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W81XWH-12-1-0160

TITLE: Investigating Genomic Mechanisms of Treatment Resistance in
Castration Resistant Prostate Cancer

PRINCIPAL INVESTIGATOR: Terence W. Friedlander, MD

CONTRACTING ORGANIZATION: University of California, San Francisco
San Francisco, California 94143

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14. ABSTRACT

Purpose and Scope: The purpose of this work is to better understand the mechanisms of resistance to androgen biosynthesis inhibitors in men with castration resistant prostate cancer, and to investigate clinical methods of overcoming resistance.

Key Accomplishments and Findings to date:

- CTCs collected in 41 men with abiraterone-naïve mCRPC at baseline, and in 12 of these men at the time of clinical resistance to abiraterone. Cells have been enumerated for CTCs, CTC clusters, CTCs expressing stem-like and mesenchymal markers. We have optimized methods for extracting DNA and performing array comparative genomic hybridization (aCGH). Currently we are in the process of analyzing the aCGH results in collaboration with the UCSF Biostatistics Core.
- We have observed that there is a wide spectrum of CTC diversity (epithelial-like, mesenchymal, stem-like) in men starting abiraterone. Correlations of this diversity with clinical outcome are underway.
- Phase II protocol for Dose-Increased Abiraterone Acetate in Men with mCRPC (PI: Friedlander) fully accrued at UCSF (n=21) and Oregon Health Sciences University (n=20).
- Phase I protocol of Abiraterone Acetate plus ARN-509 in men with mCRPC (PI: Friedlander) set to open at UCSF in mid-late 2014.
- Integration of both clinical trials with Stand Up 2 Cancer "West Coast Dream Team" castration-resistant prostate cancer biopsy protocol. Analysis of CTC-biopsy correlations underway.

15. SUBJECT TERMS

Prostate cancer, castration-resistant prostate cancer, abiraterone, androgens, circulating tumor cells, treatment resistance

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INTRODUCTION

Although androgen biosynthesis inhibitors (ABIs) including ketoconazole and abiraterone improve clinical outcomes and prolong survival in men with castration resistant prostate cancer (CRPC), none are curative, and all patients eventually develop resistance followed by disease progression and death. Resistance is hypothesized to result from either increased systemic or tumor androgen production, mutations in the androgen receptor (AR) signaling pathway leading to ligand-independent AR activity, or through AR-independent pathways. The work being carried out under this grant aims to better understand how this therapeutic resistance develops through genomic analysis (gene copy number and gene methylation status) of tumor biopsies and circulating tumor cells (CTCs) taken from men with CRPC. Further, the work here explores whether clinically targeting proposed mechanisms of resistance can improve outcomes in these patients.

BODY

Statement of Work Aim A: Determine whether resistance to androgen biosynthesis inhibitors (ABIs) is mediated by genomic upregulation of androgen synthesis or by autonomous AR function.

Work continues for this Aim as follows: A laboratory specific UCSF protocol (CC#125511) for the analysis of metastatic biopsies and circulating tumor cells (CTCs) was developed and approved by both UCSF and the DoD IRB and is undergoing renewal at present. Currently CTCs are currently being collected as part of a clinical trial of increased-dose abiraterone (CC12551) detailed below in Aim B of this summary.

At the same time that we are collecting CTCs and biopsies our group is spearheading the Stand Up 2 Cancer “West Coast Dream Team” study of metastatic CRPC biopsies to better understand treatment resistance, and the study of the metastatic tissues for this Aim has been integrated, as discussed in the 2013 Progress Report.

For CC125511 we have now collected blood from a total of 21 men starting abiraterone for CRPC. CTCs are detectable in >95% of these samples using the Vitatex VitaCaP assay, in which CTCs and white blood cells invade into a fluorescently labeled collagen adhesive matrix (CAM). Using immunocytochemistry we see that the CTCs express epithelial markers including PSMA, CK, and EpCAM; heterogeneity in this expression (i.e. differing combinations) has been observed. At the same time CTCs expressing a mesenchymal phenotype are detectable as well as those bearing markers of stemness including CD44. We have fully optimized flow cytometry, gating both for enumeration and for purification of different CTC subpopulations. We are able to increase the CTC purity from 1-10% up to >90% using these techniques, and can objectively see and count cells expressing epithelial markers, mesenchymal markers, and markers of stemness. We have optimized techniques for DNA amplification using a 10-cell Picoplex amplification protocol to yield sufficient CTC DNA for aCGH analysis. At the same time mCRPC biopsies have been collected under the aforementioned StandUp To Cancer West Coast Dream Team protocol and comprehensive integrated analysis is being performed on these samples, including aCGH of the biopsy tissue and matching CTCs. aCGH has been performed in >10 CTC samples for abiraterone naïve patients and more samples,

including those from patients with acquired abiraterone resistance, are pending. More comprehensive data analysis of aCGH results from CTCs and biopsies are planned once sufficient number of abiraterone resistant samples are obtained (anticipated by late 2014).

Statement of Work Aim B: Determine whether resistance to ABIs s can be overcome by increased inhibition of androgen synthesis.

As discussed above, a clinical protocol for increased-dose abiraterone has been written, approved by both the peer-review/scientific committees at UCSF and at Oregon Health Science University (OHSU) and their respective IRBs, and the clinical trial opened and began accruing patients at each site in March 2013. The protocol completed accrual of 41 planned patients in mid 2014. This clinical trial of dose-increased abiraterone is registered with clinicaltrials.gov with the number NCT01637402. Weekly review of compliance with study procedures, safety, and efficacy of the elevated dose continues. A total of 13 patients have had a response to abiraterone 100mg daily followed by PSA or objective progression, and have dose-increased to abiraterone 1000mg BID. To date the elevated dose appears safe and there have not been any serious safety concerns at this dose. While a number of patients have had a decline in PSA at the 1000mg BID dose, to date there have not been any PSA responses (defined as a >30% decline in PSA), and an analysis for early stopping for efficacy will be performed should the 14th patient at the elevated dose not have a PSA response. Please note that this efficacy and safety data is unpublished and proprietary information. Regardless of the efficacy outcome for the 1000mg BID dose, all patients on the 1000mg daily dose will continue to receive treatment and will be followed to collect hormone, pK, and CTC/biopsy data, which will allow for an analysis of the mechanisms of resistance to abiraterone.

Statement of Work Aim C: Determine whether resistance to ABIs can be overcome by AR-targeted therapy.

Accrual to the Phase I study of the combination of abiraterone acetate plus ARN-509 (a novel AR antagonist) described in the previous Update was placed on hold for a large portion of late 2013 and early 2014 due to a number of issues including manufacturing problems in the production of ARN-509, as well as the acquisition of Aragon by Janssen Pharmaceuticals. A revised Phase I study is therefore set to open here at UCSF in mid-late 2014. UCSF patients enrolling on this study will undergo CTC collection and will be referred for biopsy under the Stand Up to Cancer biopsy protocol active at UCSF. The Phase II protocol for the evaluation of ARN-509 given at the time of resistance to abiraterone has been authored and reviewed at the UCSF Genitourinary Oncology Site Committee, and is under consideration by Janssen, pending the outcome of the Phase I study.

KEY RESEARCH ACCOMPLISHMENTS

- CTCs collected in 21 men with abiraterone-naïve mCRPC. Significant heterogeneity has been seen in these samples with the detection of CTCs bearing epithelial,

mesenchymal, and stem like markers. CTCs are detectable >95% of men using the Vitatex VitaCaP assay. Flow cytometric sorting yields a highly pure cell population, and amplification yields DNA appropriate for genomic analysis.

- Phase II protocol for Dose-Increased Abiraterone Acetate in Men with mCRPC (CC12551, PI: Friedlander) written, IRB approved, and fully accrued at UCSF and Oregon Health Sciences University (as part of the DoD Prostate Cancer Clinical Trials Consortium).
- Revised phase I protocol of Abiraterone Acetate plus ARN-509 in men with CRPC (PI: Friedlander) set to open at UCSF in mid-late 2014
- Phase II protocol of Abiraterone Acetate plus ARN-509 in men with mCRPC (PI: Friedlander) completed, approved by UCSF site-review committee, and under consideration with the Sponsor.
- Laboratory protocol for copy number analysis of CTCs and metastatic biopsies (CC125511, PI: Friedlander) completed and undergoing reapproval evaluation by the IRBs at UCSF and the DoD.
- Integration of both clinical trials with recently awarded Stand Up 2 Cancer “West Coast Dream Team” CRPC biopsy protocol, allowing for even more comprehensive molecular and genomic analysis of mechanisms of abiraterone/ABI resistance.

REPORTABLE OUTCOMES

Two clinical protocols and a laboratory protocol for the work have been developed for this grant. Since the last Update CTC data from CC125511 has been presented at the 2013 International Symposium on Minimal Residual Cancer (oral abstract), at the 2014 ASCO annual meeting (poster discussion). A manuscript summarizing our lab experience with CTCs collected previously from 23 men with prostate cancer and analyzed on the Vitatex platform was recently published in the International Journal of Cancer¹. Based on the experience and work from this grant I have similarly authored and published a review paper discussing the potential for CTCs in prostate cancer² as well as an editorial about CTCs in the Journal of Clinical Oncology.³

CONCLUSION

Continued progress has been made in terms of achieving goals set forth in the statement of work for this project, with full accrual of the lab protocol and the dose-escalated study proposed in the grant. Integration and analysis of results for CTCs and biopsies is underway in collaboration with the Stand Up to Cancer mCRPC biopsy study.

REFERENCES

1. **Friedlander TW**, Premasekharan G, Paris PL. (2014). Detection and Characterization of Invasive Circulating Tumor Cells (iCTCs) Derived from Men with Metastatic Castration Resistant Prostate Cancer (mCRPC). *Int J Cancer*;134(10):2284-93.
2. **Friedlander TW**, Premasekharan G, Paris PL. (2014). Looking Back, to the Future of Circulating Tumor Cells”. *Pharmacol Ther*;142(3):271-280.
3. **Friedlander TW**, Fong L. (2014). The End of the Beginning: Circulating Tumor Cells as Prognostic Biomarkers in Castration Resistant Prostate Cancer. *J Clin Oncol*;32(11):1104-6.

APPENDIX/SUPPORTING DATA

1. UCSF Cancer Center (clinical) protocol 12551: A Phase II Study of Increased-Dose Abiraterone Acetate in Patients with Castration Resistant Prostate Cancer (CRPC). Notice of IRB re-approval.

2. Curriculum Vitae



**Human Research Protection Program
Committee on Human Research**

Notification of Expedited Review Approval

Principal Investigator
Terence W Friedlander

Co-Principal Investigator

Type of Submission: Continuing Review Submission Form
Study Title: CC#125511: Determination of Gene Copy Changes associated with Resistance to Androgen Biosynthesis Inhibitors in Men with Metastatic Castration Resistant Prostate Cancer

IRB #: 12-08760

Reference #: 088741

Committee of Record: Mount Zion Panel

Study Risk Assignment: Minimal

Approval Date: 06/23/2014

Expiration Date: 06/22/2015

Regulatory Determinations Pertaining to this Approval:

- The requirement for individual Research HIPAA Authorization is waived for all subjects. The use or disclosure of the requested information does not adversely affect the rights and welfare of the individuals and involves no more than a minimal risk to their privacy based on, at least, the presence of the following elements: (1) an adequate plan to protect the identifiers from improper use and disclosure; (2) an adequate plan to destroy the identifiers at the earliest opportunity consistent with conduct of the research, unless there is a health or research justification for retaining the identifiers or if such retention is otherwise required by law; (3) adequate written assurances that the requested information will not be reused or disclosed to any other person or entity, except as required by law, for authorized oversight of the research study, or for other research for which the use or disclosure of the requested information would be permitted by the Privacy Rule; (4) the research could not practicably be conducted without the waiver; and (5) the research could not practicably be conducted without access to and use of the requested information.

All changes to a study must receive CHR approval before they are implemented. Follow the [modification request](#) instructions. The only exception to the requirement for prior CHR review and approval is when the changes are necessary to eliminate apparent immediate hazards to the subject (45 CFR 46.103.b.4, 21 CFR 56.108.a). In such cases, report the actions taken by following these [instructions](#).

Expiration Notice: The iRIS system will generate an email notification eight weeks prior to the expiration of this study's approval. However, it is your responsibility to ensure that an application for [continuing review](#) approval has been submitted by the required time. In addition, you are required to submit a [study closeout report](#) at the completion of the project.

Approved Documents: To obtain a list of documents that were approved with this submission, follow these steps: Go to My Studies and open the study – Click on Submissions History – Go to Completed Submissions – Locate this submission and click on the Details button to view a list of submitted documents and their outcomes.

For a list of all currently approved documents, follow these steps: Go to My Studies and open the study – Click on Informed Consent to obtain a list of approved consent documents and Other Study Documents for a list of other approved documents.

San Francisco Veterans Affairs Medical Center (SFVAMC): If the SFVAMC is engaged in this research, you must secure approval of the VA Research & Development Committee in addition to CHR approval and follow all applicable VA and other federal requirements. The CHR [website](#) has more information.

BIOGRAPHICAL SKETCH

NAME Friedlander, Terence Ware	POSITION TITLE Assistant Clinical Professor		
eRA COMMONS USER NAME (credential, e.g., agency login)			

EDUCATION/TRAINING <i>(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable.)</i>			
INSTITUTION AND LOCATION	DEGREE <i>(if applicable)</i>	MM/YY	FIELD OF STUDY
Brown University, RI	BA	05/99	Biology
NYU Medical Center, NY	MD	05/03	Medicine
University of California, San Francisco, CA	-	06/03-06	Medicine Residency
Utrecht University, Netherlands	MA	2007	Medical Ethics
University of California, San Francisco, CA	-	2007-2010	Oncology Fellowship

A. Personal Statement

My research is focused on understanding the basic biology of bladder and prostate cancers and in developing novel therapeutic ways to treat disease. During my Oncology Fellowship at UCSF I focused on characterizing genomic changes in metastatic prostate cancer tumors and in matching circulating tumor cells (CTCs), as a means to identify biomarkers of response or resistance. This led to my interest in further studying CTCs in order to understand the molecular and genetic changes that lead to treatment resistance, and in using these to identify novel pathways for drug development. Since joining the faculty at UCSF, I have focused on prostate and bladder cancers and have designed studies and served as the Principal Investigator for multiple clinical trials in GU Oncology Group. I have become intimately familiar with the challenges faced by patients with prostate cancer and bladder cancer, and recognize the need for less costly and more efficient ways to obtain metastatic tissue (i.e. CTCs) for biologic characterization, as well as the pressing need for novel therapeutics that have less side effects and greater efficacy than our current regimens. My long-term goal is to both discover mechanisms which promote resistance to therapy in GU malignancies and, using this knowledge, devise novel hormonal, immunologic, and chemotherapeutic strategies to treat these patients.

B. Positions and Honors

Positions and Employment

2003-2006	Medical Residency, Internal Medicine, UCSF
2006-2007	Fulbright Scholar in Medical Ethics, University of Utrecht, Netherlands
2007-2010	Fellowship in Hematology and Oncology, UCSF
2009-2010	Chief Fellow, Hematology and Oncology, UCSF
2010-2011	Clinical Fellowship in Urologic Oncology, UCSF
2011-current	Assistant Clinical Professor, Division of Hematology and Oncology, UCSF

Honors

2000	Herman Goldman Scholarship, NYU Medical School
2003	Spiegel Award for Academic Excellence, NYU Medical School
2003	Alpha Omega Alpha, National Medical Honors Society
2003	Medical Degree with Honors, NYU Medical School
2006	Fulbright Scholarship in Medical Ethics, Netherlands-America Foundation
2010	ASCO Young Investigator Award, American Society of Clinical Oncology

2011	ASCO Merit Award, American Society of Clinical Oncology
2012	Physician Research Training Award, United States Department of Defense
2012	Young Investigator Award, Prostate Cancer Foundation
2012	Young Investigator Travel Award, Advances in Circulating Tumor Cells Symposium
2013	Poster award, UCSF Annual Prostate Cancer Retreat

Other Experience and Professional Memberships

2007-current	American Society of Clinical Oncology, Associate Member
2008	Cancer Education Consortium Pharmacology of Anticancer Agents Workshop Participant, Landsdowne, VA
2009	Stanford University 11th Annual Multidisciplinary Management of Cancers at Silverado, Genitourinary Tumor Board discussant
2009	ASCO/AACR Methods in Clinical Cancer Research Workshop Participant, Vail, CO
2009	Cancer and Lymphoma Group B (CALGB) Early Career Investigators Meeting, invited attendee
2010-current	San Francisco General Hospital CARE (Cancer Awareness Resources and Education), invited speaker
2010-current	American Association of Cancer Researchers, Associate Member
2010	ASCO Genitourinary Symposium, San Francisco, CA (poster)
2010	ASCO Annual Meeting, Chicago, IL (poster)
2011	ASCO Genitourinary Symposium, Orlando, FL (oral abstract)
2011	Doris Duke Workshop for Clinical Investigators on the Genetics of Complex Disorders Participant, Broad Institute, Cambridge, MA
2012	ASCO Annual Meeting, Chicago, IL (poster discussant)
2012	Advances in Circulating Tumor Cells Symposium, Athens, Greece (oral abstract)
2013	ASCO Genitourinary Symposium, Orlando, FL (poster)
2013	ASCO Annual Meeting, Chicago, IL (poster)
2013	9 th Annual International Symposium on Minimal Residual Cancer, Paris, France (oral abstract)
2014	ASCO Genitourinary Symposium, San Francisco, CA (poster)
2014	ASCO Annual Meeting, Chicago, IL (poster)

C. Selected Peer-reviewed Publications

1. **Friedlander TW**, Ryan CJ. (2009). Novel hormonal approaches in prostate cancer. *Curr Oncol Rep*;11:227-234.
2. **Friedlander TW**, Ryan CJ. (2010) Editorial Comment on Adrenocorticotrophic hormone (ACTH) regulates androgen synthesis in men receiving androgen deprivation therapy for localized prostate cancer. *J Urol*;184(5):1976.
3. **Friedlander TW**, Weinberg VK, Small EJ, Sharib J, Harzstark AL, Lin AM, Fong L, Ryan CJ. (2012). Effect of the Somatostatin Analog Octreotide Acetate on Circulating Insulin-Like Growth Factor-1 and Related Peptides in Patients with Castration-Resistant Prostate Cancer (CRPC): Results of a Phase II Study. *Urol Oncol*;30(4):408-14.
4. **Friedlander TW**, Weinberg VK, Huang Y, Mi JT, Formaker CG, Small EJ, Harzstark AL, Lin AM, Fong L, Ryan CJ. (2012). A Phase II Study of Insulin-like Growth Factor Receptor Inhibition with Nordihydroguaiaretic Acid in Men with Non-Metastatic Hormone Sensitive Prostate Cancer. *Oncol. Rep*;27(1):3-9.
5. **Friedlander TW**, Roy R, Tomlins SA, Ngo VT, Kobayashi Y, Azameera A, Rubin MA, Pienta KJ, Chinnaiyan A, Ittmann MM, Ryan, CJ, Paris PL. (2012). Common Structural and Epigenetic and Changes in the Genome of Castration Resistant Prostate Cancer. *Cancer Res*;72(3):616-25.
6. **Friedlander TW**, Ryan CJ. (2012). Targeting the Androgen Receptor. *Urol Clin North Am*;39(4):453-64.

7. **Friedlander, TW.** (2013). Whole Exome Sequencing to Map the Genomic Evolution of Metastatic Prostate Cancer. *Hum Mutat* 34:1231-1241.
8. **Friedlander TW,** Premasekharan G, Paris PL. (2014). Detection and Characterization of Invasive Circulating Tumor Cells (iCTCs) Derived from Men with Metastatic Castration Resistant Prostate Cancer (mCRPC). *Int J Cancer*;134(10):2284-93.
9. **Friedlander TW,** Premasekharan G, Paris PL. (2014). Looking Back, to the Future of Circulating Tumor Cells". *Pharmacol Ther*;142(3):271-280.
10. **Friedlander TW,** Fong L. (2014). The End of the Beginning: Circulating Tumor Cells as Prognostic Biomarkers in Castration Resistant Prostate Cancer. *J Clin Oncol*;32(11):1104-6.

D. Research Support

Current Research Support

DoD PC110126 Friedlander (PI) 01/01/12-12/31/16
Investigating Genomic Mechanisms of Treatment Resistance in Castration Resistant Prostate Cancer. Career Development Award: the goal of this study is to identify the genetic changes associated with hormonal therapy resistance that occur in men with advanced prostate cancer.
Role: PI

PCF P0044252 Friedlander (PI) 1/01/12-12/31/14
Young Investigator Award: Investigating Genomic Mechanisms of Treatment Resistance in Castration Resistant Prostate Cancer. Young Investigator Award: the goal of this study is to identify the genetic changes associated with hormonal therapy resistance that occur in men with advanced prostate cancer.
Role: PI

IMCL CP20-0902 Friedlander (Site PI) 7/1/10 – 8/8/15
Imclone Systems Inc.
An Open-Label, Multicenter, Randomized Phase 2 Study Evaluating the Safety and Efficacy of Docetaxel in Combination with Ramucirumab (IMC-1121B) Drug Product or IMC-18F1 or Without Investigational Therapy as Second-line Therapy in Patients With Locally Advanced or Metastatic Transitional Cell Carcinoma of the Bladder, Urethra, Ureter, or Renal Pelvis Following Disease Progression on First-line Platinum-based Therapy The goal of this study is to determine the anti-tumor efficacy of Ramucirumab or IMC-18F1 plus docetaxel in patients with metastatic platinum-resistant bladder cancer.
Role: Site PI

VEG113387 (PROTECT) Friedlander (Site PI) 1/10/11 – 4/2/16
GlaxoSmithKline
A Study to Evaluate Pazopanib as an Adjuvant Treatment for Localized Renal Cell Carcinoma (RCC)
The goal of this study is to determine the safety and anti-tumor efficacy of adjuvant pazopanib for localized, resected primary kidney cancer.
Role: Site PI

212082PCR2005 Friedlander (Site PI) 3/1/2013 - 12/31/15
Aragon Pharmaceuticals
The Role of Highly Selective Androgen Receptor (AR) Targeted Therapy in Men With Biochemically Relapsed Hormone Sensitive Prostate Cancer.
The goal of this study is to evaluate the antitumor efficacy of ARN-509 as monotherapy or in combination with testosterone suppression in men with non-metastatic castration-sensitive prostate cancer after definitive prostate therapy.
Role: Site PI

Previous Research Support

A114463

Friedlander (PI)

7/01/10-6/30/11

ASCO Young Investigator Award

Determination of Genotypic Markers of Docetaxel Resistance in Castration Resistant Prostate Cancer.

The goal of this study is to identify the genetic changes associated with chemotherapy resistance that occur in men with prostate cancer treated with docetaxel chemotherapy.

Role: PI