocarcinoma	Androgen naïve	NA	Biopsy - Excisional
LTL545	2012	Metastatic	NA	Metastatic Small Cell Carcinoma	CRPC	NA	Biopsy - Core
LuCaP 23.1	2013	Metastatic	Lymph Node	Metastatic Adenocarcinoma	CRPC	NA	Autopsy
LuCaP 23.12	1991	Metastatic	Liver	Metastatic Adenocarcinoma	crpc	NA	Autopsy
LuCaP 35	1993	Metastatic	Lymph Node	Metastatic Adenocarcinoma	CRPC	5+5	Biopsy - Excisional
LuCaP 35CR	2001	Metastatic	LuCaP 35	Metastatic Adenocarcinoma	CRPC	NA	PDX
LuCaP 49	1995	Metastatic	Soft Tissue	Metastatic Adenocarcinoma with Neuroendocrine Differentiation	CRPC	3+5	Biopsy - Excisional
LuCaP 58	1996	Metastatic	Lymph Node	Metastatic Adenocarcinoma	Androgen naïve	4+5	Biopsy - Excisional
LuCaP 70	1997	Metastatic	Liver	Metastatic Adenocarcinoma	CRPC	3+4	Autopsy
LuCaP 70CR	2011	Metastatic	LuCaP 70	Metastatic Adenocarcinoma	CRPC	3+4	PDX
LuCaP 73	1997	Primary	Prostate	Adenocarcinoma	CRPC	4+5	Radical prostatectomy
LuCaP 73CR	2011	Primary	LuCaP 73	Adenocarcinoma	CRPC	4+5	PDX
LuCaP 77	1998	Metastatic	Bone	Metastatic Adenocarcinoma	CRPC	NA	Autopsy
LuCaP 77CR	2010	Metastatic	LuCaP 77	Metastatic Adenocarcinoma	CRPC	NA	PDX
LuCaP 78	1998	Metastatic	Lymph Node	Metastatic Adenocarcinoma	CRPC	NA	Autopsy
LuCaP 81	1998	Metastatic	Lymph Node	Metastatic Adenocarcinoma	CRPC	NA	Autopsy
LuCaP 86.2	1999	Metastatic	Soft Tissue	Metastatic Adenocarcinoma	CRPC	NA	Biopsy - Excisional
LuCaP 86.2CR	2010	Metastatic	LuCaP 86.2	Metastatic Adenocarcinoma	CRPC	NA	PDX
LuCaP 93	1999	Primary	Prostate	Adenocarcinoma with Neuroendocrine Differentiation	Androgen responsive	NA	Other
LuCaP 96	1999	Primary	Prostate	Adenocarcinoma	Androgen responsive	5+4	TURP
LuCaP 96CR	2004	primary	LuCaP 96	Adenocarcinoma	CRPC	5+4	PDX
LuCaP 105	2000	Metastatic	Bone	Metastatic Adenocarcinoma	CRPC	5+3	Autopsy
LuCaP 105CR	2010	Metastatic	LuCaP 105	Metastatic Adenocarcinoma	CRPC	NA	PDX
LuCaP 136	2003	Metastatic	Serous cavity	Metastatic Adenocarcinoma	CRPC	5+5	Other
LuCaP 141	2005	Primary	Prostate	Adenocarcinoma	CRPC	NA	Other
LuCaP 145.1	2004	Metastatic	Liver	Metastatic Adenocarcinoma with Neuroendocrine Differentiation	CRPC	4+5	Autopsy
LuCaP 145.2	2005	Metastatic	Lymph Node	Metastatic Adenocarcinoma with Neuroendocrine Differentiation	CRPC	4+5	Autopsy
LuCaP 147	2005	Metastatic	Liver	Metastatic Adenocarcinoma	CRPC	4+5	Autopsy
LuCaP 147CR	2010	Metastatic	LuCaP 147	Metastatic Adenocarcinoma	CRPC	4+5	PDX
LuCaP 167	2012	Metastatic	Liver	Metastatic Adenocarcinoma	CRPC	NA	Autopsy
LvCaP-1	2009	Metastatic	Liver	Metastatic Adenocarcinoma	CRPC	4+4	Autopsy
LvCaP-1 CR	2012	Metastatic	LvCaP-1	Metastatic Adenocarcinoma	CRPC	4+4	PDX
LvCaP-2	2013	Metastatic	Liver	Metastatic Adenocarcinoma	CRPC	5+4	Autopsy

PDX	Year Established	Anatomic Description	Source	Diagnosis	Clinical State	Gleason Score	Procedure
MDA PCa 117-9	2004	Primary	Prostate	Adenocarcinoma	CRPC	4+5	Cystoprostatectomy
MDA PCa 118B	2003	Metastatic	Bone	Metastatic Adenocarcinoma	CRPC	NA	Biopsy - Core
MDA PCa 133-4	2007	Metastatic	Bone	Metastatic Adenocarcinoma	CRPC	NA	Surgical reconstruction (pathological fracture)
MDA PCa 144-4	2008	Primary	Prostate	Mixed Neuroendocrine Carcinoma and Adenocarcinoma	CRPC	NA	Pelvic Exenteration
MDA PCa 146-10	2009	Local Extension	Bladder	Mixed Neuroendocrine Carcinoma and Adenocarcinoma	CRPC	5+4	Cystoprostatectomy
MDA PCa 149-1	2009	Local Extension	Bladder	Adenocarcinoma	CRPC	4+5	Cystoprostatectomy
MDA PCa 150-1	2009	Metastatic	Bone	Metastatic Neuroendocrine Carcinoma	CRPC	NA	Craniectomy
MDA PCa 150-3	2009	Metastatic	Bone	Metastatic Neuroendocrine Carcinoma	CRPC	NA	Craniectomy
MDA PCa 153-14	2011	Metastatic	Thyroid	Metastatic Adenocarcinoma	CRPC	NA	Thyroidectomy
MDA PCa 153-7	2011	Metastatic	Thyroid	Metastatic Adenocarcinoma	CRPC	NA	Thyroidectomy
MDA PCa 170-1	2011	Primary	Prostate	Adenocarcinoma	CRPC	5+4	Cystoprostatectomy
MDA PCa 170-4	2011	Primary/Local Extension	Prostate/Bladder Neck	Adenocarcinoma	CRPC	5+5	Cystoprostatectomy
MDA PCa 173-2	2011	Primary	Prostate	Adenocarcinoma	Androgen naïve	3+4	TURP
MDA PCa 175-10	2011	Metastatic	Testis	Metastatic Adenocarcinoma	CRPC	NA	Orchiectomy
MDA PCa 175-6	2011	Metastatic	Testis	Metastatic Adenocarcinoma	CRPC	NA	Orchiectomy
MDA PCa 177-B	2011	Primary	Prostate	Adenocarcinoma	CRPC	NA	Biopsy - Core
MDA PCa 178-11	2011	Primary	Prostate	Adenocarcinoma	Androgen responsive	4+5	Radical Prostatectomy
MDA PCa 180-21	2011	Local Extension	Bladder	Adenocarcinoma	CRPC	5+4	Cystoprostatectomy
MDA PCa 180-30	2011	Primary	Prostate	Adenocarcinoma	CRPC	5+4	Cystoprostatectomy
MDA PCa 180-30C	2011	Primary	Prostate	Adenocarcinoma	CRPC	5+4	Cystoprostatectomy
MDA PCa 181-1	2012	Local Extension	Bladder	Mixed Neuroendocrine Carcinoma and Adenocarcinoma	CRPC	NA	Pelvic Exenteration
MDA PCa 183-A	2011	Metastatic	Bone	Metastatic Adenocarcinoma	Androgen naïve	NA	Bone Marrow Aspirate
MDA PCa 203-A	2014	Metastatic	Bone	Metastatic Adenocarcinoma	CRPC	NA	Bone Marrow Aspirate
MDA PCa 259-11	2014	Primary	Prostate	Adenocarcinoma	Androgen naïve	4+5	Radical Prostatectomy
MDA PCa 265-6	2014	Local Extension	Bladder	Adenocarcinoma with Neuroendocrine Differentiation	CRPC	NA	Cystoprostatectomy
MDA PCa 265-8	2014	Primary	Prostate	Adenocarcinoma with Neuroendocrine Differentiation	CRPC	NA	Cystoprostatectomy
MDA PCa 266-A	2014	Metastatic	Serous Cavity	Metastatic Adenocarcinoma	CRPC	NA	Paracentesis
MDA PCa 270-A	2015	Metastatic	Bone	Metastatic Adenocarcinoma	Androgen sensitive	NA	Bone Marrow Aspirate
MDA PCa 273-A	2015	Metastatic	Lymph Node	Metastatic Neuroendocrine Carcinoma	CRPC	NA	Biopsy - Core
MDA PCa 277-1	2015	Metastatic	Lymph Node	Metastatic Neuroendocrine Carcinoma	CRPC	NA	Lymph Node Dissection
MDA PCa 2b	1995	Metastatic	Bone	Metastatic Adenocarcinoma	CRPC	NA	Laminectomy
MDA PCa 2bC	1995	Metastatic	Bone	Metastatic Adenocarcinoma	CRPC	NA	Laminectomy
PC-82	1977	Local Extension	Prostate	Adenocarcinoma	Androgen naïve	NA	Radical Prostatectomy
PC-133	1981	Metastatic	Bone	Metastatic Adenocarcinoma	CRPC	NA	Autopsy
PC-135	1982	Primary	Prostate	Adenocarcinoma	Androgen naïve	NA	Radical Prostatectomy
PC-295	1990	Metastatic	Lymph Node	Metastatic Adenocarcinoma	Androgen naïve	NA	Other
PC-310	1990	Primary	Prostate	Adenocarcinoma	Androgen naïve	NA	Radical Prostatectomy
PC-324	1991	Local Extension	Prostate	Metastatic Adenocarcinoma with Neuroendocrine Differentiation	CRPC	NA	TURP
PC-339	1991	Local Extension	Prostate	Adenocarcinoma	CRPC	NA	TURP
PC-346B	1991	Local Extension	Prostate	Adenocarcinoma	Androgen responsive	NA	Other
PC-346BI	1995	Local Extension	PC346B	Adenocarcinoma	CRPC	NA	PDX
PC-346C	1991	Local Extension	Prostate	Adenocarcinoma	Androgen responsive	NA	Other
PC-346I	2016	Local Extension	PC346C	Adenocarcinoma	CRPC	NA	PDX
PC-346SIcas	1999	Local Extension	PC346C	Adenocarcinoma	CRPC	NA	PDX
PC-374	1992	Metastatic	Scrotal skin	Metastatic Adenocarcinoma	CRPC	NA	Other
PC-374F	1996	Metastatic	PC374	Metastatic Adenocarcinoma	CRPC	NA	PDX
PacMetUT1	2001	Metastatic	Lymph Node / Soft Tissue	Metastatic Adenocarcinoma	Androgen naïve	4+4	Biopsy - Core
SkCaP-1	2012	Metastatic	Lymph Node / Soft Tissue	Metastatic Adenocarcinoma	CRPC	4+3	Biopsy - Core

Table 6

Mouse strain, Grafting and IHC Information

PDX	Mouse Strain	Site of Grafting	Host	Diagnosis	Response to Castration	AR	CgA	ERG	FOXA1	ki67	p53	P-AKT	PSMA	PTEN	RB1	Vimentin	AR FISH
BM18	SCID	S.C.	Intact	Metastatic Adenocarcinoma	Yes	2	0	1	2	2	1	1	2	0	1	0	1
LTL311	NOD/SCID	Subrenal	Supplemented with T	Adenocarcinoma	NA	2	0	0	2	2	0	0	1	0	1	1	1
LTL313A	NOD/SCID	Subrenal	Supplemented with T	Adenocarcinoma	NA	2	0	2	1	2	1	2	2	2	1	0	1
LTL313C	NOD/SCID	Subrenal	Supplemented with T	Adenocarcinoma	NA	2	0	2	2	1	1	0	1	0	1	0	1
LTL313D	NOD/SCID	Subrenal	Supplemented with T	Adenocarcinoma	NA	2	0	2	0	1	1	1	0	1	0	1	0
LTL313H	NOD/SCID	Subrenal	Supplemented with T	Adenocarcinoma	NA	2	0	0	2	2	0	0	1	0	1	0	1
LTL313HR	NOD/SCID	Subrenal	Castrated	Adenocarcinoma	NA	2	0	1	2	1	1	0	2	2	1	0	1
LTL331	NOD/SCID	Subrenal	Supplemented with T	Adenocarcinoma	NA	NA	0	1	NA	NA	2	2	0	0	NA	NA	0
LTL331R	NOD/SCID	Subrenal	Castrated	Adenocarcinoma	No	0	1	0	1	2	0	0	0	2	1	2	1
LTL352	NOD/SCID	Subrenal	Intact	Metastatic Small Cell Carcinoma	No	0	1	0	2	2	0	0	0	2	0	0	1
LTL370	NOD/SCID	Subrenal	Intact	Metastatic Small Cell Carcinoma	No	0	1	0	2	2	2	2	0	0	0	0	1
LTL412	NOD/SCID	Subrenal	Supplemented with T	Metastatic Adenocarcinoma	NA	2	0	0	1	2	0	1	2	2	1	NA	1
LTL418	NOD/SCID	Subrenal	Supplemented with T	Adenocarcinoma	NA	2	0	0	2	2	1	0	2	0	1	0	1
LTL467	NOD/SCID	Subrenal	Supplemented with T	Adenocarcinoma	NA	2	0	0	2	2	1	2	1	0	1	2	1
LTL471	NOD/SCID	Subrenal	Supplemented with T	Adenocarcinoma	NA	2	0	0	2	2	0	0	2	2	1	NA	1
LTL484	NOD/SCID	Subrenal	Intact	Adenocarcinoma	NA	2	1	0	2	1	0	1	1	2	1	0	1
LTL508	NOD/SCID	Subrenal	Supplemented with T	Adenocarcinoma	NA	2	0	0	2	1	0	0	2	0	1	1	1
LTL539	NOD/SCID	Subrenal	Supplemented with T	Adenocarcinoma	NA	2	1	0	2	2	1	1	0	0	1	2	1
LTL545	NOD/SCID	Subrenal	Intact	Metastatic Small Cell Carcinoma	No	0	0	0	1	2	0	2	0	0	0	2	1
LuCaP 23.1	SCID	S.C.	Intact	Metastatic Adenocarcinoma	Yes	1	0	1	1	2	1	0	1	2	1	0	1
LuCaP 23.12	SCID	S.C.	Intact	Metastatic Adenocarcinoma	Yes	2	0	1	1	2	2	1	1	2	1	0	1
LuCaP 35	SCID	S.C.	Intact	Metastatic Adenocarcinoma	Yes	1	0	1	2	2	1	2	0	0	1	0	2
LuCaP 35CR	SCID	S.C.	Castrated	Metastatic Adenocarcinoma	No	1	0	1	1	1	1	1	0	0	1	0	2
LuCaP 49	SCID	S.C.	Intact	Metastatic Adenocarcinoma with Neuroendocrine Differentiation	No	0	0	0	1	2	1	1	0	0	0	1	1
LuCaP 58	SCID	S.C.	Intact	Metastatic Adenocarcinoma	NA	1	0	0	1	1	1	1	1	2	1	2	1
LuCaP 70	SCID	S.C.	Intact	Metastatic Adenocarcinoma	Yes	1	0	0	1	1	2	1	2	1	1	1	1
LuCaP 70CR	SCID	S.C.	Castrated	Metastatic Adenocarcinoma	No	1	0	0	1	2	1	0	1	0	1	1	1
LuCaP 73	SCID	S.C.	Intact	Adenocarcinoma	Yes	2	0	0	1	1	1	1	2	2	1	0	1
LuCaP 73CR	SCID	S.C.	Castrated	Adenocarcinoma	No	2	0	0	2	1	1	0	2	2	1	0	1
LuCaP 77	SCID	S.C.	Intact	Metastatic Adenocarcinoma	Yes	2	0	0	1	2	1	0	2	2	1	0	2
LuCaP 77CR	SCID	S.C.	Castrated	Metastatic Adenocarcinoma	No	2	0	0	1	2	1	0	2	2	1	1	1
LuCaP 78	SCID	S.C.	Intact	Metastatic Adenocarcinoma	Yes	2	0	0	1	2	1	0	0	1	1	0	1
LuCaP 81	SCID	S.C.	Intact	Metastatic Adenocarcinoma	NA	1	0	0	1	2	1	0	0	1	1	0	1
LuCaP 86.2	SCID	S.C.	Intact	Metastatic Adenocarcinoma	No	1	0	2	1	2	0	2	2	0	0	0	1
LuCaP 86.2CR	SCID	S.C.	Castrated	Metastatic Adenocarcinoma	No	1	0	1	1	2	0	2	2	0	0	0	1
LuCaP 93	SCID	S.C.	Intact	Adenocarcinoma with Neuroendocrine Differentiation	No	0	0	0	2	2	2	0	0	0	0	0	1
LuCaP 96	SCID	S.C.	Intact	Adenocarcinoma	Yes	2	0	0	2	2	0	0	2	0	1	0	1
LuCaP 96CR	SCID	S.C.	Castrated	Adenocarcinoma	No	2	0	0	1	2	0	0	2	0	1	0	2
LuCaP 105	SCID	S.C.	Intact	Metastatic Adenocarcinoma	NA	1	0	0	2	2	1	2	1	0	1	0	1
LuCaP 105CR	SCID	S.C.	Castrated	Metastatic Adenocarcinoma	No	2	0	0	1	2	1	2	1	0	1	0	1
LuCaP 136	SCID	S.C.	Intact	Metastatic Adenocarcinoma	Yes	1	0	0	1	2	0	0	0	0	1	0	NA
LuCaP 141	SCID	S.C.	Intact	Adenocarcinoma	Yes	2	1	0	2	2	0	2	1	2	1	1	1
LuCaP 145.1	SCID	S.C.	Intact	Metastatic Adenocarcinoma with Neuroendocrine Differentiation	No	0	1	0	1	2	1	0	0	1	0	2	1
LuCaP 145.2	SCID	S.C.	Intact	Metastatic Adenocarcinoma with Neuroendocrine Differentiation	No	0	0	0	1	2	2	0	0	1	0	0	2
LuCaP 147	SCID	S.C.	Intact	Metastatic Adenocarcinoma	NA	2	0	0	2	2	1	2	2	0	1	1	2
LuCaP 147CR	SCID	S.C.	Castrated	Metastatic Adenocarcinoma	No	2	0	0	2	2	1	1	2	0	1	0	1
LuCaP 167	SCID	S.C.	Intact	Metastatic Adenocarcinoma	Yes	2	0	0	2	2	1	0	0	1	1	0	1
LvCaP-1	NSG	S.C.	Intact	Metastatic Adenocarcinoma	Yes	2	0	0	2	2	2	2	0	0	1	2	2
LvCaP-1 CR	NSG	S.C.	Castrated	Metastatic Adenocarcinoma	No	2	1	0	2	2	1	0	2	0	1	NA	NA
LvCaP-2	NSG	S.C.	Intact	Metastatic Adenocarcinoma	Yes	2	0	0	2	2	0	0	0	1	0	0	2

PDX	Mouse Strain	Site of Grafting	Host	Diagnosis	Response to Castration	AR	CgA	ERG	FOXA1	ki67	p53	P-AKT	PSMA	PTEN	RB1	Vimentin	AR FISH
MDA PCa 117-9	SCID	S.C.	Intact	Adenocarcinoma	NA	2	1	0	2	2	0	0	1	2	1	0	2
MDA PCa 118B	SCID	S.C.	Intact	Metastatic Adenocarcinoma	NA	0	0	0	1	2	0	0	0	1	0	0	0
MDA PCa 133-4	SCID	S.C.	Intact	Metastatic Adenocarcinoma	NA	2	0	2	2	2	0	0	2	0	1	0	1
MDA PCa 144-4	SCID	S.C.	Intact	Neuroendocrine Carcinoma	NA	0	1	0	1	2	1	2	0	0	0	2	1
MDA PCa 146-10	SCID	S.C.	Intact	Neuroendocrine Carcinoma	NA	0	1	0	1	2	2	0	0	0	0	1	1
MDA PCa 149-1	SCID	S.C.	Intact	Adenocarcinoma	NA	2	0	2	2	2	0	1	0	0	1	0	2
MDA PCa 150-1	SCID	S.C.	Intact	Metastatic Neuroendocrine Carcinoma	NA	0	1	0	2	2	2	2	0	0	0	2	1
MDA PCa 150-3	SCID	S.C.	Intact	Metastatic Neuroendocrine Carcinoma	NA	0	1	0	1	2	2	2	0	0	0	2	1
MDA PCa 153-14	SCID	S.C.	Intact	Metastatic Adenocarcinoma	NA	2	0	2	2	1	1	0	2	0	1	0	1
MDA PCa 153-7	SCID	S.C.	Intact	Metastatic Adenocarcinoma	NA	2	0	2	2	1	1	0	2	0	1	0	1
MDA PCa 170-1	SCID	S.C.	Intact	Adenocarcinoma	NA	2	0	0	2	2	0	2	1	0	1	0	2
MDA PCa 170-4	SCID	S.C.	Intact	Adenocarcinoma	NA	1	0	0	2	2	0	2	2	0	1	0	2
MDA PCa 173-2	SCID	S.C.	Intact	Adenocarcinoma	NA	2	0	2	2	2	1	2	2	0	1	0	1
MDA PCa 175-10	SCID	S.C.	Intact	Metastatic Adenocarcinoma	NA	2	1	0	2	2	1	0	2	2	1	0	1
MDA PCa 175-6	SCID	S.C.	Intact	Metastatic Adenocarcinoma	NA	2	1	0	2	1	0	0	2	2	1	0	1
MDA PCa 177-B	SCID	S.C.	Intact	Adenocarcinoma	NA	0	0	0	1	2	2	1	0	0	0	2	1
MDA PCa 178-11	SCID	S.C.	Intact	Adenocarcinoma	NA	2	0	0	2	1	1	2	2	0	1	0	1
MDA PCa 180-21	SCID	S.C.	Intact	Adenocarcinoma	NA	2	0	0	2	2	1	0	1	2	1	0	1
MDA PCa 180-30	SCID	S.C.	Intact	Adenocarcinoma	NA	2	0	0	2	2	0	1	1	2	1	1	1
MDA PCa 180-30C	SCID	S.C.	Castrated	Adenocarcinoma	NA	2	0	0	2	2	1	0	1	2	1	0	1
MDA PCa 181-1	SCID	S.C.	Intact	Neuroendocrine Carcinoma	NA	0	0	0	1	2	2	1	0	0	0	0	1
MDA PCa 183-A	SCID	S.C.	Intact	Metastatic Adenocarcinoma	NA	2	0	2	2	1	0	2	1	0	1	0	1
MDA PCa 203-A	SCID	S.C.	Castrated	Metastatic Adenocarcinoma	NA	2	0	2	2	1	0	2	1	0	1	0	1
MDA PCa 259-11	SCID	S.C.	Intact	Adenocarcinoma	NA	2	0	1	2	2	2	2	1	0	0	0	0
MDA PCa 265-6	SCID	S.C.	Intact	Adenocarcinoma with Neuroendocrine Differentiation	NA	2	1	0	2	2	0	0	2	2	1	1	0
MDA PCa 265-8	SCID	S.C.	Intact	Adenocarcinoma with Neuroendocrine Differentiation	NA	2	1	0	2	2	0	0	2	2	1	0	0
MDA PCa 266-A	SCID	S.C.	Intact	Metastatic Adenocarcinoma	NA	1	0	1	2	2	2	2	0	0	0	2	0
MDA PCa 270-A	SCID	S.C.	Intact	Metastatic Adenocarcinoma	NA	2	0	0	2	1	0	0	2	2	1	0	0
MDA PCa 273-A	SCID	S.C.	Intact	Metastatic Neuroendocrine Carcinoma	NA	0	1	0	1	2	1	0	0	2	0	0	0
MDA PCa 277-1	SCID	S.C.	Intact	Metastatic Neuroendocrine Carcinoma	NA	0	0	0	1	2	0	0	0	2	1	0	0
MDA PCa 2b	SCID	S.C.	Intact	Metastatic Adenocarcinoma	NA	2	0	0	1	1	1	0	2	2	1	0	0
MDA PCa 2bC	SCID	S.C.	Castrated	Metastatic Adenocarcinoma	NA	2	0	0	2	1	1	0	2	2	1	0	1
PC-82	Nude	S.C.	Intact	Adenocarcinoma	Yes	2	0	2	2	2	1	0	2	0	1	1	2
PC-133	Nude	S.C.	Intact	Metastatic Adenocarcinoma	No	0	0	0	1	2	0	0	0	0	1	0	1
PC-135	Nude	S.C.	Intact	Adenocarcinoma	No	0	0	0	1	2	1	0	0	0	1	2	0
PC-295	Nude	S.C.	Supplemented with T	Metastatic Adenocarcinoma	Yes	2	0	2	2	2	0	0	2	1	1	2	0
PC-310	Nude	S.C.	Supplemented with T	Adenocarcinoma	Yes	2	0	2	2	2	0	0	2	2	1	0	0
PC-324	Nude	S.C.	Supplemented with T	Metastatic Adenocarcinoma with Neuroendocrine Differentiation	No	0	0	0	1	2	2	1	0	0	1	1	0
PC-339	Nude	S.C.	Supplemented with T	Adenocarcinoma	No	0	0	0	0	2	2	0	0	0	1	1	0
PC-346B	Nude	S.C.	Intact	Adenocarcinoma	Yes	1	0	0	1	2	1	2	1	0	1	0	1
PC-346BI	Nude	S.C.	Castrated	Adenocarcinoma	No	1	0	0	1	2	1	2	0	0	1	1	0
PC-346C	Nude	S.C.	Intact	Adenocarcinoma	Yes	2	0	0	2	2	1	2	1	0	1	0	0
PC-346I	Nude	S.C.	Castrated	Adenocarcinoma	No	2	0	0	1	2	1	1	0	0	1	1	1
PC-346SIcas	Nude	S.C.	Castrated	Adenocarcinoma	No	0	0	0	2	2	1	1	2	0	1	2	1
PC-374	Nude	S.C.	Intact	Metastatic Adenocarcinoma	No	0	0	0	1	2	1	2	0	0	1	1	1
PC-374F	Nude	S.C.	Castrated	Metastatic Adenocarcinoma	NA	1	0	0	2	2	1	2	0	0	1	0	0
PacMetUT1	NSG	S.C.	Intact	Metastatic Adenocarcinoma	No	1	0	1	2	2	1	0	0	1	1	0	1
SKCaP-1	NSG	S.C.	Supplemented with T	Metastatic Adenocarcinoma	Yes	1	0	1	1	2	0	2	1	0	1	0	NA

Table 7

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