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The 22Rv1 Prostate Cancer Cell Line Carries Mixed Genetic Ancestry: Implications for Prostate Cancer Health Disparities Research Using Pre-Clinical Models

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Abstract

BACKGROUND—Understanding how biological factors contribute to prostate cancer (PCa) health disparities requires mechanistic functional analysis of specific genes or pathways in pre-clinical cellular and animal models of this malignancy. The 22Rv1 human prostatic carcinoma cell line was originally derived from the parental CWR22R cell line. Although 22Rv1 has been well characterized and used in numerous mechanistic studies, no racial identifier has ever been disclosed for this cell line. In accordance with the need for racial diversity in cancer biospecimens and recent guidelines by the NIH on authentication of key biological resources, we sought to determine the ancestry of 22Rv1 and authenticate previously reported racial identifications for four other PCa cell lines.

METHODS—We used 29 established Ancestry Informative Marker (AIM) single nucleotide polymorphisms (SNPs) to conduct DNA ancestry analysis and assign ancestral proportions to a panel of five PCa cell lines that included 22Rv1, PC3, DU145, MDA-PCa-2b, and RC-77T/E.

RESULTS—We found that 22Rv1 carries mixed genetic ancestry. The main ancestry proportions for this cell line were 0.41 West African (AFR) and 0.42 European (EUR). In addition, we verified the previously reported racial identifications for PC3 (0.73 EUR), DU145 (0.63 EUR), MDA-PCa-2b (0.73 AFR), and RC-77T/E (0.74 AFR) cell lines.

CONCLUSIONS—Considering the mortality disparities associated with PCa, which disproportionately affect African American men, there remains a burden on the scientific community to diversify the availability of biospecimens, including cell lines, for mechanistic

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studies on potential biological mediators of these disparities. This study is beneficial by identifying another PCa cell line that carries substantial AFR ancestry. This finding may also open the door to new perspectives on previously published studies using this cell line.

Keywords

African American; ancestry analysis; health disparities; prostate cancer

INTRODUCTION

African American (AA) men and other men of African ancestry have the highest incidence and mortality rates of prostate cancer (PCa) in the world, resulting in the highest reported cancer health disparity.^{1–10} We use the term “African American” recognizing that while race is a social construct, racial classification remains extremely useful for describing general patterns of national health and health disparities, as most U.S. health data are reported by self-identified race.¹ Recent studies have provided compelling evidence in support of the notion that PCa health disparities result from the interplay of multiple factors, including biological/genetic factors.^{11–14} For instance, several recent studies have reported genomic differences between AA men with PCa and Caucasian or European American (EA) with PCa, suggesting a potential role for biological mediators in driving PCa mortality disparities.^{14–27} Understanding how these mediators contribute to increased PCa mortality in AA men requires mechanistic functional studies in pre-clinical cellular and animal models of PCa.

Given the inherent difficulties in conducting mechanistic studies in primary PCa cells, including transfection issues and high degree of molecular variability as these primary cells propagate *in vitro*,^{28–31} it is essential to have available a well-characterized, racially diverse, patient-derived cohort of immortalized cell lines representing different stages of PCa. Unfortunately, there is currently a lack of racial diversity in the human PCa cell lines that are commercially available for research. While there is over a dozen of commercially available human prostate cell lines of European ancestry that are representative of various prostate phenotypes such as normal and different PCa stages, there are only three cell lines identified as having African ancestry (Table I).^{32–45} This limits the scope of *in vitro* studies addressing mechanistic events involving potential biological factors associated with PCa health disparities.

Research into molecular and genetic mechanisms underlying PCa mortality disparities will be greatly advanced by the availability of pre-clinical cellular models of AA prostate tumors representing different stages of the disease.⁴⁶ There is a growing awareness of the need for biospecimens from AA populations that could be used for biomedical research, and an effort has been made by PCa researchers to develop AA prostate cell lines for cancer health disparities research.^{46–51} For example, RC-77N/E and RC-77T/E is a pair of African American prostate cell lines that were recently developed by one of the co-authors in this study (CY), and are available for research upon request.⁴⁶ While we recognize that the use of cell lines in cancer research has limitations, and that determinants of PCa health disparities may not be conclusively identified via *in vitro* studies, the use of racially diverse

PCa cell lines in mechanistic studies may provide important clues into contributing biological mediators.

The 22Rv1 human prostatic carcinoma cell line was derived from a xenograft of CWR22R cells serially propagated in mice after castration-induced regression.^{32,52} A number of biological characteristics were specified when 22Rv1 was derived, including epithelial lineage, DNA content, and cytogenetic data.³² The donor patient clinical information provided for the parental cell line CWR22R was the diagnosis of Stage D primary prostatic carcinoma with Gleason score of 9 advanced to osseous metastasis.⁵² While conducting studies using 22Rv1 cells we noticed that no racial identifier was ever released for this cell line or the parental CWR22R, and no subsequent DNA ancestry analysis has been conducted to identify its ethnicity.^{32,52} Recently, the National Institutes of Health established guidelines concerning the authentication of key biological resources, including cell lines, in order to ensure the identity and validity of the resource.⁵³ This authentication applies to racial identity if the cell line is being used in biological studies relevant to cancer health disparities. In this context, we sought to determine the ancestry of 22Rv1 and authenticate, using genetic ancestry analysis, a few previously racially identified cell lines used in PCa research. These included the PCa cell lines MDA-PCa-2b and RC-77T/E, previously reported as Black or AA,^{37,46} and the PC3 and DU145 cell lines, previously reported as Caucasian or EA.^{33,38}

To determine the ancestry of the 22Rv1 cell line we used a set of Ancestry Informative Markers (AIMs) validated as single nucleotide polymorphism (SNP) genotypes for population structure analyses.⁵⁴ PC3, DU145, MDA-PCa-2b, and RC-77T/E cell lines were included in the study to further validate their previously reported ethnicities and serve as controls for our SNP genotypes.^{33,37,38,46} Our analysis confirmed predominant European (EUR) ancestry in PC3 and DU145 cells as well as predominant West African (AFR) genetic ancestry in the MDA-PCa-2b and RC-77T/E cells. However, the 22Rv1 cell line was found to carry substantial AFR genetic ancestry. The racial classification of 22Rv1 as a mixed ancestry cell line with substantial AFR genetic ancestry expands diversity within the existing pool of human PCa cell lines. This finding may offer a new perspective on previously published studies using this cell line.

MATERIALS AND METHODS

Cell Culture

Cell lines were purchased from American Type Culture Collection (ATCC) and grown in a humidified incubator with 5% CO₂ at 37°C. Cells were routinely tested for mycoplasma contamination using MycoAlert™ PLUS Mycoplasma Detection Kit (Lonza, Cat. # LT07). PC3 (ATCC, Cat. # CRL-1435), DU145 (ATCC, Cat. # HTB-81), and 22Rv1 (ATCC, Cat. # CRL-2505) cell lines were cultured in RPMI 1640 medium (Corning, Cat. # 10-040-CV) supplemented with 10% fetal bovine serum (Corning, Cat. # 35010CV), penicillin-streptomycin (Corning, Cat. # 30001CI), and gentamicin (Gibco, Cat. # 15710064) as recommended by the supplier. MDA-PCa-2b (ATCC, Cat. # CRL-2422) cell line was cultured in F-12K medium (ATCC, Cat. # 30-2004) supplemented with 20% fetal bovine serum (Corning, Cat. # 35010CV), cholera toxin (Sigma-Aldrich, Cat. # C8052), epidermal

growth factor (Sigma-Aldrich, Cat. # E4127), α -phosphoethanolamine (Sigma-Aldrich, Cat. # P0503), hydrocortisone (Sigma-Aldrich, Cat. # H0888), selenious acid (ACROS Organics, Cat. # AC19887), bovine insulin (Sigma-Aldrich, Cat. # I6634), and penicillin-streptomycin as recommended by the supplier. 0.2% Normocin (Invivogen, Cat. # ANT-NR-1) was added to the medium for PC3, DU145, 22Rv1, and MDA-PCa-2b cell lines to prevent contamination by mycoplasma, bacteria, or fungi. The RC-77T/E cell line was cultured with keratinocyte serum-free medium (K-SFM) with L-glutamine supplemented with bovine pituitary extract and recombinant epidermal growth factor 1-53 (Life Technologies, Cat. # 17-005-042). Fungizone (0.4%, Gibco, Cat. # 15290018) was added to this medium to prevent contamination by yeast or multicellular fungi. To ensure proper cell attachment to culture plates, RC-77T/E cell line was cultured in 1% collagen-treated dishes (100mm $\times$ 20mm) (Life Technologies, Cat. # A1064401).

DNA Extraction

DNA was extracted using the QIAamp DNA mini kit (Qiagen, Valencia, CA, Cat. # 51104) following the manufacturer's protocol. Briefly, cells were grown in a monolayer at confluency no greater than 5×10^6 . Medium was aspirated and cells were washed with Dulbecco's phosphate-buffered saline (PBS) (Corning, Cat. # 21030CM) and then trypsinized with 0.25% trypsin (Corning, Cat. # 25-053-CI) to detach them from the culture flask. Cells were then collected in appropriate medium, transferred to a 1.5 ml microcentrifuge tube, and centrifuged for 5 minutes at $300 \times g$. Supernatant was removed and cell pellet was resuspended in PBS to a final volume of 200 μ l. After this, 20 μ l Proteinase K and 200 μ l of Qiagen buffer AL were added and the sample was incubated for 10 min at 56°C. Then, 200 μ l of ethanol was added, and DNA was purified using the columns provided in the kit according to the manufacturer's instructions. The samples yielded approximately 6 μ g of DNA with an A_{260}/A_{280} ratio of 1.7–1.9. The samples were diluted in distilled water and stored at -20°C. Samples were shipped to the UCLA Sequencing and Genotyping Core, Department of Human Genetics, David Geffen School of Medicine, University of California, Los Angeles for subsequent SNP genotyping.

Selection of Ancestry Informative Marker (AIM) SNPs

We used 29 Ancestry Informative Marker (AIM) SNPs validated in a previous study involving determination of admixture proportions in human continental populations.⁵⁴ We selected the smallest subset of 24 AIMs and 5 additional SNPs from the previous study, as a cost-and time-effective strategy, to determine the ancestry of the above-mentioned PCa cell lines of interest. All the 29 selected SNPs exhibited large allele frequency differences between four continental populations- AFR, EUR, Amer-Indian (AMI), and East Asian (EAS) in the previous study.⁵⁴ We confirmed the allele frequency differences between these ancestral groups for each of the 29 AIM SNPs using the NCBI dbSNP database (<https://www.ncbi.nlm.nih.gov/snp>).

SNP Genotyping

SNP genotyping was done using the Fluidigm Biomark HD system (South San Francisco, CA) at the UCLA Genotyping core facility. SNPtypeTM assays and reagents for each of the SNPs were purchased from Fluidigm. DNA samples and assays were loaded onto GT 96*96

Dynamic array and processed per Fluidigm protocol. The genotyping calls were made using Fluidigm SNP genotyping software. Each DNA sample was analyzed in triplicate to ensure accuracy.

DNA Ancestry Analysis

Data for the current study was analyzed as part of a larger project involving 1078 DNA samples. Markers showing different alleles at very high frequency in distinct putative parental populations were used to efficiently distinguish different populations and for the inference of ancestry membership proportions. In this study, we used 29 selected unlinked AIM SNPs as 29 loci for the STRUCTURE software version 2.3.4, for providing an estimation of genetic ancestry.^{55–56} Allele frequencies for the 29 AIM SNPs are provided in Table II. The software was installed and run locally after importing the SNP data for 1078 DNA samples in the form of a matrix where the data for samples were in rows and the loci were in columns (.txt file). For the determination of true K or the assumed number of populations, length of the Burnin period used was 5,000, the number of Markov Chain Monte Carlo (MCMC) replicates after the Burnins was 50,000 and K was set for 1–10 with 3 replicates for each K. True K was determined to be equal to 2~3 by L(K) (log likelihood of each K) method, using Structure Harvester with a zipped version of the results from STRUCTURE, as an input file.^{57–58} After the determination of true K, STRUCTURE was run three times, without any prior population assignment for an admixture population using 100,000 as the length of the Burnin period and 500,000 as the number of MCMC replicates after the burnins, for K=2 and K=3. The missing value was represented as -9, the maximum value of ALPHA (ALPHAMAX) was 10.0 and the SD of proposal for updating ALPHA was 0.025, while rest of the parameters used were default. The three clusters obtained as an output from STRUCTURE were assigned EUR, AFR, and AMI ancestries by crosschecking the allele frequencies for AIMs of multiple DNA samples within each cluster against those found in NCBI dbSNP. The bar plot was generated by STRUCTURE for visualization of ancestral proportions in each sample. PC3 and DU145 cells along with MDA-PCa-2b and RC-77T/E cells served as controls for EUR and AFR ancestries, respectively, and aligned well with the results where K=3.

RESULTS

Informative Ancestry Markers for Distinguishing Ancestral Proportions

An initial subset of 24 AIM SNPs was selected from a validated larger set of 128 SNPs previously identified to define critical genome candidate regions and used to characterize samples from diverse population groups (Table II).⁵⁴ This subset of 24 AIMs contained specific SNPs capable of distinguishing the following four continent populations: EUR, AFR, AMI, and EAS.⁵⁴ Five additional AIMs from the validated larger set of 128 AIMs were added to the smaller subset of 24 AIMs, bringing our total quantity of AIMs used for DNA ancestry analysis to 29 (Table II).

Determination of Ancestral Proportions of Prostate Cancer Cell Lines

Each PCa cell line analyzed in our study possessed heterogeneous ancestral proportions (Figure 1, Table III). However, for each cell line where ATCC provided a racial

classification, we observed a predominant ancestral proportion that was consistent with what was previously reported. For instance, PC3 and DU145 cell line samples were included in our analysis as EUR controls, and MDA-PCa-2b and RC-77T/E as AFR controls based on their previously reported racial classification.^{33,37,38,46} PC3 is classified by ATCC as Caucasian and was found to carry 0.73 proportion of EUR ancestry. This cell line was also found to carry 0.19 AFR and 0.08 AMI ancestral proportions. DU145 is also classified by ATCC as Caucasian and was found to carry 0.63 proportion of EUR ancestry. Interestingly, DU145 was also found to carry 0.28 AFR and 0.09 AMI ancestral proportions. MDA-PCa-2b is classified by ATCC as Black and was found to carry 0.73 proportion of AFR ancestry as well as 0.13 AMI and 0.14 EUR ancestral proportions. RC-77T/E is not available through ATCC, however it was reported by one of the authors of this study (C. Yates) as an AA cell line,⁴⁶ and our results confirmed this. RC-77T/E was found to carry 0.74 proportion of AFR ancestry as well as 0.09 AMI and 0.17 EUR ancestral proportions. To date, there has never been a racial classification assigned to 22Rv1. Our results indicated the following ancestral proportions for 22Rv1: 0.41 AFR, 0.17 AMI, and 0.42 EUR. Therefore, 22Rv1 should be considered as a mixed ancestry cell line.

DISCUSSION

In concordance with recent NIH guidelines requiring authentication of cell lines, this study sought to determine the racial identity of 22Rv1 PCa cells since racial classification/ethnicity for this cell line has never been reported to ATCC or previously published.^{32,52,53} It has been our experience in personal communication with other groups that that 22Rv1 is widely considered as a EA PCa cell line, and we had no reason to explore this assumption until we began incorporating various EA and AA cell lines in our studies.^{27,59,60} We initially conducted a thorough literature search and contacted ATCC to determine if there was any indication of racial identification for either 22Rv1 or its parental cell line CWR22R. In addition, we communicated with key authors of the initial reports on the characterization of both cell lines to determine if there was any racial identification linked to the tumor biospecimen from which these cell lines were derived.^{32,52} Unfortunately, these efforts did not provide any indication as to the racial classification of these cell lines. This lack of information prompted us to conduct DNA ancestry analysis on 22Rv1 cells and provide this information to the PCa research community and the scientific community at large. We felt this was a very important issue given the need for a comprehensive panel of racially diverse patient-derived cell lines for mechanistic studies on potential biological mediators of PCa mortality disparities.

SNP genotyping and subsequent DNA ancestry analysis were chosen as appropriate methods to determine the ancestry for 22Rv1. Other investigators have shown that differences in allele frequencies can be used to identify continental population groups.^{61,62} This is because several studies have demonstrated that large differences in allele frequencies between two or more continental populations can be attributed to many individual SNPs distributed throughout the genome.^{63–67} In this study, 29 AIM SNPs were chosen from a validated larger set of 128 SNPs previously identified by others to define critical genome candidate regions and used to characterize samples from diverse population groups.⁵⁴ Differences in population structure were addressed by extending genetic associations to minority groups

that include extensive admixture between continents.⁵⁴ This genetic association extension was essential to evaluate ethnic disparities in disease risk among multiethnic and admixed populations as a large number of whole genome association studies have evaluated populations of predominantly EUR ancestry.⁵⁴

Our analysis revealed that the 22Rv1 PCa cell line carries AFR genetic ancestry, with almost equal proportions for AFR and EUR ancestry (41% and 42%, respectively). While this study authenticates the previously reported racial identification of several PCa cell lines (PC3, DU145, MDA-PC-2b, and RC-77T/E), it establishes for the first time the ancestral composition of 22Rv1. Because 22Rv1 was derived from a xenograft of CWR22R which was serially propagated in mice,^{32,52} it would be appropriate to also assign this mixed ancestral racial classification to CWR22R. The implications of 22Rv1 and CWR22R carrying AFR genetic ancestry are far reaching as genetic factors associated with AFR ancestry have been recently linked to prostate tumor aggressive properties.^{15–26} Further studies are needed to determine if these cell lines carry any of these factors. In addition, our findings open the door for closely examining previous studies using 22Rv1 and CWR22R cells under the new perspective that these cell lines carry almost equal proportions of AFR and EUR genetic ancestry.

We suggest that 22Rv1 and CWR22R can be added to the current roster of PCa cell lines that are classified as Black, AA, or carry substantial AFR genetic ancestry, including MDA-PCa-2b, E006AA-hT, RC-77N/E, and RC-77T/E.^{33,36,45} Alternatively, they could be classified in a separate category as racially mixed ancestry cell lines. We cannot rule out the possibility that CWR22R was originally derived from a Hispanic male of Caribbean heritage as the proportions of AFR, AMI, and EUR genetic ancestry found in the 22RV1 cell line are common in men from this geographical region.^{68–70} For example, recent studies have highlighted the high tri-hybrid admixture (Native American, European, West African) of the Puerto Rican population.^{68–69} Knowledge that 22Rv1 carries substantial AFR ancestry would be beneficial to efforts to expand the selection of racially diverse cell lines for *in vitro* mechanistic studies focusing on potential determinants PCa mortality disparities that could be attributable to genetically- and racially-driven tumor biological differences. It is important to note that while this study contributes to the increasing diversity of PCa cell lines, thus far all available AA PCa cell lines are derived from patients with metastatic disease and tumors with functional androgen and glucocorticoid receptors. There remains a critical need for AA PCa cell lines derived from metastatic androgen-independent tumors (e.g. equivalent to the EA PC3 and DU145). This would be important for mechanistic studies on different disease stages using AA PCa cell lines.

CONCLUSIONS

The field of PCa health disparities research continues to advance, with recent attention to potential biological determinants or mediators of these disparities that could be mechanistically examined using a panel of racially diverse, patient-derived cell lines. The identification of 22Rv1 as a mixed ancestry PCa cell line carrying substantial AFR genetic ancestry provides an additional resource to advance molecular/cellular research in the context of PCa health disparities. While there still remains a deficit in the availability of AA

PCa cell lines and clinical biospecimens, the trend towards acquiring additional specimens appears to be growing.^{47–51} The knowledge to be gained from mechanistic studies incorporating biospecimens (cell lines plus tumor samples) from AA men will not only have the potential to reduce the mortality disparities associated with this malignancy but also benefit the field of PCa research in general.

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References

1. DeSantis CE, Siegel RL, Sauer AG, Miller KD, Fedewa SA, Alcaraz KI, Jemal A. Cancer Statistics for African Americans, 2016: Progress and opportunities in reducing racial disparities. *CA Cancer J Clin.* 2016; 66(4):290–308. [PubMed: 26910411]
2. Siegel RL, Miller KD, Jemal A. Cancer Statistics, 2017. *CA Cancer J Clin.* 2017; 67(1):7–30. [PubMed: 28055103]
3. Mahal BA, Chen YW, Muralidhar V, Mahal AR, Choueiri TK, Hoffman KE, Hu JC, Sweeney CJ, Yu JB, Feng FY, Kim SP, Beard CJ, Martin NE, Trinh QD, Nguyen PL. Racial disparities in prostate cancer outcome among prostate-specific antigen screening eligible populations in the United States. *Ann Oncol.* 2017; 28(5):1098–1104. [PubMed: 28453693]
4. Tsodikov A, Gulati R, de Carvalho TM, Heijnsdijk EAM, Hunter-Merrill RA, Mariotto AB, de Koning HJ, Etzioni R. Is prostate cancer different in black men? Answers from 3 natural history models. *Cancer.* 2017; doi: 10.1002/cncr.30687
5. Singh GK, Jemal A. Socioeconomic and racial/ethnic disparities in cancer mortality, incidence, and survival in the United States, 1950–2014: Over six decades of changing patterns and widening inequalities. *J Environ Public Health.* 2017; 2017:2819372. [PubMed: 28408935]
6. Krok-Schoen JL, Fisher JL, Baltic RD, Paskett ED. White-Black differences in cancer incidence, stage at diagnosis, and survival among adults aged 85 years and older in the United States. *Cancer Epidemiol Biomarkers Prev.* 2016; 25(11):1517–1523. [PubMed: 27528599]
7. Benjamins MR, Hunt BR, Raleigh SM, Hirschtick JL, Hughes MM. Racial disparities in prostate cancer mortality in the 50 largest US cities. *Cancer Epidemiol.* 2016; 44:125–131. [PubMed: 27566470]
8. Kelly SP, Rosenberg PS, Anderson WF, Andreotti G, Younes N, Cleary SD, Cook MB. Trends in the incidence of fatal prostate cancer in the United States by race. *Eur Urol.* 2017; 71(2):195–201. [PubMed: 27476048]
9. Kinlock BL, Thorpe RJ Jr, Howard DL, Bowie JV, Ross LE, Fakunle DO, LaVeist TA. Racial disparity in time between first diagnosis and initial treatment of prostate cancer. *Cancer Control.* 2016; 23(1):47–51. [PubMed: 27009456]
10. DeSantis CE, Siegel RL, Sauer AG, Miller KD, Fedewa SA, Alcaraz KI, Jemal A. Cancer statistics for African Americans, 2016: Progress and opportunities in reducing racial disparities. *CA Cancer J Clin.* 2016; 66(4):290–308. [PubMed: 26910411]
11. Deshmukh SK, Azim S, Ahmad A, Zubair H, Tyagi N, Srivastava SK, Bhardwaj A, Singh S, Rocconi RP, Singh AP. Biological basis of cancer health disparities: Resources and challenges for research. *Am J Cancer Res.* 2017; 7(1):1–12. [PubMed: 28123843]

12. Rebbeck TR. Prostate cancer genetics: Variation by race, ethnicity, and geography. Prostate cancer genetics: Variation by race, ethnicity, and geography. *Semin Radiat Oncol.* 2017; 27(1):3–10. [PubMed: 27986209]
13. Tan DS, Mok TS, Rebbeck TR. Cancer genomics: Diversity and disparity across ethnicity and geography. *J Clin Oncol.* 2016; 34(1):91–101. [PubMed: 26578615]
14. Jones J, Grizzle W, Wang H, Yates C. MicroRNAs that affect prostate cancer: Emphasis on prostate cancer in African Americans. *Biotech Histochem.* 2013; 88(7):410–24. [PubMed: 23901944]
15. Gusev A, Shi H, Kichaev G, Pomerantz M, Li F, Long HW, Ingles SA, Kittles RA, Strom SS, Rybicki BA, Nemesure B, Isaacs WB, Zheng W, Pettaway CA, Yeboah ED, Tettey Y, Biritwum RB, Adjei AA, Tay E, Truelove A, Niwa S, Chokkalingam AP, John EM, Murphy AB, Signorello LB, Carpten J, Leske MC, Wu SY, Hennis AJ, Neslund-Dudas C, Hsing AW, Chu L, Goodman PJ, Klein EA, Witte JS, Casey G, Kaggwa S, Cook MB, Stram DO, Blot WJ, Eeles RA, Easton D, Kote-Jarai Z, Al Olama AA, Benlloch S, Muir K, Giles GG, Southey MC, Fitzgerald LM, Gronberg H, Wiklund F, Aly M, Henderson BE, Schleutker J, Wahlfors T, Tammela TL, Nordestgaard BG, Key TJ, Travis RC, Neal DE, Donovan JL, Hamdy FC, Pharoah P, Pashayan N, Khaw KT, Stanford JL, Thibodeau SN, McDonnell SK, Schaid DJ, Maier C, Vogel W, Luedeke M, Herkommer K, Kibel AS, Cbulski C, Wokolorczyk D, Kluzniak W, Cannon-Albright L, Teerlink C, Brenner H, Dieffenbach AK, Arndt V, Park JY, Sellers TA, Lin HY, Slavov C, Kaneva R, Mitev V, Batra J, Spurdle A, Clements JA, Teixeira MR, Pandha H, Michael A, Paulo P, Maia S, Kierzek A, Conti DV, Albanes D, Berg C, Berndt SI, Campa D, Crawford ED, Diver WR, Gapstur SM, Gaziano JM, Giovannucci E, Hoover R, Hunter DJ, Johansson M, Kraft P, Le Marchand L, Lindstrom S, Navarro C, Overvad K, Riboli E, Siddiq A, Stevens VL, Trichopoulos D, Vineis P, Yeager M, Trynka G, Raychaudhuri S, Schumacher FR, Price AL, Freedman ML, Haiman CA, Pasaniuc B. PRACTICAL consortium. Atlas of prostate cancer heritability in European and African-American men pinpoints tissue-specific regulation. *Nat Commun.* 2016; 7:10979. [PubMed: 27052111]
16. Han Y, Rand KA, Hazelett DJ, Ingles SA, Kittles RA, Strom SS, Rybicki BA, Nemesure B, Isaacs WB, Stanford JL, Zheng W, Schumacher FR, Berndt S, Wang Z, Xu J, Rohland N, Reich D, Tandon A, Pasaniuc B, Allen A, Quinque D, Mallick S, Notani D, Ronsenfeld MG, Jayani RS, Kolb S, Gapstur SM, Stevens VL, Pettaway CA, Yeboah ED, Tettey Y, Biritwum RB, Adjei AA, Tay E, Truelove A, Niwa S, Chokkalingam AP, John EM, Murphy AB, Signorello LB, Carpten J, Leske MC, Wu SY, Hennis AJ, Neslund-Dudas C, Hsing AW, Chu L, Goodman PJ, Klein EA, Zheng SL, Witte JS, Casey G, Lubwama A, Pooler LC, Sheng X, Coetzee GA, Cook MB, Chanock SJ, Stram DO, Watya S, Blot WJ, Conti DV, Henderson BE, Haiman CA. Prostate cancer susceptibility in men of African ancestry at 8q24. *J Natl Cancer Inst.* 2016; 108(7) Pii:djv431.
17. Rand KA, Rohland N, Tandon A, Stram A, Sheng X, Do R, Pasaniuc B, Allen A, Quinque D, Mallick S, Le Marchand L, Kaggwa S, Lubwama A, Stram DO, Watya S, Henderson BE, Conti DV, Reich D, Haiman CA. African Ancestry Prostate Cancer GWAS Consortium; ELLIPSE/GAME-ON Consortium. Whole-exome sequencing of over 4100 men of African ancestry and prostate cancer risk. *Hum Mol Genet.* 2016; 25(2):371–81. [PubMed: 26604137]
18. Han Y, Signorello LB, Strom SS, Kittles RA, Rybicki BA, Stanford JL, Goodman PJ, Berndt SI, Carpten J, Casey G, Chu L, Conti DV, Rand KA, Diver WR, Hennis AJ, John EM, Kibel AS, Klein EA, Kolb S, Le Marchand L, Leske MC, Murphy AB, Neslund-Dudas, Park JY, Pettaway C, Rebbeck TR, Gapstur SM, Zheng SL, Wu SY, Witte JS, Xu J, Isaacs W, Ingles SA, Hsing A, Easton DF, Eeles RA, Schumacher FR, Chanock S, Nemesure B, Blot WJ, Stram DO, Henderson BE, Haiman CA. PRACTICAL Consortium; ELLIPSE GAME-ON Consortium. Generalizability of established prostate cancer risk variants in men of African ancestry. *Int J Cancer.* 2015; 136(5): 1210–7. [PubMed: 25044450]
19. Powell IJ, Dyson G, Land S, Ruterbusch J, Bock CH, Lenk S, Herawi M, Everson R, Giroux CN, Schwartz AG, Bollig-Fischer A. Genes associated with prostate cancer are differentially expressed in African American and European American men. *Cancer Epidemiol Biomarkers Prev.* 2013; 22(5):891–7. [PubMed: 23515145]
20. Hoffman RM, Gilliland FD, Eley JW, Harlan LC, Stephenson RA, Stanford JL, Albertson PC, Hamilton AS, Hunt WC, Potosky AL. Racial and ethnic differences in advanced-stage prostate cancer: the Prostate Cancer Outcomes Study. *J Natl Cancer Inst.* 2001; 93(5):388–95. [PubMed: 11238701]

21. Batai K, Murphy AB, Nonn L, Kittles RA. Vitamin D and immune response: Implications for prostate cancer in African Americans. *Front Immunol.* 2016 Feb 22;7:53. [PubMed: 26941739]
22. Wallace TA, Prueitt RL, Yi M, Howe TM, Gillespie JW, Yfantis HG, Stephens RM, Caporaso NE, Loffredo CA, Ambis S. Tumor immunobiological differences in prostate cancer between African-American and European-American men. *Cancer Res.* 2008; 68:927–36. [PubMed: 18245496]
23. Kinseth MA, Jia Z, Rahmatpanah F, Sawyers A, Sutton M, Wang-Rodriguez J, Mercola D, McGuire KL. Expression differences between African American and Caucasian prostate cancer tissue reveals that stroma is the site of aggressive changes. *Int J Cancer.* 2014; 134:81–91. [PubMed: 23754304]
24. Reams RR, Agrawal D, Davis M, Yoder S, Odedina F, Kumar N, Higginbotham JM, Akinremi T, Suther S, Soliman KF. Microarray comparison of prostate tumor gene expression in African-American and Caucasian American males: a pilot project study. *Infect Agent Cancer.* 2009; 4:S3. [PubMed: 19208208]
25. Haiman CA, Chen GK, Blot WJ, Strom SS, Berndt SI, Kittles RA, Rybicki BA, Isaacs WB, Ingles SA, Stanford JL, Diver WR, Witte JS, Chanock SJ, Kolb S, Signorello LB, Yamamura Y, Neslund-Dudas C, Thun MJ, Murphy A, Casey G, Sheng X, Wan P, Pooler LC, Monroe KR, Waters KM, Le Marchand L, Kolonel LN, Stram DO, Henderson BE. Characterizing genetic risk at known prostate cancer susceptibility loci in African Americans. *PLoS Genet.* 2011; 7(5):e1001387. [PubMed: 21637779]
26. Wang BD, Ceniccola K, Hwang S, Andrawis R, Horvath A, Freedman JA, Olender J, Knapp S, Ching T, Garmire L, Patel V, Garcia-Blanco MA, Patierno SR, Lee NH. Alternative splicing promotes tumour aggressiveness and drug resistance in African American prostate cancer. *Nat Commun.* 2017; 8:15921. [PubMed: 28665395]
27. Sanchez TW, Zhang G, Li J, Dai L, Mirshahidi S, Wall NR, Yates C, Wilson C, Montgomery S, Zhang JY, Casiano CA. Immunoseroproteomic profiling in African American men with prostate cancer: Evidence for an autoantibody response to glycolysis and plasminogen-associated proteins. *Mol Cell Proteomics.* 2016; 15(12):3564–3580. [PubMed: 27742740]
28. Caspar A, Mostertz J, Leymann M, Ziegler P, Evert K, Evert M, Zimmermann U, Brandenburg LO, Burchardt M, Stope MB. In vitro cultivation of primary prostate cancer cells alters the molecular biomarker pattern. *In Vivo.* 2016; 30(5):573–9. [PubMed: 27566074]
29. Karaki S, Benizri S, Mejias R, Baylot V, Branger N, Nguyen T, Vialet B, Oumzil K, Barthelemy P, Rocchi P. Lipid-oligonucleotide conjugates improve cellular uptake and efficiency of TCTP-antisense in castration-resistant prostate cancer. *J Control Release.* 2017; 258:1–9. [PubMed: 28472637]
30. Bai WK, Wu ZH, Shen E, Zhang JZ, Hu B. The improvement of liposome-mediated transfection of pEGFP DNA into human prostate cancer cells by combining low-frequency and low-energy ultrasound with microbubbles. *Oncol Rep.* 2012; 27(2):475–80. [PubMed: 22020492]
31. Chen B, Pan R, Askhatova D, Chen P. Effective small interfering RNA delivery in vitro via a new stearylated cationic peptide. *Int J Nanomedicine.* 2015; 10:3303–14. [PubMed: 25999710]
32. Sramkoski RM, Pretlow TG 2nd, Giaconia JM, Pretlow TP, Schwartz S, Sy MS, Marengo SR, Rhim JS, Zhang D, Jacobberger JW. A new human prostate carcinoma cell line, 22Rv1. *In Vitro Cell Dev Biol Anim.* 1999; 35(7):403–9. [PubMed: 10462204]
33. Stone KR, Mickey DD, Wunderli H, Mickey GH, Paulson DF. Isolation of a human prostate carcinoma cell line (DU 145). *Int J Cancer.* 1978; 21(3):274–81. [PubMed: 631930]
34. Koochekpour S, Willard SS, Shourideh M, Ali S, Liu C, Azabdaftari G, Saleem M, Attwood K. Establishment and characterization of a highly tumorigenic African American prostate cancer cell line, E006AA-hT. *Int J Biol Sci.* 2014; 10(8):834–45. [PubMed: 25076860]
35. Klein KA, Reiter RE, Redula J, Moradi H, Zhu XL, Brothman AR, Lamb DJ, Marcelli M, Belldgrun A, Witte ON, Sawyers CL. Progression of metastatic human prostate cancer to androgen independence in immunodeficient SCID mice. *Nat Med.* 1997; 3(4):402–8. [PubMed: 9095173]
36. Horoszewicz JS, Leong SS, Kawinski E, Karr JP, Rosenthal H, Chu TM, Mirand EA, Murphy GP. LNCaP model of human prostatic carcinoma. *Cancer Res.* 1983; 43(4):1809–18. [PubMed: 6831420]

37. Navone NM, Olive M, Ozen M, Davis R, Troncso P, Tu SM, Johnston D, Pollack A, Pathak S, von Eschenbach AC, Logothetis CJ. Establishment of two human prostate cancer cell lines derived from a single bone metastasis. *Clin Cancer Res.* 1997; 3(12 Pt 1):2493–500. [PubMed: 9815652]
38. Kaighn ME, Narayan KS, Ohnuki Y, Lechner JF, Jones LW. Establishment and characterization of a human prostatic carcinoma cell line (PC-3). *Invest Urol.* 1979; 17(1):16–23. [PubMed: 447482]
39. Webber MM, Trakul N, Thraves PS, Bello-DeOcampo D, Chu WW, Storto PD, Huard TK, Rhim JS, Williams DE. A human prostatic stromal myofibroblast cell line WPMY-1: a model for stromal-epithelial interactions in prostatic neoplasia. *Carcinogenesis.* 1999; 20(7):1185–92. [PubMed: 10383888]
40. Webber MM, Bello D, Kleinman HK, Wartinger DD, Williams DE, Rhim JS. Prostate specific antigen and androgen receptor induction and characterization of an immortalized adult human prostatic epithelial cell line. *Carcinogenesis.* 1996; 17(8):1641–6. [PubMed: 8761420]
41. Weijerman PC, Romijn HC, Peehl DM. Human papilloma virus type 18 DNA immortalized cell lines from the human prostate epithelium. *Prog Clin Biol Res.* 1994; 386:67–9. [PubMed: 7800701]
42. Bello D, Webber MM, Kleinman HK, Wartinger DD, Rhim JS. Androgen responsive adult human prostatic epithelial cell lines immortalized by human papillomavirus 18. *Carcinogenesis.* 1997; 18(6):1215–23. [PubMed: 9214605]
43. Loberg RD, St John LN, Day LL, Neeley CK, Pienta KJ. Development of the VCaP androgen-independent model of prostate cancer. *Urol Oncol.* 2006; 24(2):161–8. [PubMed: 16520280]
44. Pinthus JH, Waks T, Schindler DG, Harmelin A, Said JW, Beldegrun A, Ramon J, Eshhar Z. WISH-PC2: A unique xenograft model of human prostatic small cell carcinoma. *Cancer Res.* 2000; 60(23):6563–7. [PubMed: 11118033]
45. Webber MM, Quader ST, Kleinman HK, Bello-DeOcampo D, Storto PD, Bice G, DeMendonca-Calaca W, Williams DE. Human cell lines as an in vitro/in vivo model for prostate carcinogenesis and progression. *Prostate.* 2001; 47(1):1–13. [PubMed: 11304724]
46. Theodore S, Sharp S, Zhou J, Turner T, Li H, Miki J, Ji Y, Patel V, Yates C, Rhim JS. Establishment and characterization of a pair of non-malignant and malignant tumor derived cell lines from an African American prostate cancer patient. *Int J Oncol.* 2010; 37(6):1477–82. [PubMed: 21042716]
47. Hagiwara N, Berry-Bobovski L, Francis C, Ramsey L, Chapman RA, Albrecht TL. Unexpected findings in the exploration of African American underrepresentation in biospecimen collection and biobanks. *J Cancer Educ.* 2014; 29(3):580–7. [PubMed: 24243440]
48. Luque JS, Quinn GP, Montel-Ishino FA, Arevalo M, Bynum SA, Noel-Thomas S, Wells KJ, Gwede CK, Meade CD. Tampa Bay Community Cancer Network Partners. Formative research on perceptions of biobanking: what community members think. *J Cancer Educ.* 2012; 27(1):91–9. [PubMed: 21927867]
49. Patel YR, Carr KA, Magjuka D, Mohammadi Y, Dropcho EF, Reed AD, Moore ML, Waddell MJ, Shedd-Steele R, Sweeney CJ, Hahn NM. Successful recruitment of healthy African American men to genomic studies from high-volume community health fairs: implications for future genomic research in minority populations. *Cancer.* 2012; 118(4):1075–82. [PubMed: 21766294]
50. Dash C, Wallington SF, Muthra S, Dodson E, Mandleblatt J, Adams-Campbell LL. Disparities in knowledge and willingness to donate research biospecimens: a mixed-methods study in an underserved urban community. *J Community Genet.* 2014; 5(4):329–36. [PubMed: 24771039]
51. Dang JH, Rodriguez EM, Luque JS, Erwin DO, Meade CD, Chen MS Jr. Engaging diverse populations about biospecimen donation for cancer research. *J Community Genet.* 2014; 5(4):313–27. [PubMed: 24664489]
52. Pretlow TG, Wolman SR, Micale MA, Pelley RJ, Kursh ED, Resnick MI, Bodner DR, Jaccobberger JW, Delmoro CM, Giaconia JM. Xenografts of primary human prostatic carcinoma. *Journal of the National Cancer Institute.* 1993; 85(5)
53. National Institute of Health. Implementing Rigor and Transparency in NIH & AHRQ Research Grant Applications. Notice Number: NOT-OD-16–011 Release Date: October 9, 2015.
54. Kosoy R, Nassir R, Tian C, White PA, Butler LM, Silva G, Kittles R, Alarcon-Riquelme ME, Gregersen PK, Belmont JW, De La Vega FM, Seldin MF. Ancestry Informative Marker sets for

determining continental origin and admixture proportions in common populations in America. *Hum Mutat.* 2009; 30(1):69–78. [PubMed: 18683858]

55. Falush D, Stephens M, Pritchard JK. Inference of population structure using multilocus genotype data: linked loci and correlated allele frequencies. *Genetics.* 2003; 164(4):1567–1587. [PubMed: 12930761]
56. Pritchard JK, Stephens M, Donnelly P. Inference of population structure using multilocus genotype data. *Genetics.* 2000; 155(2):945–959. [PubMed: 10835412]
57. Rosenberg NA, Burke T, Elo K, Feldman MW, Freidlin PJ, Groenen MA, Hillel J, Maki-Tanila A, Tixier-Boichard M, Vignal A, Wimmers K, Weigend S. Empirical evaluation of genetic clustering methods using multilocus genotypes from 20 chicken breeds. *Genetics.* 2001; 159(2):699–713. [PubMed: 11606545]
58. Earl DA, vonHoldt BM. STRUCTURE HARVESTER: a website and program for visualizing STRUCTURE output and implementing the Evanno method. *Conservation Genet Resour.* 2012; 4(2):359–361.
59. Basu A, Banerjee H, Rojas H, Martinez SR, Roy S, Jia Z, Lilly MB, De Leon M, Casiano CA. Differential expression of peroxiredoxins in prostate cancer: consistent upregulation of PRDX3 and PRDX4. *Prostate.* 2011; 71(7):755–65. [PubMed: 21031435]
60. Basu A, Drame A, Munoz R, Gijssbers R, Debyser Z, De Leon M, Casiano CA. Pathway specific gene expression profiling reveals oxidative stress genes potentially regulated by transcription co-activator LEDGF/p75 in prostate cancer cells. *Prostate.* 2012; 72(6):597–611. [PubMed: 21796653]
61. Rosenberg NA, Li LM, Ward R, Pritchard JK. Informativeness of genetic markers for inference of ancestry. *Am J Hum Genet.* 2003; 73(6):1402–1422. [PubMed: 14631557]
62. Rosenberg NA, Mahajan S, Ramachandran S, Zhao C, Pritchard JK, Feldman MW. Clines, clusters, and the effect of study design on the inference of human population structure. *PLoS Genet.* 2005; 1(6):e70. [PubMed: 16355252]
63. Mao X, Bigham AW, Mei R, Gutierrez G, Weiss KM, Brutsaert TD, Leon-Velarde F, Moore LG, Vargas E, McKeigue PM, Shriver MD, Parra EJ. A genome-wide admixture mapping panel for Hispanic/Latino populations. *Am J Hum Genet.* 2007; 80(6):1171–1178. [PubMed: 17503334]
64. Price AL, Patterson N, Yu F, Cox DR, Waliszewska A, McDonald GJ, Tandon A, Schirmer C, Neubauer J, Bedoya G, Duque C, Villegas A, Bortolini MC, Salzano FM, Gallo C, Mazzotti G, Tello-Ruiz M, Riba L, Aguilar-Salinas CA, Canizales-Quinteros S, Menjivar M, Klitz W, Henderson B, Haiman CA, Winkler C, Tusie-Luna T, Ruiz-Linares A, Reich D. A genome-wide admixture map for Latino populations. *Am J Hum Genet.* 2007; 80(6):1024–1036. [PubMed: 17503322]
65. Smith MW, Patterson N, Lautenberger JA, Truelove AL, McDonald GJ, Waliszewska A, Kessing BD, Malasky MJ, Scafe C, Le E, De Jager PL, Mignault AA, Yi Z, De The G, Essex M, Sankale JL, Moore JH, Poku K, Phair JP, Goedert JJ, Vlahov D, Williams SM, Tishkoff SA, Winkler CA, De La Vega FM, Woodage T, Sninsky JJ, Hafler DA, Altshuler D, Gilbert DA, O'Brien SJ, Reich D. A high-density admixture map for disease gene discovery in african americans. *Am J Hum Genet.* 2004; 74(5):1001–1013. [PubMed: 15088270]
66. Tian C, Hinds DA, Shigeta R, Adler SG, Lee A, Pahl MV, Silva G, Belmont JW, Hanson RL, Knowler WC, Gregersen PK, Ballinger DG, Seldin MF. A genome-wide single-nucleotide-polymorphism panel for Mexican American admixture mapping. *Am J Hum Genet.* 2007; 80(6):1014–1023. [PubMed: 17557415]
67. Tian C, Hinds DA, Shigeta R, Kittles R, Ballinger DG, Seldin MF. A genome-wide single-nucleotide-polymorphism panel with high ancestry information for african american admixture mapping. *Am J Hum Genet.* 2006; 79(4):640–649. [PubMed: 16960800]
68. Irizarry-Ramirez M, Kittles RA, Wang X, Salgado-Montilla J, Nogueras-Gonzalez GM, Sanchez-Ortiz R, Guerrios L, Rivera K, Shah E, Prokhorova I, Roberson P, Troncoso P, Pettaway CA. Genetic ancestry and prostate cancer susceptibility SNPs in Puerto Rican and African American men. *Prostate.* 2017; 77:1118–1127. [PubMed: 28543179]
69. Via M, Gignoux CR, Roth LA, Fejerman L, Galanter J, Choudhry S, Toro-Labrador G, Viera-Vera J, Oleksyk TK, Beckman K, Ziv E, Risch N, Burchard EG, Martinez-Cruzado JC. History shaped

the geographic distribution of genomic admixture on the island of Puerto Rico. PLoS ONE. 2011; 6:e16513. [PubMed: 21304981]

70. Torres JB, Doura MB, Keita SO, Kittles RA. Y chromosome lineages in men of West African descent. PLoS ONE. 2012; 7:e29687. [PubMed: 22295064]

Figure 1. DNA Ancestry Analysis Reveals Ancestral Proportions of Prostate Cancer Cell Lines

DNA was isolated and extracted from five PCa cell lines and submitted to the UCLA genomics core for SNP genotyping. Cell lines included EUR controls PC3 and DU145, AFR controls MDA-PCa-2b and RC-77T/E, and the racially ambiguous 22Rv1. DNA samples were analyzed in triplicate to ensure accuracy. The bar plot shown in this figure was generated using STRUCTURE for visualization of the ancestral proportion in each sample. Subsequent analysis of population substructure was also conducted using STRUCTURE, a model-based clustering software able to detect the underlying genetic population among a set of individuals genotyped at multiple markers with the help of a bayesian approach (<http://pritch.bsd.uchicago.edu/software/structure2>).⁵⁷ Using STRUCTURE, we were able to compute the proportion of the genome of the cell lines originating from each inferred population. Blue: AFR ancestry; Green: AMI ancestry; Red: EUR ancestry.

Table I

Commonly Used Commercially Available Prostate Cell Lines

Cell Line	Site of Origin	Race/Ethnicity	Source	Reference
22Rv1	Primary xenograft	Not Available	ATCC	Sramkoski RM <i>et al.</i> 1999 ³²
DU145	Brain metastasis	Caucasian	ATCC	Stone KR <i>et al.</i> 1978 ³³
E006AA-hT	Primary tumor	African American	ATCC	Koochekpour S <i>et al.</i> 2014 ³⁴
LAPC-3	Prostate xenograft	Not Available	ATCC	Klein KA <i>et al.</i> 1997 ³⁵
LAPC-4	Lymph node metastasis	Not Available	ATCC	Klein KA <i>et al.</i> 1997 ³⁵
LNCaP	Lymph node metastasis	Caucasian	ATCC	Horoscewicz JS <i>et al.</i> 1983 ³⁶
MDA-PCa-2a	Bone metastasis	Black	ATCC	Navone NM <i>et al.</i> 1997 ³⁷
MDA-PCa-2b	Bone metastasis	Black	ATCC	Navone NM <i>et al.</i> 1997 ³⁷
PC3	Bone metastasis	Caucasian	ATCC	Kaighn ME <i>et al.</i> 1979 ³⁸
PrEC	Normal human primary prostate epithelial cells	Variable	Lonza	Not applicable
PrSC/WPMY-1	Normal prostate/stroma	Caucasian	ATCC, Lonza	Webber MM <i>et al.</i> 1999 ³⁹
PWR-1E	Normal prostate	Caucasian	ATCC	Webber MM <i>et al.</i> 1996 ⁴⁰
PZ-HPV-7	Normal prostate/epithelial	Caucasian	ATCC	Weijerman PC, <i>et al.</i> 1994 ⁴¹
RWPE-1	Normal prostate	Caucasian, White	ATCC	Bello D <i>et al.</i> 1997 ⁴²
RWPE2	Normal prostate	Caucasian, White	ATCC	Bello D <i>et al.</i> 1997 ⁴²
RWPE2-W99	Normal prostate	Caucasian, White	ATCC	Bello D <i>et al.</i> 1997 ⁴²
VCaP	Vertebral metastasis	Caucasian	ATCC	Loberg RD <i>et al.</i> 2006 ⁴³
WISH-PC2	Neuroendocrine xenograft prostate small-cell carcinoma	Caucasian	ATCC	Pinthus JH <i>et al.</i> 2008 ⁴⁴
WPE1-NA22	Prostate	Caucasian, White	ATCC	Webber MM <i>et al.</i> 2001 ⁴⁵
WPE1-NB11	Normal prostate	Caucasian	ATCC	Webber MM <i>et al.</i> 2001 ⁴⁵
WPE1-NB14	Normal prostate/epithelial	Caucasian, White	ATCC	Bello D <i>et al.</i> 1997 ⁴²
WPE1-NB26	Normal prostate	Caucasian, White	ATCC	Webber MM <i>et al.</i> 2001 ⁴⁵

ATCC, American Type Culture Collection

Table II

Validated Ancestry Informative Marker Single Nucleotide Polymorphisms to Determine Ancestry Assignment of Prostate Cancer Cell Lines

	dbSNP ID	Chromosome Number	Nucleotide Change	Allele 1 frequency	
				EUR	AFR
1	rs4891825	18	G/A	0.89	0.09
2	rs9530435	13	T/C	0.79	0.07
3	rs1040045	6	T/C	0.73	0.10
4	rs7554936	1	T/C	0.34	0.99
5	rs772262	12	G/A	0.06	0.87
6	rs3784230	14	T/C	0.58	0.00
7	rs260690	2	C/A	0.91	0.36
8	rs10007810	4	G/A	0.25	0.96
9	rs7657799	4	T/G	0.05	0.86
10	rs6548616	3	T/C	0.25	0.96
11	rs4908343	1	G/A	0.82	0.04
12	rs11652805	17	T/C	0.14	0.98
13	rs9522149	13	T/C	0.76	0.03
14	rs10108270	8	C/A	0.35	0.97
15	rs6451722	5	G/A	0.24	0.90
16	rs9319336	13	T/C	0.05	0.11
17	rs870347	5	T/G	0.95	0.95
18	rs7803075	7	G/A	0.29	1.00
19	rs2416791	12	G/A	0.08	0.94
20	rs6422347	5	G/A	0.10	0.89
21	rs7997709	13	T/C	0.06	0.23
22	rs731257	7	G/A	0.09	0.02
23	rs4918842	10	T/C	0.15	0.09
24	rs10496971	2	T/G	0.09	0.06
25	rs12629908	3	G/A	0.05	0.16
26	rs3907047	20	C/T	0.06	0.00
27	rs1513181	3	C/T	0.14	0.21
28	rs1471939	8	C/T	0.17	0.32

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dbSNP ID	Chromosome Number	Nucleotide Change	Allele 1 frequency		
			EUR	AFR	
29	rs2125345	17	A/G	0.32	0.99

dbSNP, NCBI Single Nucleotide Polymorphism database; EUR, European; AFR, West African

Table III

Ancestry Proportions in Panel of Prostate Cancer Cell Lines

Cell Line	Proportion of Ancestry		
	AFR	AMI	EUR
PC3	0.19	0.08	0.73
DU145	0.28	0.09	0.63
MDA-PCa-2b	0.73	0.13	0.14
RC-77T/E	0.74	0.09	0.17
22Rv1	0.41	0.17	0.42

Samples were analyzed in triplicates to ensure accuracy.

AFR, African ancestry; AMI, Ameri-Indian ancestry; EUR, European ancestry.