AWARD NUMBER: W81XWH-21-1-0431

TITLE: Developing Strategies to Overcome Prostate Stromal-Derived NRG1-Mediated Resistance to AR Blockade in High-Risk Locally Advanced Prostate Cancer

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CONTRACTING ORGANIZATION: Sloan Kettering Institute for Cancer Research, New York, NY

REPORT DATE: July 2023

TYPE OF REPORT: Annual

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

REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

1. REPORT DATE	2. REPORT TYPE	3. DATES COVERED
July 2023	Annual	01Jun2022-31May2023

4. TITLE AND SUBTITLE

Developing Strategies to Overcome Prostate Stromal-Derived NRG1-Mediated Resistance to AR Blockade in High-Risk Locally Advanced Prostate Cancer

5a. CONTRACT NUMBER

5b. GRANT NUMBER
W81XWH-21-1-0431

5c. PROGRAM ELEMENT NUMBER

6. AUTHOR(S)

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5d. PROJECT NUMBER

5e. TASK NUMBER

5f. WORK UNIT NUMBER

7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)

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Cancer Research
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New York, NY 10021

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

Through pre-clinical studies, we have recently discovered for the first time, that the prostate cancer microenvironment stromal cells, secrete neuregulin (NRG1) in response to androgen blockade, which promotes the cancer cells to survival an androgen deprived state. Our recent finding explains in part why, so few high-risk primary prostate cancers achieve a complete pathologic response to androgen blockade. Importantly, the neuregulin signaling pathway is therapeutically actionable with several clinical agents either FDA approved for other malignancies or in early development. Our research proposal aims to define the downstream mechanisms through which NRG1 promotes cell survival and determine the therapeutic strategies targeting the NRG1 pathway which enhance response to androgen blockade. Our work has the potential to improve our understanding of how the cancer environment influences resistance to androgen blockade and lead to novel combination therapies vastly changing the standard of care for men with high-risk prostate cancer.

15. SUBJECT TERMS
Prostate cancer, androgen receptor, tumor microenvironment, mechanism of resistance, targeted therapy, cancer associated fibroblasts

16. SECURITY CLASSIFICATION OF:			17. LIMITATION OF ABSTRACT	18. NUMBER OF PAGES	19a. NAME OF RESPONSIBLE PERSON
a. REPORT	b. ABSTRACT	c. THIS PAGE			USAMRDC
Unclassified	Unclassified	Unclassified	Unclassified	13	19b. TELEPHONE NUMBER *(include area code)*

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1. INTRODUCTION:

High-grade locally advanced prostate cancer occurs in approximately 30% of newly diagnosed patients and represents a therapeutic challenge as greater than 60% of patients will recur and progress following initial therapy. To improve clinical outcomes for men with high-risk localized prostate cancer, several clinical trials have evaluated the role of complete androgen blockade prior to prostatectomy. Despite ongoing enthusiasm for this approach, complete pathologic response rates are only observed in 4 to 10% of patients. Through pre-clinical studies, we have recently discovered that the prostate cancer microenvironment stromal cells secrete neuregulin (NRG1) in response to androgen blockade, which promotes the cancer cells to survival an androgen deprived state. Our recent finding explains in part why, so few high-risk primary prostate cancers achieve a complete pathologic response to androgen blockade. Importantly, the neuregulin signaling pathway is therapeutically actionable with several clinical agents either FDA approved for other malignancies or in early development. Our research proposal aims to define the downstream mechanisms through which NRG1 promotes cell survival and determine the therapeutic strategies which enhance response to androgen blockade in this context. We have designed studies to define the prevalence of NRG1 expression in high-risk localized prostate cancer following AR inhibition and correlate NRG1 expression with clinical and pathologic response. This work could lead to NRG1 expression as a biomarker of response/resistance to androgen blockade. Our studies are responsive to the overarching challenge of improving survival for men with high-risk localized prostate cancer. Our work has the potential to improve our understanding of how the cancer environment influences resistance to androgen blockade and lead to novel combination therapies vastly changing the standard of care for men with high-risk prostate cancer. Working closely with Dr. Dana Rathkopf (Medical Oncologist) we have developed a neoadjuvant clinical trial (Phase Ib/II) for high-risk primary prostate cancer combining AR and NRG1 pathway inhibition prior to prostatectomy. Through this work, we aim to dramatically improve the cure rates for men with high-risk localized prostate cancer.

2. KEYWORDS:

Prostate cancer, cancer microenvironment, androgen receptor, drug resistance, neuregulin, HER2/3 signaling, targeted therapies

3. ACCOMPLISHMENTS:

What were the major goals of the project?

Specific Aim 1: Dissecting the molecular mechanisms through which NRG1 drives resistance to AR targeted therapies in the prostate cancer epithelial cell.
Major Task 1: Defining the downstream pathways promoting NRG1 mediated resistance to AR inhibition. 1 – 12 months (100% completed)

Major Task 2: Evaluating the NRG1 Paracrine mediated cytokine autocrine loop. 4 – 16 months (100% completed)

Specific Aim 2: Targeting individual nodes of the NRG1- HER2/3-PI3K pathway to overcome resistance to AR targeted therapies.
Major Task 1: Pre-clinical therapeutic trials to overcome NRG1 mediated resistance to AR blockade. 12 – 20 months (90% completed)

Major Task 2: Defining the functional activity of NRG1 in maintaining prostate luminal cells. 20 – 24 months (20% completed)

Specific Aim 3: Evaluating NRG1 expression levels in prostate cancers and response to AR targeted therapies.
Major Task 1: Evaluation of NRG1 expression and response to AR inhibition in high-risk localized prostate cancer. 12 – 36 months (80% completed)

What was accomplished under these goals?

During the second year of our award funding, we have made substantial progress in completing our major goals while furthering our understanding of how NRG1 promotes resistance to AR targeted therapy. Previous work from our group and others defined the role of PI3K-AKT signaling in promoting resistance to AR targeted therapies in the context of genetic loss of PTEN. While genomic loss of PTEN is present in approximately 20 to 40% of high-grade prostate cancers, a substantial percentage of PTEN wild-type prostate cancers demonstrate incomplete response to AR targeted therapies. Our recent work demonstrated that in PTEN wild-type prostate cancer models, microenvironment derived NRG1 activates HER2/3 on the cancer cell promoting resistance to AR inhibition. Through *in vitro* and *in vivo* modeling, we have comprehensively characterized the downstream signaling dependencies promoting resistance to AR inhibition in the context of NRG1 stimulation and the genetic context in which this mechanism is selected for. Furthermore, we have established the therapeutic role of targeting HER2/3 to overcome the resistance to AR inhibition driven by NRG1 secretion from cancer associated fibroblasts.

Specific Aim 1: Dissecting the molecular mechanisms through which NRG1 drives resistance to AR targeted therapies in the prostate cancer epithelial cell.

During our previous reporting period, we defined the role and downstream signaling mechanisms through which secreted NRG1 drives resistance to AR inhibition in PTEN wild-type cancer cells. We had previously discovered that NRG1 stimulation promotes the upregulation and secretion of AREG from the cancer cells. We next asked whether the secretion of AREG plays a role in the AR inhibitor resistance mediated by NRG1. Surprisingly, we discovered that AREG activated upstream PI3K signaling to a significantly lesser degree than NRG1. Furthermore, AREG did not promote resistance to AR inhibition in our model systems and that the phenotype was sole driven through NRG1 mediated kinase signaling (Figure 1).

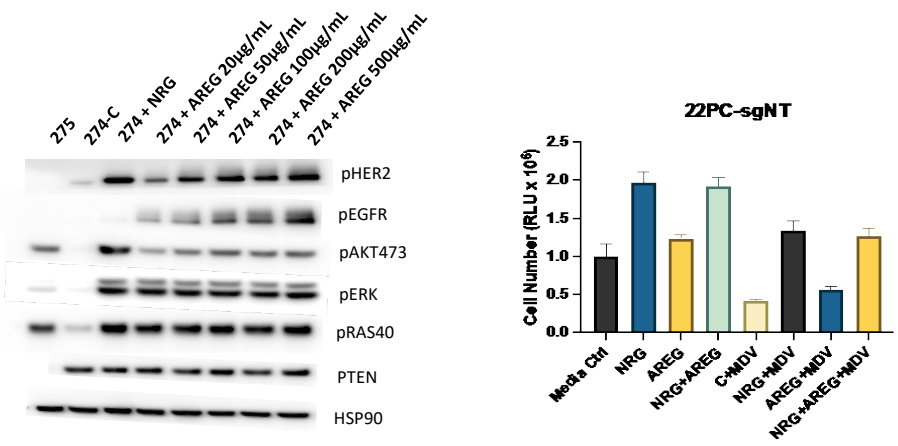

Figure 1. AREG stimulation and impact on AR inhibitor responsiveness in 22PC PTEN wild-type cells.

Specific Aim 2: Targeting individual nodes of the NRG1- HER2/3-PI3K pathway to overcome resistance to AR targeted therapies.

Based on the results of our initial *in vitro* studies targeting the NRG1 - HER2/3 axis performed over the previous progress report period, we conducted *in vivo* studies to address whether targeting HER2/3 can 1) over come NRG1 mediated resistance to AR targeted therapies, and 2) determine if PTEN status is a biomarker of response/resistance. To accomplish this, xenograft models were established using isogenic pairs of PTEN-wildtype and PTEN null 22PC cells with or without CAFs. In accordance with our previously reported *in vitro* studies, we indeed observe that CAFs promoted resistance to AR targeted therapy in the context of wild-type PTEN, and this could be overcome by targeting HER2/3 using an inhibitory antibody (Figure 2).

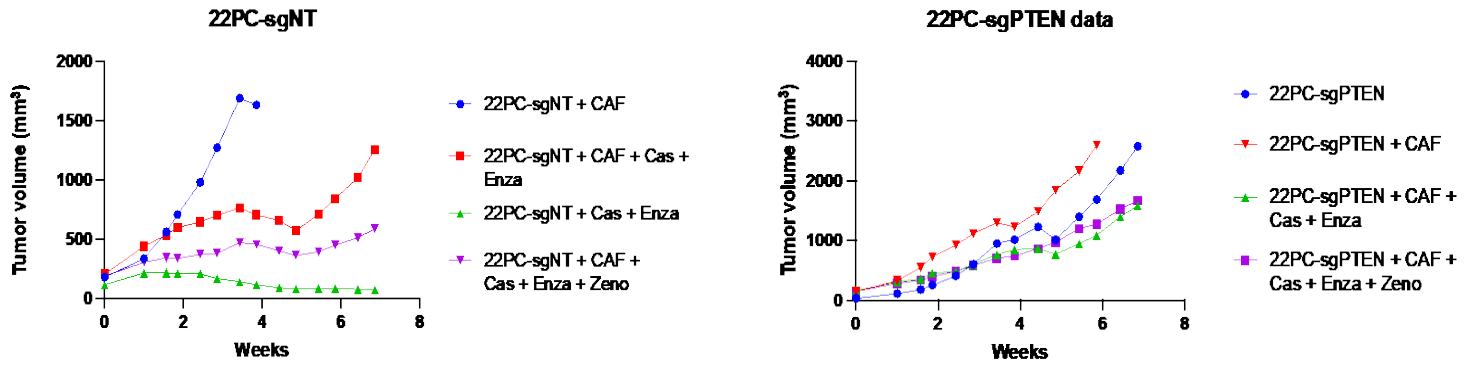

Figure 2. *In vivo* pre-clinical therapeutic studies evaluating the impact of CAFs on promoting resistance to AR inhibition, and the efficacy of targeting HER2/3 (Zeno) to overcome this in the context of PTEN wild-type and PTEN deficient models.

Importantly, in the setting of PTEN loss, these tumors were inherently resistant to AR inhibition, this resistance was not impacted by associated CAFs, and targeting HER2/3 did not offer therapeutic benefit. Histopathologic analysis of the PTEN wild-type tumors demonstrated that associated CAFs promoted HER3 phosphorylation in the tumor cells, this was reduced following HER2/3 inhibition, there was a dramatic reduction in viable cancer cells following treatment, and complete inhibition of proliferation (Figure 3).

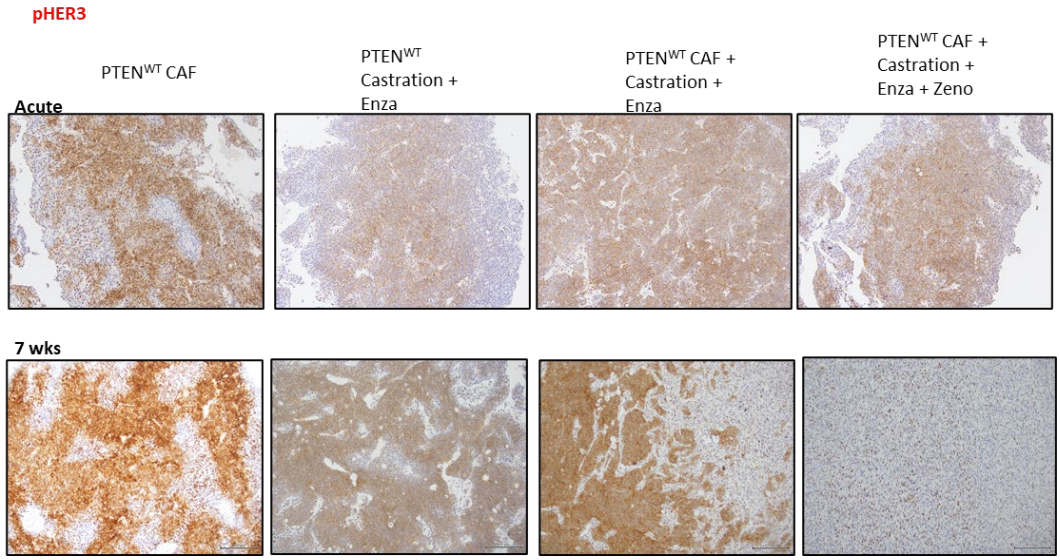

Figure 3. Immunohistochemical staining for NRG1 in our PTEN wildtype 22PC cells with/without CAFs and following AR +/- HER2/3 inhibition.

Specific Aim 3: Evaluating NRG1 expression levels in prostate cancers and response to AR targeted therapies.

In collaboration with Anuradha Gopalan, MD (Pathology) we have established a NRG1 immunohistochemical protocol and have completed staining on 45 prostatectomy specimens to date following neoadjuvant treatment with AR inhibition. Importantly, we have observed staining of NRG1 in the prostate microenvironment stromal cells as well as macrophages surrounding both persistent normal prostate glands and prostate cancer (Figure 4). We are currently in the process of completing staining on the hormonally intact control cohort. NRG1 staining is observed in the microenvironmental cells following AR inhibition regardless of cancer PTEN status indicating that this phenotype may not be dependent of the cancer cell genotype. Data analysis, clinical and pathologic response correlates will be performed once staining is finalized.

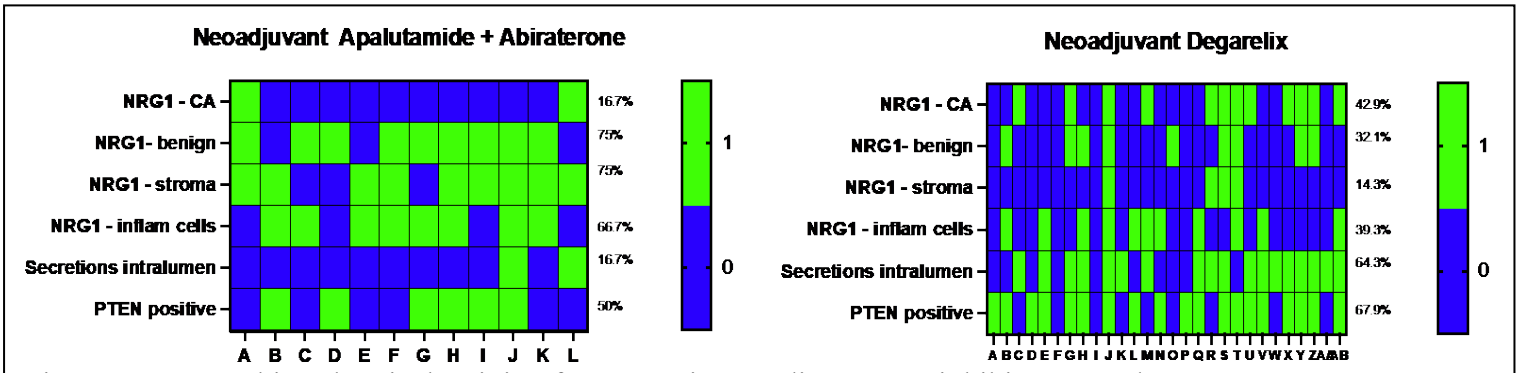

Figure 4. Immunohistochemical staining for NRG1 in neoadjuvant AR inhibitor treated prostate cancer.

What opportunities for training and professional development has the project provided?

Brian Shinder, MD, a T32 urologic oncology fellow at MSKCC joined the lab in August of 2023 and has assisted Ninghui Mao, PhD with the *in vitro* and *in vivo* experiments. Brain is mentored by Ninghui and me and he recently received an ASCO Young Investigator award for his work on the role of NRG1 in promoting resistance to AR targeted therapies. Brian has gained valuable experience in during his time in the lab in experimental design and conduct and has presented his work at lab meetings and seminars at MSKCC. Ninghui has gained further experience in mentoring junior lab members.

How were the results disseminated to communities of interest?

Nothing to Report

What do you plan to do during the next reporting period to accomplish the goals?

During the next reporting period we plan to continue our studies evaluating the role of NRG1 in promoting resistance to AR targeted therapies and optimize strategies to overcome this. We will also complete our immunohistochemical studies for the patient specimens and begin to perform clinical, pathologic, and molecular correlates characterized by NRG1 positivity versus negativity. Also, during the next year, we will begin evaluating role of NRG1 secretion by endogenous prostate stromal cells and maintenance of luminal cells following surgical castration in our genetically engineered mouse models of prostate cancer. We have begun finalizing a manuscript on our studies and aim to submit this at the beginning of the next reporting period. Additionally, we have been discussing with colleagues the development of a phase I/II neoadjuvant clinical trial targeting AR and HER2/3 in patients with PTEN wild-type high grade localized prostate cancer.

4. IMPACT:

What was the impact on the development of the principal discipline(s) of the project?

Our studies have provided us a further understanding of the mechanisms driving NRG1 mediated resistance to AR targeted therapies and have identified therapeutic vulnerabilities that will allow us to restore sensitivity. While our work is still ongoing, our studies have provided the rationale for the initiation of a Phase I/II neoadjuvant clinical trial evaluating combined PI3K and AR pathway inhibition in high-risk localized prostate cancer to improve pathological response rates. This trial is set to open in the next reporting period and is being led by Dana Rathkopf.

What was the impact on other disciplines?

Our studies defining the role of microenvironment derived NRG1 as a mechanism of resistance to therapy has broad implications across malignancies, including hormonal dependent malignancies such as breast cancer.

Since the publication of our initial study, the scientific community has garnered a renewed interest in defining the role of stromal cells in promoting therapeutic resistance.

What was the impact on technology transfer?

Nothing to Report

What was the impact on society beyond science and technology?

Nothing to Report

5. CHANGES/PROBLEMS:

Changes in approach and reasons for change

Nothing to Report

Actual or anticipated problems or delays and actions or plans to resolve them

We have not encountered any problems or delays in conducting our scientific plans.

Changes that had a significant impact on expenditures

Nothing to Report

Significant changes in use or care of human subjects, vertebrate animals, biohazards, and/or select agents

Significant changes in use or care of human subjects

Nothing to Report

Significant changes in use or care of vertebrate animals

Nothing to Report

Significant changes in use of biohazards and/or select agents

Nothing to Report

6. PRODUCTS:

- **Publications, conference papers, and presentations**

  Journal publications.

  Nothing to Report

  Books or other non-periodical, one-time publications.

  Nothing to Report

  Other publications, conference papers and presentations.

Nothing to Report

- **Website(s) or other Internet site(s)**

  Nothing to Report

- **Technologies or techniques**

  Nothing to Report

- **Inventions, patent applications, and/or licenses**

  Nothing to Report

- **Other Products**

  Nothing to Report

7. PARTICIPANTS & OTHER COLLABORATING ORGANIZATIONS

What individuals have worked on the project?

Name:	Brett S Carver, MD
Project Role:	Principal Investigator
Nearest person month worked:	3 calendar months
Contribution to Project:	Dr. Carver oversaw the project studies, directed the experimental designs, analyzed, interpreted the data, and developed the experimental plans.
Funding Support:	Dr. Carver's salary and fringe associated with his effort on this project is supported by this DoD award

Name:	Anuradha Gopalan, MD
Project Role:	Co-Investigator
Nearest person month worked:	1 calendar month
Contribution to Project:	Dr. Gopalan performed the immunohistochemical studies on the patient specimens and analyzed the data.
Funding Support:	Dr. Gopalan's salary and fringe associated with her effort on this project is supported by this DoD award

Name:	Dana Rathkopf, MD
Project Role:	Co-Investigator
Nearest person month worked:	1 calendar month
Contribution to Project:	Dr. Rathkopf participated in the studies of the patient prostate cancer specimens.
Funding Support:	Dr. Rathkopf's salary and fringe associated with her effort on this project is supported by this DoD award

Name:	Irina Ostrovnaya, PhD
Project Role:	Co-Investigator
Nearest person month worked:	1 calendar months

Contribution to Project:	Dr. Ostrovnaya worked on the experimental statistical designs and will participate in the analysis of the human IHC studies
Funding Support:	Dr. Ostrovnaya's salary and fringe associated with her effort on this project is supported by this DoD award
Name:	Ninghui Mao, PhD
Project Role:	Research Scientist
Nearest person month worked:	6 calendar months
Contribution to Project:	Dr. Mao conducted the in vitro and in vivo experiments, analyzed, and interpreted the data.
Funding Support:	Dr. Mao's salary and fringe associated with her effort on this project is supported by this DoD award
Name:	Jillian Love
Project Role:	Research Technician
Nearest person month worked:	1 calendar month
Contribution to Project:	Ms. Love assisted with the in vitro studies evaluating NRG1 stimulation in the cell line assays
Funding Support:	Ms. Love's salary and fringe associated with her effort on this project is supported by this DoD award

Has there been a change in the active other support of the PD/PI(s) or senior/key personnel since the last reporting period?

Dr. Brett Carver (PI) had one new active grant during this reporting period:

*Title: SPORE in Prostate Cancer; Co-Leader, RP2 (Overcoming Microenvironment-Mediated Resistance to AR Pathway Inhibition in High-Risk Prostate Cancer); Co-Director, Preclinical Models Core
*Major Goals: We are applying recent advances in our understanding of the biology of prostate cancer to develop new treatments that are matched to the specific underlying causes of an individual's disease. Our program will allow us to better distinguish between slow-growing cancers and those that require more aggressive treatment, better predict which drugs will work for which patients, better assess if the treatments patients are receiving are working, and develop new therapies for men with advanced disease who are not or no longer benefitting from available treatments.
*Status of Support: Active
Project Number: 2P50CA092629-21A1
Name of PD/PI: Scher, H / Chen, Y
Source of Support: NIH/NCI
Primary Place of Performance: Sloan Kettering Institute For Cancer Research
Project/Proposal Start and End Date (MM/YYYY): 9/21/2022 - 8/31/2027
*Total Award Amount (including Indirect Costs):
Person Months (Calendar/Academic/Summer) per budget period: 1.2 calendar months

Dr. Anuradha Gopalan (Co-Investigator) had four new active grants during this reporting period:

*Title: Functional Characterization and Development of Therapeutic Paradigms for DNA Damage Repair (DDR)–deficient Lethal Prostate Cancer
*Major Goals: Metastatic castration resistant prostate cancer remains an incurable disease. Increasingly, alterations in the genes that regulate the body's processes for repairing damaged DNA have been linked to treatment failure and poor prognosis for men with prostate cancer. In this project, we aim to comprehensively investigate the role of such alterations in the development of lethal, castration resistant prostate cancer with the goal of identifying and developing improved treatments, such as targeted immunotherapy and radiotherapy.
*Status of Support: Active
Project Number: R01CA27496-01A1

Name of PD/PI: Pillarsetty, N
Source of Support: Mount Sinai School of Medicine / National Institutes of Health
Primary Place of Performance: Sloan Kettering Institute For Cancer Research
Project/Proposal Start and End Date (MM/YYYY): 4/1/2023 - 3/31/2028
*Total Award Amount (including Indirect Costs):
Person Months (Calendar/Academic/Summer) per budget period: 0.36 calendar months

*Title: Novel theranostic agents for neuroendocrine prostate cancer
*Major Goals: It is estimated that up to 20% of patients with advanced PCa eventually develop neuroendocrine prostate cancer (NEPC), which is typified by aggressiveness and extreme lethality. Multiple challenges must be overcome to address the needs of NEPC patients, the most critical of which include: (1) identifying the presence of NEPC in patients through an approach that is non invasive, sensitive, and specific; and (2) developing novel treatment strategies that provide durable responses. Toward addressing these challenges, our PCF TACTICAL grant proposal explores the potential of a clinically translatable antibody targeting DLL3 as a molecularly targeted theranostic for the diagnosis and treatment of NEPC with further applications in small cell lung cancer (SCLC).
*Status of Support: Active
Project Number: 2022 PCF TACTICAL Award
Name of PD/PI: Lewis, J
Source of Support: Prostate Cancer Foundation (formerly CaP CURE)
Primary Place of Performance: Sloan Kettering Institute For Cancer Research
Project/Proposal Start and End Date (MM/YYYY): 2/15/2023 - 2/14/2026
*Total Award Amount (including Indirect Costs):
Person Months (Calendar/Academic/Summer) per budget period: 1.2 calendar months

*Title: Enhancing Antigen Presentation and Anti Tumor Immunity for Prostate Cancers through an Intratumorally Delivered, Fc Enhanced Anti CD40 Antibody Therapy
*Major Goals: Our central hypothesis is that agonist anti CD40 antibody therapy leads to induction of a robust intratumoral immune response and formation of tertiary
lymphoid stuctures in the prostate TME, thereby supporting systemic anti tumor immunity. Our long term goal is to clinically develop locally delivered CD40 agonistic antibodies as a novel treatment for prostate cancer.
*Status of Support: Active
Project Number: 22CHAL12
Name of PD/PI: Scher, H
Source of Support: Prostate Cancer Foundation (formerly CaP CURE)
Primary Place of Performance: Sloan Kettering Institute For Cancer Research
Project/Proposal Start and End Date (MM/YYYY): 2/6/2023 2/5/2025
*Total Award Amount (including Indirect Costs):
Person Months (Calendar/Academic/Summer) per budget period: 0.3 calendar months

*Title: SPORE in Prostate Cancer; Core Director, Biospecimen Repository Core; Co-Investigator, Preclinical Models Core
*Major Goals: We are applying recent advances in our understanding of the biology of prostate cancer to develop new treatments that are matched to the specific underlying causes of an individual’s disease. Our program will allow us to better distinguish between slow-growing cancers and those that require more aggressive treatment, better predict which drugs will work for which patients, better assess if the treatments patients are receiving are working, and develop new therapies for men with advanced disease who are not or no longer benefitting from available treatments.
*Status of Support: Active
Project Number: 2P50CA092629-21A1
Name of PD/PI: Scher, H / Chen, Y
Source of Support: NIH/NCI
Primary Place of Performance: Sloan Kettering Institute For Cancer Research
Project/Proposal Start and End Date (MM/YYYY): 9/21/2022 - 8/31/2027
*Total Award Amount (including Indirect Costs):

Person Months (Calendar/Academic/Summer) per budget period: 1.8 calendar months

Dr. Dana Rathkopf (Co-Investigator) had three new active grants during this reporting period:

*Title: SPORE in Prostate Cancer; Clinical Co-Leader, RP2 (Overcoming Microenvironment-Mediated Resistance to AR Pathway Inhibition in High-Risk Prostate Cancer); Co-Director, Career Enhancement Program
*Major Goals: We are applying recent advances in our understanding of the biology of prostate cancer to develop new treatments that are matched to the specific underlying causes of an individual's disease. Our program will allow us to better distinguish between slow-growing cancers and those that require more aggressive treatment, better predict which drugs will work for which patients, better assess if the treatments patients are receiving are working, and develop new therapies for men with advanced disease who are not or no longer benefitting from available treatments.
*Status of Support: Active
Project Number: 2P50CA092629-21A1
Name of PD/PI: Scher, H / Chen, Y
Source of Support: NIH/NCI
Primary Place of Performance: Sloan Kettering Institute For Cancer Research
Project/Proposal Start and End Date (MM/YYYY): 9/21/2022 - 8/31/2027
*Total Award Amount (including Indirect Costs):
Person Months (Calendar/Academic/Summer) per budget period: 0.8 calendar months

*Title: IRONMAN: Enhancing Survivorship in Men with Advanced Prostate Cancer
*Major Goals: The end result of this proposal will be the establishment of a population based, diverse cohort of advanced prostate cancer survivors, and provide a detailed investigation of survivorship in this understudied patient population. The data elements and biospecimens in this study will provide a comprehensive investigation of survivorship in an understudied patient population.
*Status of Support: Active
Project Number: 260388-5124676
Name of PD/PI: Rathkopf, D
Source of Support: T.H. Chan School of Public Health / Prostate Cancer Foundation (formerly CaP CURE)
Primary Place of Performance: Sloan Kettering Institute For Cancer Research
Project/Proposal Start and End Date (MM/YYYY): 1/20/2023 - 1/19/2025
*Total Award Amount (including Indirect Costs):
Person Months (Calendar/Academic/Summer) per budget period: 0.36 calendar months

*Title: The Prostate Cancer Clinical Trials Consortium: Application for Coordinating Center with Affiliate Clinical Research Sites
*Major Goals: The overarching objective is to build on the Consortium's successful acceleration and streamlining of the clinical trial process to fulfill our mission to design, implement, and complete hypothesis driven, early phase trials in prostate cancer, translating scientific discoveries to improved standards of care.
*Status of Support: Active
Project Number: DoD W81XWH22-2-0020
Name of PD/PI: Scher, H
Source of Support: Congressionally Directed Medical Research Programs
Primary Place of Performance: Sloan Kettering Institute For Cancer Research
Project/Proposal Start and End Date (MM/YYYY): 9/30/2022 - 9/29/2026
*Total Award Amount (including Indirect Costs):
Person Months (Calendar/Academic/Summer) per budget period: 1.2 calendar months

Dr. Irina Ostrovnaya (Co-Investigator) had three new active grants during this reporting period:

*Title: Defining the function of Complex I truncating mutations in cancer
*Major Goals: We propose integrative computational/experimental studies to test the overarching hypothesis that CI truncating mutations produce physiologically significant and therapeutically actionable metabolic

changes in tumors.
*Status of Support: Active
Project Number: 1R37CA276200 01
Name of PD/PI: Reznik, E
Source of Support: National Institutes of Health
Primary Place of Performance: Sloan Kettering Institute For Cancer Research
Project/Proposal Start and End Date (MM/YYYY): 1/1/2023 12/31/2027
*Total Award Amount (including Indirect Costs):
Person Months (Calendar/Academic/Summer) per budget period: 0.6 calendar months

*Title: Defining Mechanisms of Progression and Treatment Resistance in Localized Bladder Cancer
*Major Goals: Patients with early-stage bladder cancer have seen few meaningful treatment advances in the last 20 years. Focusing on two groups with particularly acute need for new treatment options, we aim to discover and validate genomic contributors to disease progression and chemotherapy resistance, and to preclinically examine HER2 directed antibody drug conjugate therapy as an alternative to platinum based cytotoxic chemotherapy. Our long-term translational goal is to use integrated clinical and laboratory studies to develop more effective and less toxic treatments for patients with localized bladder cancer, a frequently fatal yet understudied disease.
*Status of Support: Active
Project Number: 1R37CA276946 01
Name of PD/PI: Pietzak, E
Source of Support: National Institutes of Health
Primary Place of Performance: Sloan Kettering Institute For Cancer Research
Project/Proposal Start and End Date (MM/YYYY): 12/1/2022 11/30/2027
*Total Award Amount (including Indirect Costs):
Person Months (Calendar/Academic/Summer) per budget period: 0.6 calendar months

*Title: Regulation of Nectin 4 expression as a mechanism of intrinsic and acquired resistance to enfortumab vedotin in urothelial cancer
*Major Goals: Aim 1: Determine the natural history of Nectin 4 expression as bladder cancer progresses from NMIBC to MIBC to lymph node metastasis. Aim 2: Identify upstream regulators of Nectin 4 expression and characterize additional acquired resistance mechanisms to ADC therapy in UC.
*Status of Support: Active
Project Number: ASCO YIA Award
Name of PD/PI: Chu, C
Source of Support: American Society of Clinical Oncology
Primary Place of Performance: Sloan Kettering Institute For Cancer Research
Project/Proposal Start and End Date (MM/YYYY): 7/1/2022 6/30/2023
*Total Award Amount (including Indirect Costs):
Person Months (Calendar/Academic/Summer) per budget period: 0.6 calendar months

What other organizations were involved as partners?

Nothing to Report

8. SPECIAL REPORTING REQUIREMENTS

COLLABORATIVE AWARDS: Not Applicable

QUAD CHARTS: Not Applicable

9. APPENDICES: None