AWARD NUMBER: **W81XWH-19-1-0726**

TITLE: Carcinoma-Associated Fibroblasts from African American Prostate Cancer Promote Aggressive Tumors: Implications for Developing Novel Therapy

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REPORT DATE: October 2023

TYPE OF REPORT: Annual Report

PREPARED FOR: U.S. Army Medical Research and Development Command
Fort Detrick, Maryland 21702-5012

DISTRIBUTION STATEMENT: Approved for Public Release;
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REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

Public reporting burden for this collection of information is estimated to average 1 hour per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing this collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden to Department of Defense, Washington Headquarters Services, Directorate for Information Operations and Reports (0704-0188), 1215 Jefferson Davis Highway, Suite 1204, Arlington, VA 22202-4302. Respondents should be aware that notwithstanding any other provision of law, no person shall be subject to any penalty for failing to comply with a collection of information if it does not display a currently valid OMB control number. **PLEASE DO NOT RETURN YOUR FORM TO THE ABOVE ADDRESS.**

1. REPORT DATE	**2. REPORT TYPE**	**3. DATES COVERED**
October 2023	Annual	09/30/2022 to 09/29/2023

4. TITLE AND SUBTITLE

Carcinoma-Associated Fibroblasts from African American Prostate Cancer Promote Aggressive Tumors: Implications for Developing Novel Therapy

5a. CONTRACT NUMBER W81XWH-19-1-0726

5b. GRANT NUMBER

5c. PROGRAM ELEMENT NUMBER

6. AUTHOR(S)

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5d. PROJECT NUMBER

5e. TASK NUMBER

5f. WORK UNIT NUMBER

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8. PERFORMING ORGANIZATION REPORT NUMBER

9. SPONSORING / MONITORING AGENCY NAME(S) AND ADDRESS(ES)

U.S. Army Medical Research and Development Command
Fort Detrick, Maryland 21702-5012

10. SPONSOR/MONITOR'S ACRONYM(S)

11. SPONSOR/MONITOR'S REPORT NUMBER(S)

12. DISTRIBUTION / AVAILABILITY STATEMENT

Approved for Public Release; Distribution Unlimited

13. SUPPLEMENTARY NOTES

14. ABSTRACT: Metabolic reprogramming is one of the key characteristics of cancer and tumor microenvironment for fueling the rapid and self-sufficient growth of cancer cells. L-3-phosphoserine phosphatase (PSPH) is one of the five rate-limiting enzymes in the biosynthesis of serine from glucose, which generates nucleotides to support cell proliferation. Here, we aim to understand the role of PSPH expression and its regulation in prostate cancer (PCa) and its relation to PCa disparity. We discovered that MDAPCa2b cells, which were derived from African American (AA) PCa, and AA carcinoma-associated fibroblasts (CAFs)express much higher levels of PSPH protein compared to benign associated fibroblasts, normal prostate epithelial cells, and PCa cell lines from European American PCa. Knock-down of PSPH expression in MDA-PCa2b and LNCaP cells significantly altered the expression of genes related to metabolisms (sex steroid hormone and cholesterol biosynthesis) and immunity (antigen process and presentation and interferon-related genes), which is consistent with our previous analysis of publicly available PCa RNA seq databases. The results suggest that targeting PSPH in prostate cancer can regulate tumor metabolism and immunity.

15. SUBJECT TERMS

NONE LISTED

16. SECURITY CLASSIFICATION OF:			**17. LIMITATION OF ABSTRACT**	**18. NUMBER OF PAGES**	**19a. NAME OF RESPONSIBLE PERSON** USAMRDC
a. REPORT	**b. ABSTRACT**	**c. THIS PAGE**			**19b. TELEPHONE NUMBER** *(include area code)*
U	U	U	UU	9	

Standard Form 298 (Rev. 8-98)
Prescribed by ANSI Std. Z39.18

TABLE OF CONTENTS

Page No.

1. Introduction
2. Keywords
3. Accomplishments
4. Impact
5. Changes/Problems
6. Products
7. Participants & Other Collaborating Organizations
8. Special Reporting Requirements
9. Appendices
10. References

TABLE OF CONTENTS

Page 1

1. Introduction

Metabolic changes have been recognized as a hallmark of cancer, which include dysregulation of serine metabolism. In general, extracellular serine alone that enters cells via amino acid transporters is sufficient to meet the needs of tumor cell proliferation. However, some tumor cells can increase de novo serine synthesis through glycolysis intermediates even when sufficient extracellular serine is supplied. Actively synthesized serine is utilized for promoting nucleotide synthesis, redox homeostasis, amino acid transport, and folic acid metabolism, thereby enhancing tumor cell proliferation.

Kinseth et al. examined the differences in gene expression between AA and EA PCa by matching for age and pathological stage or Gleason scores as well as tumor-cell content and stroma-cell content. Striking differences in gene expression were observed in the stroma of AA patients relative to EA: 1016 genes with significant differences between the expression of EA and AA patients were observed. The vast majority (82%) of significant differences were downregulated. In this study, components of extracellular matrix (ECM), mediators of cellular immunity, mediators of the epithelial-to-mesenchymal transition (EMT), and L-3-phosphoserine phosphatase (PSPH) were the top differentially expressed genes in the stroma of AA compared to EA men. PSPH was previously described as a gene expression biomarker in tumor tissues for identifying prostate, breast, endometrial, and colorectal cancers specifically in AAs. However, our study was the first to show overexpression of the PSPH gene in the cancer stroma of AA patients compared to EAs.

Therefore, in this study, we aim to understand the biological functions of PSPH and its regulatory mechanisms in the stroma of AA *versus* EA PCa using primary cultures of carcinoma-associated fibroblasts (CAFs) and tissue recombination model in the sub-renal capsule of SCID mice.

2. Keywords

PSPH, prostate cancer disparity, TGF-beta, carcinoma-associated fibroblasts, distant carcinoma-associated fibroblasts, normal associated fibroblasts.

3. Accomplishments

Aim 1: determine the differential ability of AA CAFs vs. EA CAFs to transform BPH-1 cells and the normal mouse prostate into tumorigenic products

Major Task 1: Establishment of primary CAFs and distant CAFs culture from prostatectomy specimens of both AA and EA prostate cancer patients.

In order to obtain more appropriate controls, an additional **3** normal-associated fibroblasts (NAFs, 2 EAs and 1 AAs) were also developed from the tumor-free prostate of cystoprostatectomy specimens of EA patients during the past year.

In addition, **6 AA CAFs (out of 9 AA patients)** and **20 EA CAFs (out of 24 EA patients) and their matched dCAFs** were developed during the past year. All the CAFs were validated for the origin of CAFS derived from the near stroma of prostate cancer through pathological examination.

RNA sequencing of gene expression profiles of these AA and EA CAFs lines, as well as NAFs lines are in progress.

Major Task 2: Sub-Renal capsule recombination xenograft assay.

We have continued our effects on evaluating the tumor initiation and growth of xenografts of nontumorigenic BPH-1 cells using two different lines of patient-derived AA CAFs and two lines of EA CAFs. Each of the four lineages was implanted under the kidney capsule of 5 adult male severe combined immunodeficiency (SCID) mice each. The initiation and growth of the xenografts was followed by in vivo magnetic resonance imaging (MRI) imaging at the UCI OncoImaging Core using a T9.4 Brunker small animal. More pairs of CAF/BPH-1 recombination is also in progress.

Major Task 3: Prostate orthotopic xenograft experiment

Pairs of AA and EA CAFs have been injected into the prostate of the NOD/SCID mice. Experiments are in progress to monitor tumor growth in the prostate.

Specific Aim 2: determine the impact of the enzymatic activity or expression of PSPH and high serine production in in vivo tumor growth.

Major Task 4: Whether PSPH expression and activity affect the CAFs mediated tumorigenic transformation or tumor growth?

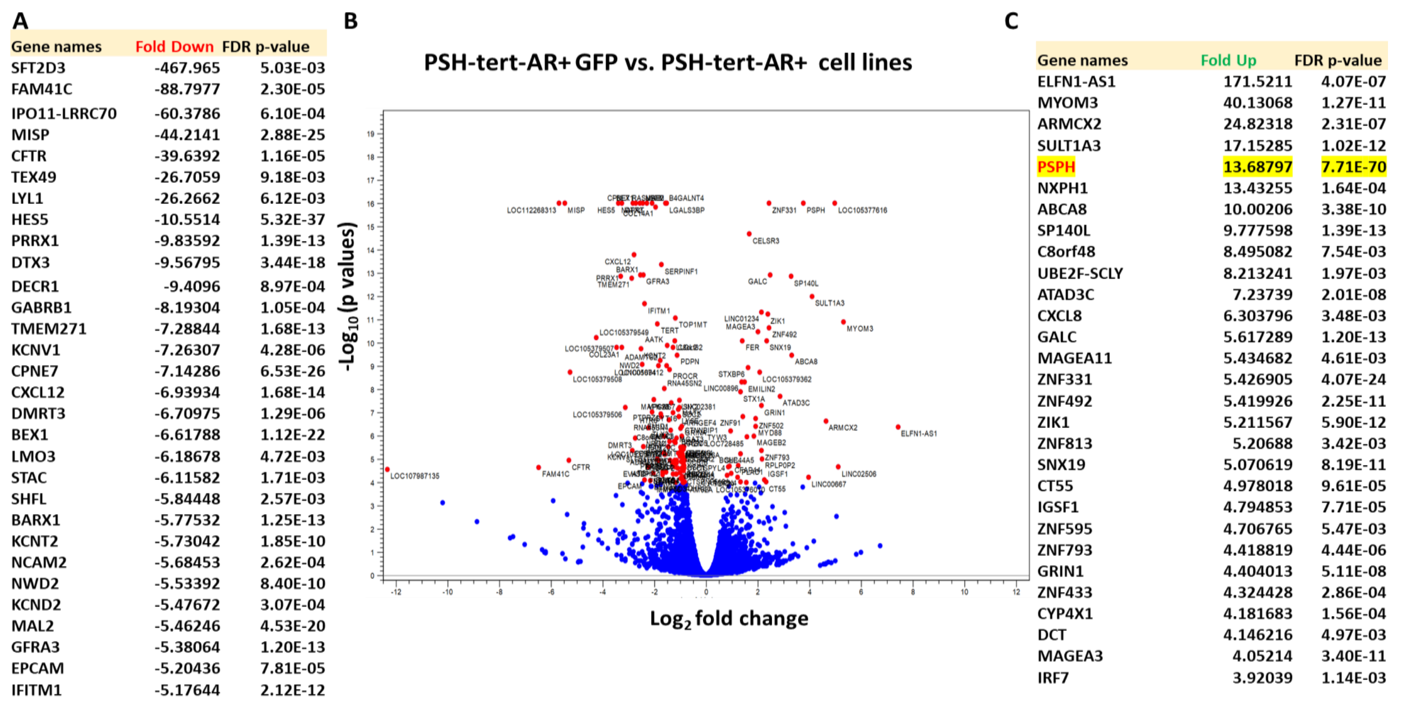
CAFs with stable overexpression of PSPH and stable suppression of PSPH, and MDA-PCa 2b with stable suppression have been made and further characterization for growth and activities is in progress. After characterization, tissue recombination experiments will be performed.

Specific Aim 3: determine the combined effects of docetaxel and TGF- beta inhibitor on the growth of MDA-PCa 2b/CAFs recombination

Major Task 5: Evaluation of in vivo anticancer efficacy of docetaxel in combination with TGF-beta inhibitor.
- Establish orthotopic xenograft models of AA CAFs and MDA-PCa2 cells.
- Randomizing into different treatment groups.
- MRI monitoring tumor growth
- Histology analysis of xenograft tumors.
- Evaluating the effect of the TGF beta inhibitor and /or docetaxel on collagen disposition and expression of biomarkers
- Statistical analysis

Experimental results.

1. The identification of differentially expressed genes (DEGs) in PSPH overexpressing PSH-tert-AR+ vs. control PSH-tert-AR+ cells. We have obtained the immortalized fibroblast cells with androgen receptor (AR) expression (PSH-tert-AR+ cells) from benign prostate tissues from Dr. Peng Lee (New York University). Then, we stably overexpressed PSPH in PSH-tert-AR+ cells. In the previous annual report, we have shown that PSPH overexpression or amplification in primary prostate tumor tissues is associated with alterations in metabolism and immunity. Here, we have performed a systematic transcriptome analysis of immortalized prostate stroma cells with PSPH overexpression. The results identified DEGs related to **1)** metabolisms, including ABCA8 and ATAD3C for cholesterol metabolism, GALC, Dct, IGSF1, and SNX19; **2)** immunity, including some interferon-stimulated gene (ISG), such as SHFL, IFITM1, IRF7, etc. antigen-related genes (SP140L MAGEA11, CT55, MAL2, and MAGE-A3), CXCL12, CXCL8, etc. **3)** Ion channels related genes, such as CFTR, KCND2 STAC3, KCNE1, GRIN1 etc. **4)** NOTCH signaling, such as DTX3, HES1 and HES5; **5)** Zinc Finger proteins, including ZNF331, ZNF595, ZIK1 etc. There are some significant similarities in DEGs between PSPH overexpressing cell line and prostate cancer tumor samples. **(Figure 1).**



Gene names	Fold Down	FDR p-value
SFT2D3	-467.965	5.03E-03
FAM41C	-88.7977	2.30E-05
IPO11-LRRC70	-60.3786	6.10E-04
MISP	-44.2141	2.88E-25
CFTR	-39.6392	1.16E-05
TEX49	-26.7059	9.18E-03
LYL1	-26.2662	6.12E-03
HES5	-10.5514	5.32E-37
PRRX1	-9.83592	1.39E-13
DTX3	-9.56795	3.44E-18
DECR1	-9.4096	8.97E-04
GABRB1	-8.19304	1.05E-04
TMEM271	-7.28844	1.68E-13
KCNV1	-7.26307	4.28E-06
CPNE7	-7.14286	6.53E-26
CXCL12	-6.93934	1.68E-14
DMRT3	-6.70975	1.29E-06
BEX1	-6.61788	1.12E-22
LMO3	-6.18678	4.72E-03
STAC	-6.11582	1.71E-03
SHFL	-5.84448	2.57E-03
BARX1	-5.77532	1.25E-13
KCNT2	-5.73042	1.85E-10
NCAM2	-5.68453	2.62E-04
NWD2	-5.53392	8.40E-10
KCND2	-5.47672	3.07E-04
MAL2	-5.46246	4.53E-20
GFRA3	-5.38064	1.20E-13
EPCAM	-5.20436	7.81E-05
IFITM1	-5.17644	2.12E-12

Gene names	Fold Up	FDR p-value
ELFN1-AS1	171.5211	4.07E-07
MYOM3	40.13068	1.27E-11
ARMCX2	24.82318	2.31E-07
SULT1A3	17.15285	1.02E-12
PSPH	13.68797	7.71E-70
NXPH1	13.43255	1.64E-04
ABCA8	10.00206	3.38E-10
SP140L	9.777598	1.39E-13
C8orf48	8.495082	7.54E-03
UBE2F-SCLY	8.213241	1.97E-03
ATAD3C	7.23739	2.01E-08
CXCL8	6.303796	3.48E-03
GALC	5.617289	1.20E-13
MAGEA11	5.434682	4.61E-03
ZNF331	5.426905	4.07E-24
ZNF492	5.419926	2.25E-11
ZIK1	5.211567	5.90E-12
ZNF813	5.20688	3.42E-03
SNX19	5.070619	8.19E-11
CT55	4.978018	9.61E-05
IGSF1	4.794853	7.71E-05
ZNF595	4.706765	5.47E-03
ZNF793	4.418819	4.44E-06
GRIN1	4.404013	5.11E-08
ZNF433	4.324428	2.86E-04
CYP4X1	4.181683	1.56E-04
DCT	4.146216	4.97E-03
MAGEA3	4.05214	3.40E-11
IRF7	3.92039	1.14E-03

Figure 1. RNA-seq analysis of PSPH overexpressing prostate stromal cells. A) The top list of differentially down-regulated genes by PSPH overexpression in immortalized benign prostate fibroblasts (n = 4). B) Volcano plot of significant differentially expressed genes. C) The top list of differentially up-regulated genes by PSPH overexpression in immortalized benign prostate fibroblasts (n = 4).

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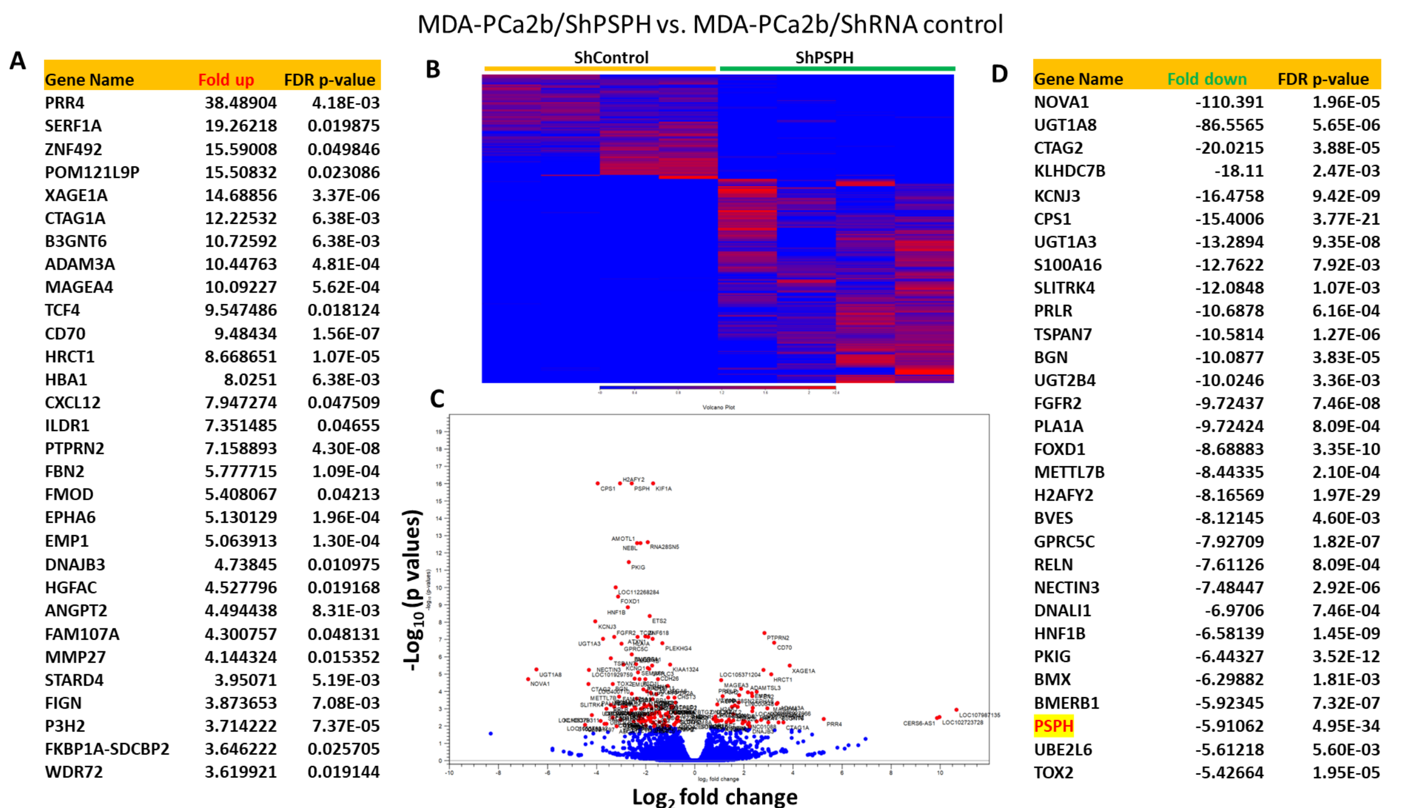
2. DEGs are associated with PSPH suppression in prostate cancer cell lines derived from AA (MDA-PCa 2b) and EA (LNCaP) and patients. We next examined how the suppression of PSPH expression in prostate cancer

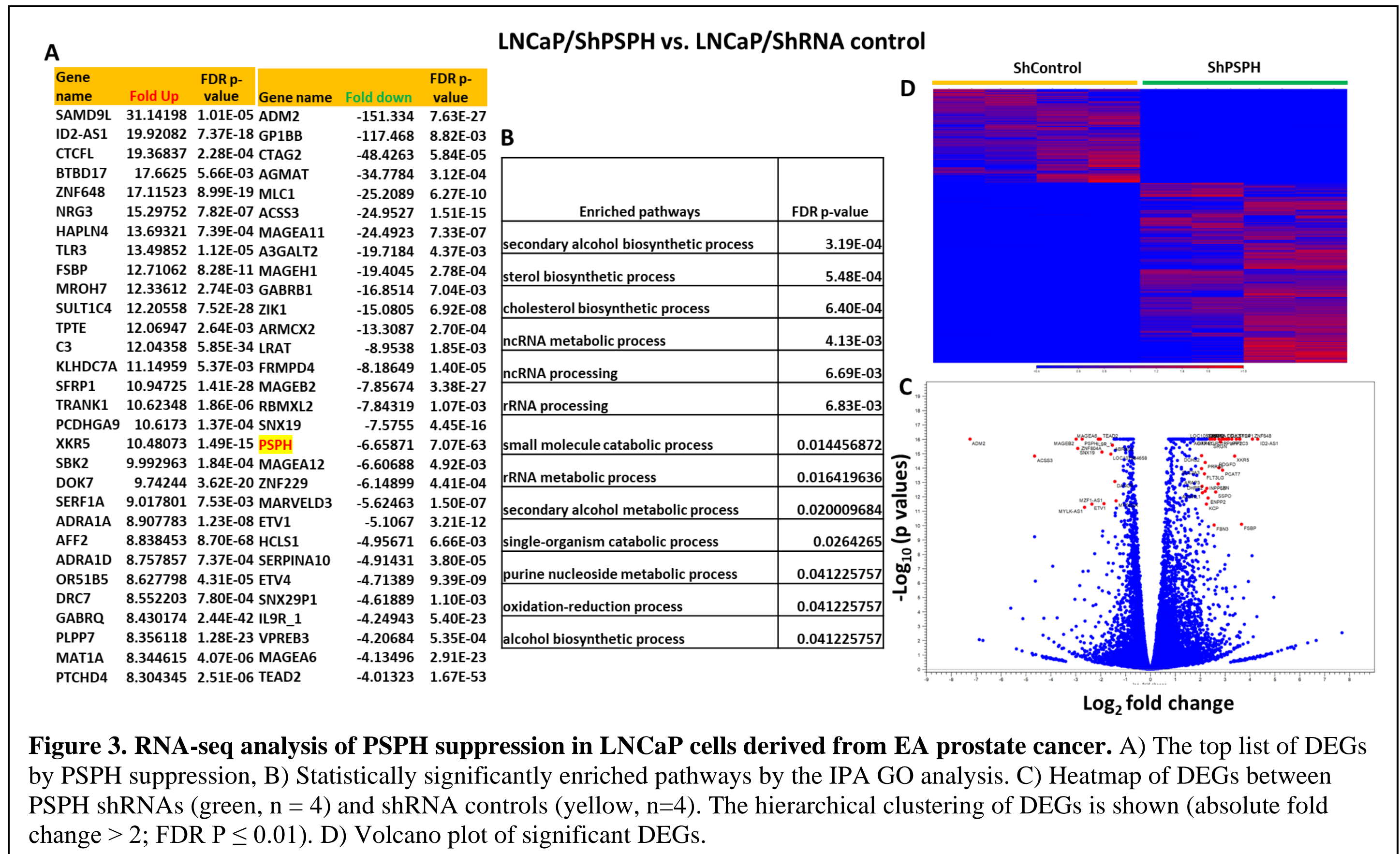


Gene Name	Fold up	FDR p-value
PRR4	38.48904	4.18E-03
SERF1A	19.26218	0.019875
ZNF492	15.59008	0.049846
POM121L9P	15.50832	0.023086
XAGE1A	14.68856	3.37E-06
CTAG1A	12.22532	6.38E-03
B3GNT6	10.72592	6.38E-03
ADAM3A	10.44763	4.81E-04
MAGEA4	10.09227	5.62E-04
TCF4	9.547486	0.018124
CD70	9.48434	1.56E-07
HRCT1	8.668651	1.07E-05
HBA1	8.0251	6.38E-03
CXCL12	7.947274	0.047509
ILDR1	7.351485	0.04655
PTPRN2	7.158893	4.30E-08
FBN2	5.777715	1.09E-04
FMOD	5.408067	0.04213
EPHA6	5.130129	1.96E-04
EMP1	5.063913	1.30E-04
DNAJB3	4.73845	0.010975
HGFAC	4.527796	0.019168
ANGPT2	4.494438	8.31E-03
FAM107A	4.300757	0.048131
MMP27	4.144324	0.015352
STARD4	3.95071	5.19E-03
FIGN	3.873653	7.08E-03
P3H2	3.714222	7.37E-05
FKBP1A-SDCBP2	3.646222	0.025705
WDR72	3.619921	0.019144

Gene Name	Fold down	FDR p-value
NOVA1	-110.391	1.96E-05
UGT1A8	-86.5565	5.65E-06
CTAG2	-20.0215	3.88E-05
KLHDC7B	-18.11	2.47E-03
KCNJ3	-16.4758	9.42E-09
CPS1	-15.4006	3.77E-21
UGT1A3	-13.2894	9.35E-08
S100A16	-12.7622	7.92E-03
SLITRK4	-12.0848	1.07E-03
PRLR	-10.6878	6.16E-04
TSPAN7	-10.5814	1.27E-06
BGN	-10.0877	3.83E-05
UGT2B4	-10.0246	3.36E-03
FGFR2	-9.72437	7.46E-08
PLA1A	-9.72424	8.09E-04
FOXD1	-8.68883	3.35E-10
METTL7B	-8.44335	2.10E-04
H2AFY2	-8.16569	1.97E-29
BVES	-8.12145	4.60E-03
GPRC5C	-7.92709	1.82E-07
RELN	-7.61126	8.09E-04
NECTIN3	-7.48447	2.92E-06
DNALI1	-6.9706	7.46E-04
HNF1B	-6.58139	1.45E-09
PKIG	-6.44327	3.52E-12
BMX	-6.29882	1.81E-03
BMERB1	-5.92345	7.32E-07
PSPH	-5.91062	4.95E-34
UBE2L6	-5.61218	5.60E-03
TOX2	-5.42664	1.95E-05

Figure 2. RNA-seq analysis of PSPH suppression in MDA-PCa 2b cells derived from AA prostate cancer. A) The top list of differentially up-regulated by PSPH suppression, B) Heatmap of DEGs between PSPH shRNAs (green, n = 4) and shRNA controls (yellow, n=4). The hierarchical clustering of DEGs is shown (absolute fold change > 2; FDR P ≤ 0.01). C) Volcano plot of significant DEGs. C) The top list of differentially down-regulated genes by PSPH suppression.

cell lines regulate global gene expression profiling. **Figure 2** shows the statistically significant DEGs, including 1) Metabolism-related genes, such as *PLA1A, GPRC5C, DNAJB3, HGFAC, HBA1, STARD4, UGT2B4 UGT1A3 UGT1A8, B3GNT6, CPS1, PRLR, BMX,* etc. 2) Immunity related genes, such as tumor antigens (*XAGE1A X, CTAG1A, MAGEA4, CTAG2*), novel immune targets (*CD70, TOX2 and PTPN22*), chemokines (*CXCL12*). 3) Extracellular matrix remodeling, such as *FMOD, ADAM3A, FBN2, MMP27, P3H2, BGN, RELN, etc. 4)* Ion channels and regulation-regulated genes, such *as* KCNJ3, S100A16, etc.



Gene name	Fold Up	FDR p-value	Gene name	Fold down	FDR p-value
SAMD9L	31.14198	1.01E-05	ADM2	-151.334	7.63E-27
ID2-AS1	19.92082	7.37E-18	GP1BB	-117.468	8.82E-03
CTCFL	19.36837	2.28E-04	CTAG2	-48.4263	5.84E-05
BTBD17	17.6625	5.66E-03	AGMAT	-34.7784	3.12E-04
ZNF648	17.11523	8.99E-19	MLC1	-25.2089	6.27E-10
NRG3	15.29752	7.82E-07	ACSS3	-24.9527	1.51E-15
HAPLN4	13.69321	7.39E-04	MAGEA11	-24.4923	7.33E-07
TLR3	13.49852	1.12E-05	A3GALT2	-19.7184	4.37E-03
FSBP	12.71062	8.28E-11	MAGEH1	-19.4045	2.78E-04
MROH7	12.33612	2.74E-03	GABRB1	-16.8514	7.04E-03
SULT1C4	12.20558	7.52E-28	ZIK1	-15.0805	6.92E-08
TPTE	12.06947	2.64E-03	ARMCX2	-13.3087	2.70E-04
C3	12.04358	5.85E-34	LRAT	-8.9538	1.85E-03
KLHDC7A	11.14959	5.37E-03	FRMPD4	-8.18649	1.40E-05
SFRP1	10.94725	1.41E-28	MAGEB2	-7.85674	3.38E-27
TRANK1	10.62348	1.86E-06	RBMXL2	-7.84319	1.07E-03
PCDHGA9	10.6173	1.37E-04	SNX19	-7.5755	4.45E-16
XKR5	10.48073	1.49E-15	PSPH	-6.65871	7.07E-63
SBK2	9.992963	1.84E-04	MAGEA12	-6.60688	4.92E-03
DOK7	9.74244	3.62E-20	ZNF229	-6.14899	4.41E-04
SERF1A	9.017801	7.53E-03	MARVELD3	-5.62463	1.50E-07
ADRA1A	8.907783	1.23E-08	ETV1	-5.1067	3.21E-12
AFF2	8.838453	8.70E-68	HCLS1	-4.95671	6.66E-03
ADRA1D	8.757857	7.37E-04	SERPINA10	-4.91431	3.80E-05
OR51B5	8.627798	4.31E-05	ETV4	-4.71389	9.39E-09
DRC7	8.552203	7.80E-04	SNX29P1	-4.61889	1.10E-03
GABRQ	8.430174	2.44E-42	IL9R_1	-4.24943	5.40E-23
PLPP7	8.356118	1.28E-23	VPREB3	-4.20684	5.35E-04
MAT1A	8.344615	4.07E-06	MAGEA6	-4.13496	2.91E-23
PTCHD4	8.304345	2.51E-06	TEAD2	-4.01323	1.67E-53

Enriched pathways	FDR p-value
secondary alcohol biosynthetic process	3.19E-04
sterol biosynthetic process	5.48E-04
cholesterol biosynthetic process	6.40E-04
ncRNA metabolic process	4.13E-03
ncRNA processing	6.69E-03
rRNA processing	6.83E-03
small molecule catabolic process	0.014456872
rRNA metabolic process	0.016419636
secondary alcohol metabolic process	0.020009684
single-organism catabolic process	0.0264265
purine nucleoside metabolic process	0.041225757
oxidation-reduction process	0.041225757
alcohol biosynthetic process	0.041225757

Figure 3. RNA-seq analysis of PSPH suppression in LNCaP cells derived from EA prostate cancer. A) The top list of DEGs by PSPH suppression, B) Statistically significantly enriched pathways by the IPA GO analysis. C) Heatmap of DEGs between PSPH shRNAs (green, n = 4) and shRNA controls (yellow, n=4). The hierarchical clustering of DEGs is shown (absolute fold change > 2; FDR P ≤ 0.01). D) Volcano plot of significant DEGs.

The statistically significant DEGs of PSPH-suppressed LNCaP cells also contain 1) Metabolism-related genes, such as *MAT1A, SULT1C4, ADRA1A, ADRA1D, LRAT, A3GALT2, ACSS3, AGMAT, etc.* 2) Immunity related genes, such as tumor antigens (*CTAG2, MAGEA6, MAGEA12, MAGEA11*), and others VPREB3, SAMD9L, BTBD17, TLR3, C3, IL9R_1), 3) Cell migration and extracellular matrix remodelling, such as ID2-AS1, HAPLN4, FSBP, etc.(**Figure 3**).

The highly enriched pathways by PSPH shRNA knockdown in LNCaP cells are sex steroid biosynthesis, cholesterol biosynthesis, ncRNA processing, purine nucleoside metabolic process, and secondary alcohol biosynthetic process (**Figure 3B**). These results are consistent with our previous analyses with the data from RNA seq analysis of human prostate cancer specimens.

3. Reduced mRNA expression of numerous Interferon stimulated genes (ISGs) in AA CAFs vs EA CAFs.Our transcriptome analysis of 4 pairs AA CAFs and EA CAFs identified 1308 genes that are statistically significantly differentially expressed (corrected p < 0.05 and 1.5<FC ≥ 1.5) in AA CAFs compared to EA CAFs. Of these differentially expressed genes 154 belong to the interferon-stimulated genes (ISGs) (**Figure 4**) as identified in the INTERFEROME Database of ~1800 identified ISGs. Of the 154 differentially expressed ISG mRNAs, 34 (22%) were upregulated in AA CAFs compared to EA CAFs (**Figure 4A**) and 120 (78%) were down-regulated in AA compared to EA CAFs. Of these 120 genes, 24 were classified in the database as Type 1 IFN stimulated genes, 17 by Type- 2, and 68 stimulated by both Type- 1 and Type 2 according to the INTERFEROME database. The 24 Type-1 ISGs downregulated in AA CAFs compared to EA CAFs were associated with gene ontology functions such as the processing and presentation of endogenous antigens, cell adhesion, activin inhibitor as well as activation of MAPK and bone morphogenic protein (BMP) signaling. The 17 Type-2 regulated ISGs that were down-regulated in AA vs EA CAFs are associated with gene ontology functions such as ATP binding, autophagy, and vacuole formation, apoptosis, B cell proliferation, and cell adhesion. The 68

Type-1 ISGs and Type-2 ISGs that were down-regulated in AA vs EA CAFs are associated with gene ontology functions such as cell adhesion formation, acute phase response, JNK activity, MAPK activity, and caspase activity (Figure S2). Thus, there appears to be a marked decrease in the expression of interferon-related regulation in AA CAFs relative to EA CAFs, especially for the regulation of antigen presentation. There were 8 Type-1 regulated ISGs that were upregulated in AA vs EA CAFs. These were associated with gene ontology functions such as DNA binding transcriptional factor, extracellular matrix function, and G protein-coupled receptor signaling. There were 4 Type-2 ISGs that were upregulated in AA vs EA CAFs. Gene ontologies terms such as eukaryotic translation intimation factor including 4F complex and chemokine activity were associated with these genes. There were 20 genes controlled by both Type-1 and Type-2 ISGs that were up-regulated in AA vs EA CAFs. These genes were associated with actin binding, angiogenesis, anti-apoptosis, cell aging, calcium ion bindings. Six more genes were associated with Type-1 & and Type-3 and seven genes with all three IFN types.

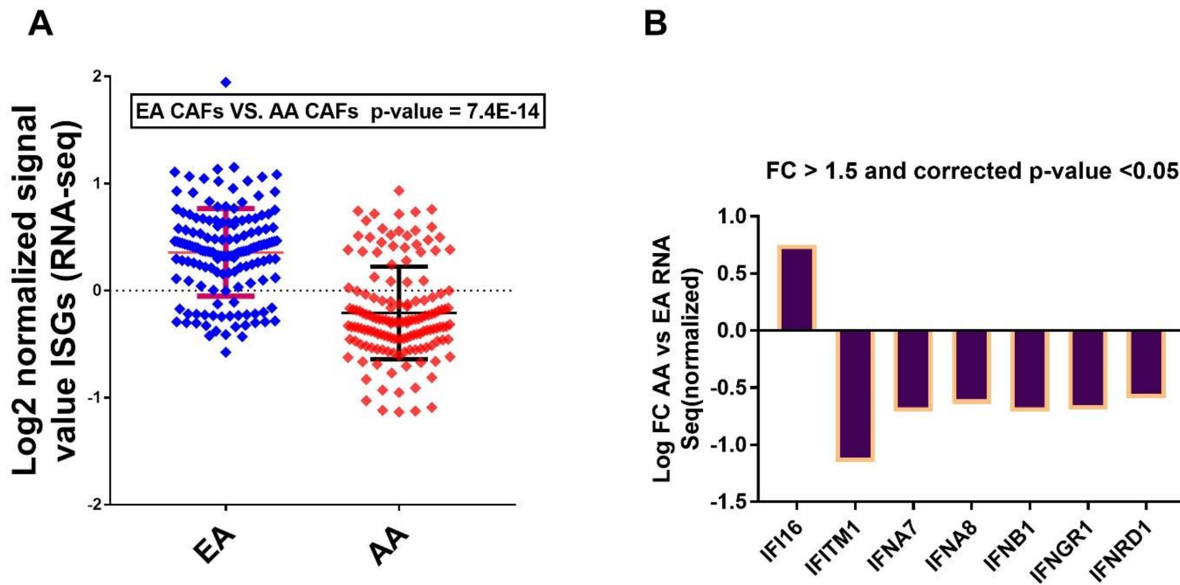


Figure 4: Expression of Interferon stimulated genes in AA and EA CAFs. (A) Expression of 154 statistically significantly differentially expressed (corrected $p < 0.05$ and FC >1.5) interferon stimulated genes (ISGs) in AA and EA PCa CAFs. (B) Members of the IFN gene family were also differentially expressed at the RNA levels

4. RNA expression differences between tumor-adjacent CAFs and distant CAFs. We also compared four pairs of CAFs derived from near tumor, to four dCAFs from a distant contralateral area where tumor was not detected. CAFs and dCAFs from the same individual were usually more similar to each other than to other patients (data not shown). Many genes were differentially expressed at the RNA levels and a few pathways were over-represented (p<0.05). Pathways that are different between CAFs and dCAFs in either direction should be of interest for identifying “field effects”; gene expression that is affected by proximity to tumor in a patient prostate. Pathways more highly expressed in CAFs than in dCAFs, including DNA replication, carbon metabolism, folate metabolism, synthesis and degradation of ketone bodies, meiotic recombinations, double-stranded break repair, vitamin 12 metabolism and steroid biosynthesis, and immunoregulatory interaction between lymphoid and non-lymphoid cells. The latter is of particular interest as a potential immunological field effect. Among the genes more highly expressed in CAFs than in dCAFs, were (IGF2, IFI27, VCAM1, and HSBP1) and among the genes expressed at highly lower levels in CAFs vs dCAFs were home box genes (HOXA9, HOXA11, HOXA10, HOXB1m HOXB13, HOXC4, HOXC5).

4. Impact

4.1. We have developed unique resources of CAFs and dCAFs from many different AA and EA PCa patients and NAFs from the tumor-free prostate of cystoprostatectomy specimens. The ethnic

identification was determined by large SNP analysis. These resources provide an important tool for dissecting the stroma regulatory features of PCa of different races.

4.2. We discovered that targeting PSPH by shRNAs can alter gene-expression of metabolism and immunity related genes in prostate cancer cells.

4.3. There are significant changes between AA and EA CAFs in expression of numerous Interferon stimulated genes

5. Changes/Problems

None.

6. Products

6.1. Patient-derived CAFs, distant CAFs and NAFs.
6.2. PSPH overexpress and suppression stable cell lines.

7. Participants & Other Collaborating Organizations

Name:	Xiaolin Zi (UCI)
Project Role:	PI
Researcher Identifier (e.g. ORCID ID):	n/a
Nearest person month worked:	3
Contribution to Project:	*Supervision, experimental design, and grant management, development of patient-derived CAFs, dCAFs and NAFs.*
Funding Support:	*n/a*

Name:	Michael Lilly (MUSC)
Project Role:	PI
Researcher Identifier (e.g. ORCID ID):	n/a
Nearest person month worked:	0.6
Contribution to Project:	*IRB maintenance, collecting prostatectomy specimens from AA patients*
Funding Support:	*n/a*

Name:	Liankun Song
Project Role:	Assistant Project Scientist
Researcher Identifier (e.g. ORCID ID):	n/a
Nearest person month	6

worked:	
Contribution to Project:	*Development and maintenance of patient derived CAFs, dCAFs and NAFs. RNA seq analysis*
Funding Support:	*n/a*

Name:	Huayu Wu
Project Role:	Post-doctoral fellow
Researcher Identifier (e.g. ORCID ID):	n/a
Nearest person month worked:	6
Contribution to Project:	*Sub-Renal capsule recombination xenograft assay and Prostate orthotopic xenograft experiment*
Funding Support:	*n/a*

Name:	Chandra, Kishore
Project Role:	Post-doctoral fellow
Researcher Identifier (e.g. ORCID ID):	n/a
Nearest person month worked:	6
Contribution to Project:	*The combined effects of docetaxel and TGF- beta inhibitor on the growth of MDA-PCa 2b/CAFs recombination*
Funding Support:	*n/a*

Name:	Vinh Le
Project Role:	Junior Specialist
Researcher Identifier (e.g. ORCID ID):	n/a
Nearest person month worked:	6
Contribution to Project:	*Sub-Renal capsule recombination xenograft assay and Prostate orthotopic xenograft experiment*
Funding Support:	*n/a*

8. Special Reporting Requirements: N/A

9. Appendices: None