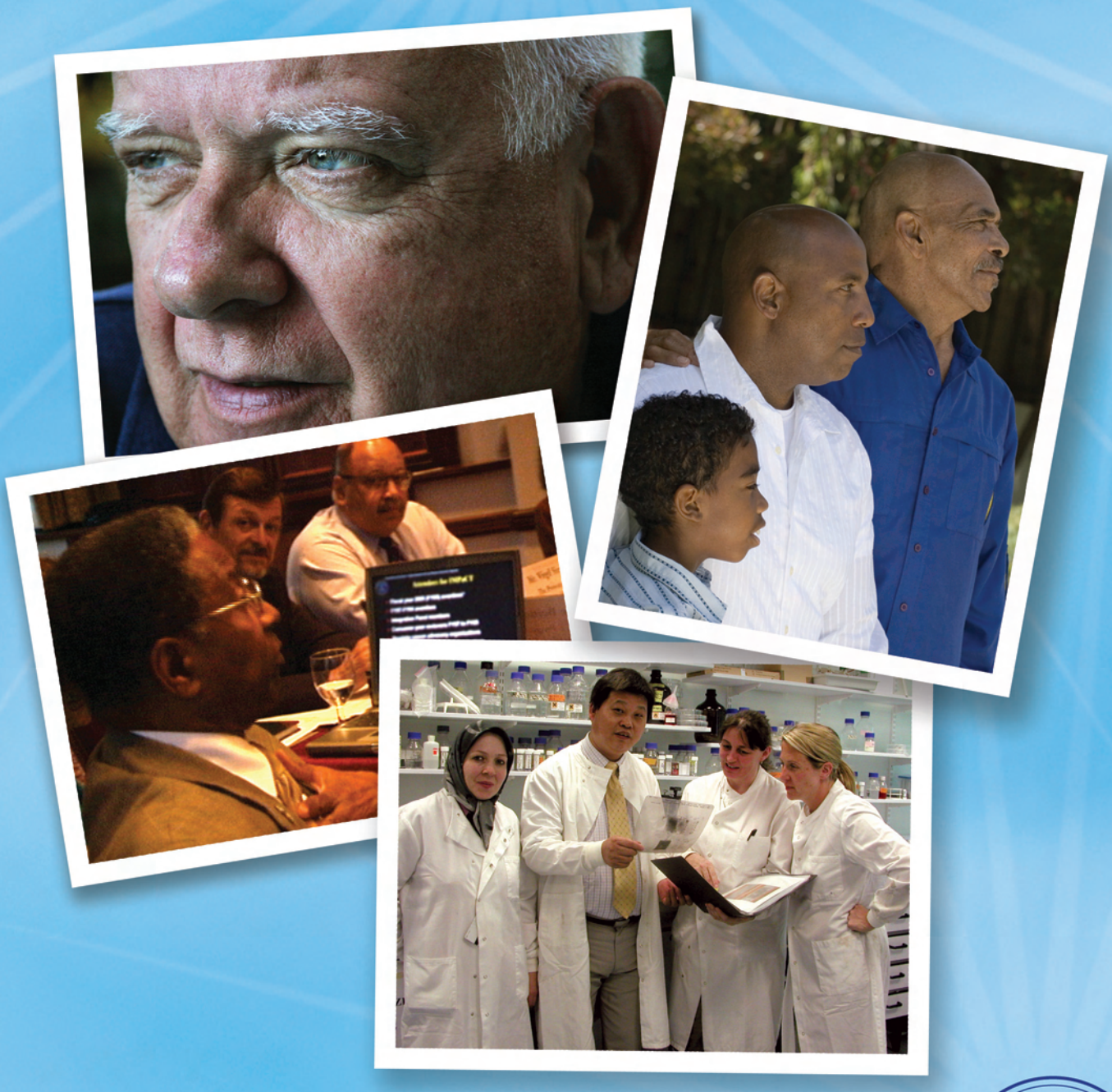
Innovative Minds in Prostate Cancer Today

IMPaCT



PROGRAM

September 5–8, 2007

Hyatt Regency Atlanta

IMPaCT

ABOUT THE MEETING

The Innovative Minds in Prostate Cancer Today (IMPaCT) meeting is sponsored by the Department of Defense (DOD) Prostate Cancer Research Program (PCRP). The purpose of the IMPaCT meeting is to highlight the PCRP's research accomplishments and success. Since its inception in 1997, the PCRP has funded research at universities, hospitals, nonprofit and for-profit institutions, private industry, and state and federal agencies targeted toward conquering prostate cancer. Recognizing that the war against cancer must be fought on multiple fronts, the DOD PCRP has developed a multidisciplinary research portfolio that encompasses both basic and clinical research aimed at preventing, detecting, and treating prostate cancer, and improving the quality of life for men with prostate cancer and their families. The involvement of prostate cancer consumer advocates brings a sense of urgency to the research and ensures that the research is relevant to the understanding and eventual conquest of prostate cancer. The work of PCRP scientists, prostate cancer survivors, and consumer advocates is designed to complement and push the boundaries of prostate cancer research rather than to duplicate more traditional research. The meeting will serve as a forum for the prostate cancer community to discuss current topics in prostate cancer and explore new avenues of research.

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DEPARTMENT OF THE ARMY
US ARMY MEDICAL RESEARCH AND MATERIEL COMMAND
504 SCOTT STREET
FORT DETRICK, MD 21702-5012

REPLY TO
ATTENTION OF

September 5, 2007

Office of the Director

Dear Colleagues:

The proceedings of the U.S. Department of Defense (DOD) Prostate Cancer Research Program (PCRP) Innovative Minds in Prostate Cancer Today (IMPaCT) meeting represent the combined efforts of many individuals committed to addressing the impact of prostate cancer on the lives of those affected and their families and friends. This year's meeting is a milestone: it marks the tenth anniversary of the DOD PCRP – the culmination of a decade of research in our efforts to conquer prostate cancer.

One of the DOD PCRP's most notable and continuing hallmarks is the collaboration among the U.S. Government, the research community, and prostate cancer consumers. This collaboration rests on the efforts of prostate cancer survivors and advocates, the decision of Congress to continue funding research efforts, and the wisdom, vision, and dedication of scientists and health care providers. This collaboration also represents the commitment of the U.S. Army Medical Research and Materiel Command to manage the DOD PCRP in a manner responsive to the vision and equal to the dedication of all of our partners.

The purpose of the IMPaCT meeting is to highlight the PCRP's research accomplishments and success in funding high-impact research, addressing health disparities, and training the next generation of prostate cancer researchers. The meeting will also serve as a forum for the prostate cancer community to discuss current topics in prostate cancer and to explore new avenues of research.

My staff and I thank you for your continuing partnership in our efforts to challenge disease, sustain health, and improve the quality of life for those living with prostate cancer.

Sincerely,

William H. Howell
Senior Executive Service
Director, USAMRMC

DEPARTMENT OF THE ARMY
US ARMY MEDICAL RESEARCH AND MATERIEL COMMAND
1077 PATCHEL STREET
FORT DETRICK, MD 21702-5024

REPLY TO
ATTENTION OF

September 5, 2007

Congressionally Directed Medical Research Programs

Dear Colleagues:

It is a pleasure to welcome you to the Innovative Minds in Prostate Cancer Today (IMPaCT) meeting. For the first time in its 10-year history, the Department of Defense (DOD) Prostate Cancer Research Program (PCRP) is bringing together the scientists and consumer advocates who have built the program.

The IMPaCT meeting provides an unprecedented opportunity for scientists and physicians, prostate cancer survivors and consumer advocates, policymakers, and the military to come together to share and discuss the latest advances in prostate cancer research, convey important information to the public, capture progress in specific fields of research, and identify gaps and new research questions. I express my deepest gratitude to the following individuals for their participation in the DOD PCRP:

- Prostate cancer survivors and advocates, whose courage and commitment created this program. They continue to infuse this program with their passion, inspiration, and vision of conquering prostate cancer.
- The scientists and clinicians funded by this program, who are rising to the challenge. They are our hope for finding a cure.
- Past and present members of the Integration Panel, who, on a yearly basis, crafted an invigorating, responsive, and comprehensive program. They are creative, skilled, visionary, and dedicated to setting fiscally responsible investment strategies, creating award mechanisms to complement these strategies, and then identifying research that will most effectively move us closer to a cure.
- The many members of the DOD PCRP peer review panels, who have met the challenge of reviewing over 7,500 proposals during the past 10 years. Without their expertise and perseverance, the DOD PCRP goal to fund innovative and scientifically meritorious research could not have been accomplished.
- Members of the DOD, the US Army Medical Research and Materiel Command, the PCRP Program Management Team, and support staff, whose energy, enthusiasm, and diligence sustain the DOD PCRP on a daily basis.

I gratefully acknowledge the strength, vision, and devotion of all these dedicated individuals. They have created a research program that is pivotal in forging new pathways toward conquering prostate cancer. The IMPaCT meeting is the culmination of a decade of progress of the DOD PCRP in the fight against prostate cancer. I am pleased to have your participation.

Sincerely,

Janet R. Harris

Janet Harris, Ph.D., RN
Colonel, U.S. Army Nurse Corps
Director

TECHNICAL PLANNING COMMITTEE

Virgil Simons
(TPC Co-Chair)
President
The Prostate Net

Timothy Ratliff, Ph.D.
(TPC Co-Chair)
Director, Cancer Center
Professor of Comparative Pathobiology, School of Veterinary Medicine
Purdue University

Theresa Miller, Ph.D.
(Conference Chair)
CDMRP, USAMRMC

Anne Bertinuson, Ph.D.
(Program Representative)
Science Applications International Corporation

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Deputy Director, CDMRP, USAMRMC

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Assistant Dean for Cancer Programs
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Assistant Professor
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Shuk-mei Ho, Ph.D.
Professor and Chair of the Department of Environmental Health
University of Cincinnatti

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Professor of Surgery
James F. Hardymon Chair of Urological Research
Professor of Molecular and Cellular Biochemistry
University of Kentucky

We recognize the contribution of:
Gayle Vaday, Ph.D.
Program Manager, BCRP, CDMRP, USAMRMC
As initial Program Chair

Donald Miller, M.D., Ph.D.
Director, James Graham Brown Cancer Center
University of Louisville School of Medicine

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Professor of Oncology, Urology, Pharmacology, Medicine, Pathology Associate Director, Translational Research
Sidney Kimmel Comprehensive Cancer Center
John Hopkins Medical Institutions

Merel Nissenberg, J.D.
President
National Alliance of State Prostate Cancer Coalitions

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Professor of Internal Medicine and Urology
University of Michigan

Marva Price, Dr.P.H., R.N., F.N.P.
Director, Family Nurse Practitioner Program
Duke University School of Nursing

Gail S. Prins, Ph.D.
Professor and Director of the University Andrology Laboratory
University of Illinois at Chicago

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Professor, Departments of Radiation Oncology & Urology
Chairman, Department of Radiation Oncology
University of California, San Francisco

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Comprehensive Men's Health Initiative of Atlanta
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Howard R. Soule, Ph.D.
Executive Vice President
Discovery and Translation
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Donald J. Tindall, Ph.D.
Director of Urologic Research
Vice-Chair, Department of Urology,
Professor of Biochemistry and Molecular Biology,
Professor of Urology
Mayo Clinic

James E. Williams, Jr., COL (Ret)
Intercultural Cancer Council, Pennsylvania Prostate Cancer Coalition, Alliance for Prostate Cancer Prevention

The Honorable Ralph Burnett, J.D.
National Prostate Cancer Coalition

Since his prostate cancer diagnosis in 1996, Judge Ralph Burnett worked tirelessly as an advocate for patient rights and prostate cancer prevention. He served as Chairman of the National Prostate Cancer Coalition from 1999 to 2001. Judge Burnett served the Prostate Cancer Research Program as a scientific peer reviewer, as a member of the Integration Panel, and as a member of the IMPaCT Technical Planning Committee. He was actively engaged in planning for the IMPaCT meeting at the time of his death on May 10, 2007. He will be gratefully remembered for his prostate cancer advocacy and as noted by the National Prostate Cancer Coalition, as "a leader and a friend."

TECHNICAL PLANNING SUBCOMMITTEES

Abstract Review Subcommittee

Natasha Kyprianou, M.B., Ph.D. (Chair)
Cory T. Abate-Shen, Ph.D.
The Honorable Ralph Burnett, J.D.
Ralph W. deVere White, M.B., B.Ch., B.A.O.
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Shankar Vallabhajosula, Ph.D.
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Consumer Subcommittee

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Biomedical Scientist, Science Applications International Corporation

Steve Irving, Ph.D.
Peer Review Coordinator, Constella Group, LLC

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Timothy Ratliff, Ph.D. (Chair)
Purdue University

Howard R. Soule, Ph.D. (Chair-Elect)
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Virgil Simons (Member-at-Large)
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John Willey
National Prostate Cancer Coalition

INVITED SPEAKERS

Hal Ackerman
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James Allison, Ph.D.
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Thomas Blank, Ph.D.
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Robert Carey
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Co-sponsored by the Westminster at Lake Ridge and Potomac Hospital

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Tex Us TOO

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President and CEO
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Cancer Coalition, Alliance for Prostate Cancer Prevention

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MODERATORS (CONT.)

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Prostate Cancer Foundation

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Professor of Biochemistry and Molecular Biology,
Professor of Urology
Mayo Clinic and Foundation

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Chief Executive Officer
National Prostate Cancer Coalition

Greg Bielawski
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Raul Blasini
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Us TOO International, Inc.

Maurice Denton
Us TOO International, Inc.

Quince Fleming, Jr.
Rex Healthcare

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Science and Technology Management Associates, LLC

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Central Florida African American Men's Task Force on Prostate Cancer

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Phillip Olsen
Us Too Hawaii Regional Director

Robert Samuels
Founder, Florida Prostate Cancer Net
Founding Chairman, National Prostate Cancer Coalition

Westley Sholes, M.P.A.
California State Prostate Cancer Coalition

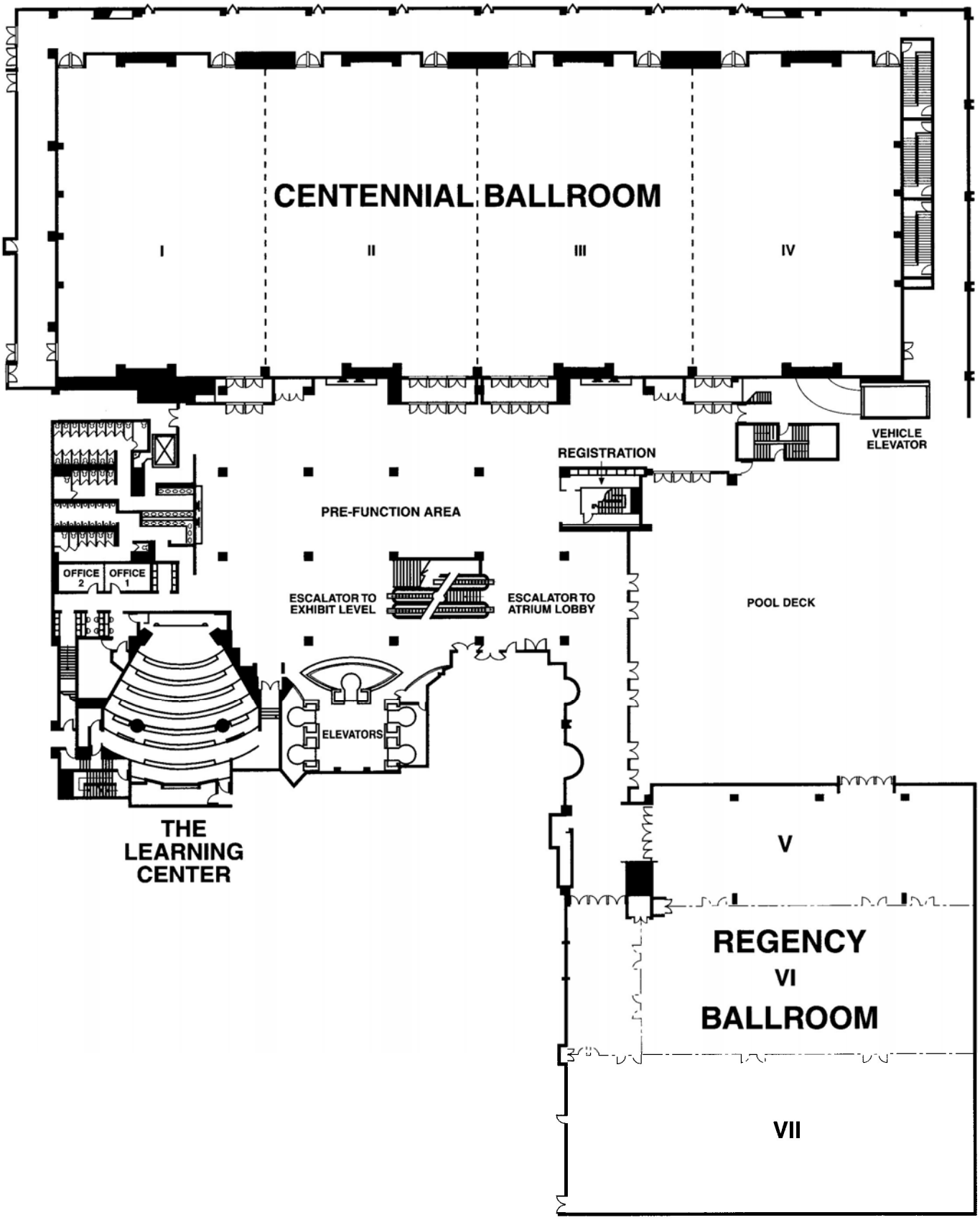
Virgil Simons
President, The Prostate Net

Alan Simpson, Ph.D.
Comprehensive Men's Health Initiative of Atlanta
Morehouse School of Medicine

Justin Sucato
Us TOO Western New York

John Willey
Pennsylvania Prostate Cancer Coalition and Intercultural Cancer Council

HYATT REGENCY ATLANTA FLOOR PLAN



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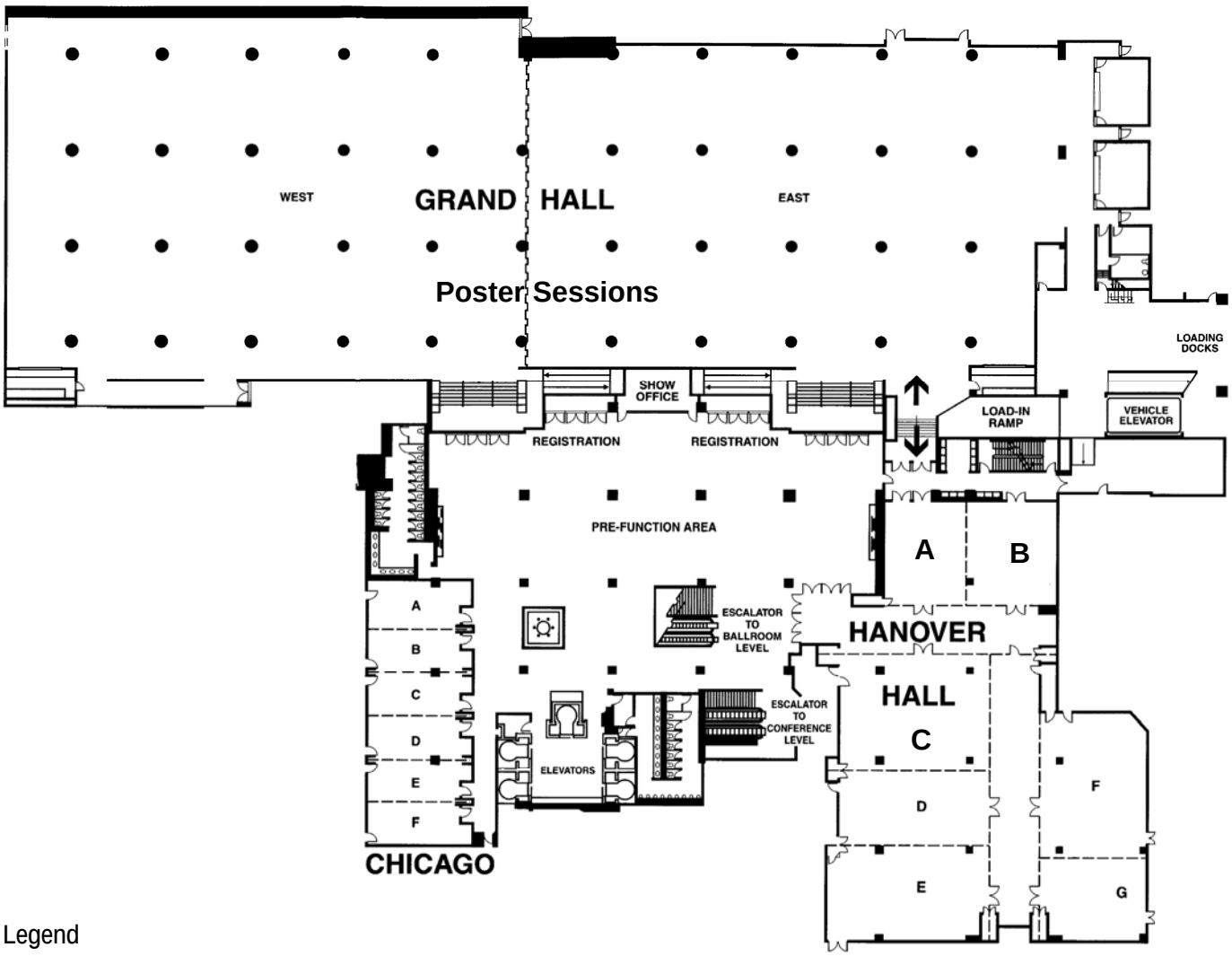

Legend

Press Room	Hanover Hall A
Meeting Office	Hanover Hall B
Speaker Ready Room	Hanover Hall C

GENERAL INFORMATION

All events will start and end on time. Your promptness and consideration for the speakers who have devoted significant time to the preparation of outstanding presentations are deeply appreciated. In addition, please be reminded that this is a non-smoking meeting.

Meeting Location

Hyatt Regency Atlanta
265 Peachtree Street NE
Atlanta, GA 30303
404-577-1234

Registration and Information Center

All participants must register for the meeting. Your name badge provides admittance to all scientific sessions, meals and refreshments concurrent with the scientific sessions, one copy of the Meeting Proceedings and Program Books, and meeting materials. **Name badges must be worn at all times.**

IMPaCT meeting staff will be available at the Registration and Information Center throughout the meeting to answer questions and provide assistance. Messages will be posted on a message board near the Registration and Information Center.

Registration and Information Center Hours

Wednesday, September 5, 2007, 12:00 p.m.–9:00 p.m.
Thursday, September 6, 2007, 6:30 a.m.–6:30 p.m.
Friday, September 7, 2007, 7:00 a.m.–9:00 p.m.
Saturday, September 7, 2007, 7:00 a.m.–12:00 p.m.

Special Assistance

The DOD is committed to making this meeting accessible to all participants. Registrants with special requirements for transportation or hotel accommodations should visit the Registration and Information Center for assistance.

Internet Access

Four terminals with free Internet access are available near the Registration and Information Center.

Audiovisual Presentations

All speaker presentations must be prepared in presentation software compatible with Microsoft PowerPoint and must be on a CD, zip disk, or USB flash drive. **Conventional slide projectors or overhead projectors will not be available at the meeting.** Presentations will be distributed to the meeting rooms via a computer network. Thus you must bring your presentation on disk to the **Speaker Ready Room** as far in advance of your presentation as possible. Please do not bring your presentation on disk directly to the meeting room. Do not plan to connect your individual computer to the projector.

Speaker Ready Room

The Speaker Ready Room is located in Hanover Hall C. Speakers are required to deliver their presentations in presentation software compatible with Microsoft PowerPoint on a CD, zip disk, or USB flash drive to a staff member in the Speaker Ready Room no less than 1 hour before their session begins. We recommend delivering the presentation upon arrival at the meeting so that there is ample time to load and review the presentation. The Speaker Ready Room is open from 1:00 p.m. to 9:00 p.m. on Wednesday, September 5; from 6:30 a.m. to 7:00 p.m. on Thursday, September 6 and Friday September 7; and from 7:00 a.m. to 12:00 p.m. on Saturday, September 8.

Concurrent Sessions

Early Morning
Four sessions are scheduled to run concurrently during each early morning block; participants may move among Early Morning Sessions. Presenters are asked to adhere to the strict time schedule of 1 hour.

Symposia
Six sessions are scheduled to run concurrently during each Symposia block, and participants may move among Symposia. Presenters are asked to adhere to the strict time schedule of 1.5 hours.

Poster Sessions

Posters will be displayed in the Grand Hall. Poster board assignments and sessions can be found beginning on page 50. An informational kiosk detailing the exact location of each poster will be located in the front of the Grand Hall.

Poster boards will be available for setup from 1:00 p.m. to 8:00 p.m. on Wednesday, September 5 and from 7:00 a.m. to 11:00 a.m. on Thursday, September 6. All individuals with invited abstracts are obliged to have their posters assembled and ready for display in the Grand Hall by 11:00 a.m. on Thursday, September 6.

Investigators must be available at their posters for discussion during their scheduled poster session. Within each poster session, odd-numbered poster presenters

must attend their posters during the first hour; even-numbered presenters during the second hour. All posters must be removed by 1 p.m. on Saturday, September 8.

Receptions

All registrants are invited and encouraged to attend the following activities that have been planned as additional opportunities to interact with colleagues and make new acquaintances.

- Welcome Reception: Wednesday, 7:30 p.m.–9:00 p.m.
- Dinner Reception: Friday, 7:00 p.m.–9:00 p.m.

Lunch

Lunch will be provided in the Grand Hall during the poster sessions on Thursday and Friday. Lunch will be provided in the Centennial Ballroom on Saturday.

Transportation

Transportation to/from Airport:
From Hartsfield International airport, go to the transportation booth and request a shuttle to the Hyatt Regency Atlanta Downtown. The Atlanta Link is recommended (404-524-3400). No reservations are necessary. It runs from 6 a.m. to midnight from the airport and leaves the hotel every 10 and 40 minutes after each hour. The cost is $16 one way/$28 round trip per person.

Taxi Service:
The cost for taxi service to/from the airport is $30.00 plus $2.00 per each additional person.

Metered Rates:
$2.50 = First 1/7 mile
$2.00 = Each additional 1 mile
$21.00/hr = Waiting time

Amtrak: (3 miles North of hotel) Taxi service is approximately $11 plus tax.
Greyhound/Trailways: Taxi service is approximately $7 plus tax. Taxi service within downtown Atlanta has an $8 minimum.

Local Transit System:
M.A.R.T.A. – (Metro Atlanta Rapid Transit Authority) $1.75 per ride. The rail system runs approximately every 10 minutes. The Hyatt is connected to the Peachtree Center Train Station via Peachtree Center Mall (15 minutes from the airport). (Airport pickup is at the baggage claim, TH Terminal.) To get to the Hyatt, take M.A.R.T.A. to the Peachtree Center Station and exit North East toward Peachtree Center Mall.

Travel Assistance

A representative of SAIC travel is available at the Registration and Information Center to provide travel assistance for participants whose travel was booked by SAIC travel.

Abstracts Available on the Internet

All participants will receive one copy of the Meeting Proceedings and Program Books. Following the IMPaCT meeting, all abstracts presented at the meeting will be available at www.cdmrpcures.com.

Press Relations

The Press Room is in Hanover Hall A. All members of the press should report to the Press Room to register. Dr. Heather Sansbury is the IMPaCT Press Manager. All press activities will be managed by Michael Beckerich and Toni Haubert of Dorland Global Health.

Continuing Education Accreditation

Please see program addendum.

Wednesday, September 5, 2007

		Registration 12:00 p.m.– 9:00 p.m.	Poster Board/ Exhibitor Setup 1:00 p.m.– 8:00 p.m.
5:00–6:00 p.m.	**WELCOME AND MOMENT OF SILENCE (Session 1)** COL Janet Harris, CDMRP Director USAMRMC Representative PCRP Video Moment of Silence Virgil Simons: The Role of Consumers in Science		
6:00–7:30 p.m.	**KEYNOTE SPEAKERS (Session 2)** **The Internationalization of Medicine** Michael Milken Andrew von Eschenbach Don Coffey		
7:30–9:00 p.m.	**Welcome Reception**		

PROGRAM AT-A-GLANCE

Thursday, September 6, 2007

<table>
<tr><td rowspan="3">7:00–8:00 a.m.</td><td colspan="12">EARLY MORNING EDUCATIONAL SESSIONS</td><td rowspan="11">Registration
6:30 a.m.–6:30 p.m.</td><td rowspan="11">Poster Board/ Exhibitor Setup
7:00 a.m.–11:00 a.m.</td></tr>
<tr><td colspan="3">Session 3</td><td colspan="3">Session 4</td><td colspan="3">Session 5</td><td colspan="3">Session 6</td></tr>
<tr><td colspan="3">Therapeutics Advances
Speakers: Charles Rubin, Martin Gleave, and Daniel George</td><td colspan="3">The Business of Medicine
Speakers: Westley Sholes, Monica Liebert, and David Penson</td><td colspan="3">Immunotherapy Trials
Speakers: James Kiefert, Charles Drake, and Susan Slovin</td><td colspan="3">Prostate Cancer 101
Speakers: Larry Junker and Kenneth Pienta</td></tr>
<tr><td>8:15–9:30 a.m.</td><td colspan="12">WELCOME AND MOMENT OF SILENCE
PLENARY SESSION: HEALTH DISPARITIES (Session 7)
Moderator: Lovell Jones
Speakers: James Williams, Harold Freeman, and Mack Roach III</td></tr>
<tr><td>9:45–11:00 a.m.</td><td colspan="12">SPOTLIGHT SYMPOSIUM: Racial Differences in Prostate Cancer: The North Carolina–Louisiana Prostate Cancer Project (PCaP) (Session 8)
Speakers: James Williams and James Mohler</td></tr>
<tr><td>11:00–11:15 a.m.</td><td colspan="12">BREAK</td></tr>
<tr><td rowspan="3">11:15 a.m.–12:45 p.m.</td><td colspan="12">SYMPOSIA SESSIONS I</td></tr>
<tr><td colspan="2">Session 9</td><td colspan="2">Session 10</td><td colspan="2">Session 11</td><td colspan="2">Session 12</td><td colspan="2">Session 13</td><td colspan="2">Session 14</td></tr>
<tr><td colspan="2">Genetics of Prostate Cancer</td><td colspan="2">Advocacy and Community Involvement</td><td colspan="2">Understanding Angiogenesis</td><td colspan="2">Androgen Receptor I</td><td colspan="2">Stem Cells</td><td colspan="2">Novel Therapeutics</td></tr>
<tr><td>1:00–3:00 p.m.</td><td colspan="12">POSTER SESSION/LUNCH (Session 15)</td></tr>
<tr><td>3:00–3:15 p.m.</td><td colspan="12">BREAK</td></tr>
<tr><td>3:15–5:00 p.m.</td><td colspan="12">PLENARY SESSION: TRANSLATIONAL RESEARCH (Session 16)
Moderator: Timothy Ratliff
Speakers: Greg Bolden, Arul Chinnaiyan, James Allison, Shuming Nie, and John Carpten</td></tr>
<tr><td rowspan="3">5:15–6:45 p.m.</td><td colspan="12">SYMPOSIA SESSIONS II</td></tr>
<tr><td colspan="2">Session 17</td><td colspan="2">Session 18</td><td colspan="2">Session 19</td><td colspan="2">Session 20</td><td colspan="2">Session 21</td><td colspan="2">Session 22</td></tr>
<tr><td colspan="2">Molecular Pathways of Cancer Progression</td><td colspan="2">Epidemiology and Biomarkers</td><td colspan="2">Preclinical Drug Discovery</td><td colspan="2">Prostate Cancer Imaging</td><td colspan="2">Signal Transduction I</td><td colspan="2">Training the Next Generation</td></tr>
</table>

Friday, September 7, 2007

<table>
<tr><td rowspan="2">7:00–8:00 a.m.</td><td colspan="4">EARLY MORNING EDUCATIONAL SESSIONS</td><td rowspan="15">Registration

7:00 a.m.–
9:00 p.m.
↓</td></tr>
<tr><td>Session 23
Chemoprevention
Speakers: Richard Gillespie, Eric Klein, and Michael Pollak</td><td>Session 24
Lifestyle Issues
Speakers: Manuel Vasquez, Meir Stampfer, and Edward Giovannucci</td><td>Session 25
Imaging
Speakers: William Sproat, Hedvig Hricak, and Mukesh Harisinghani</td><td>Session 26
Prostate Cancer Screening
Speakers: Merel Nissenberg, William Catalona, and Eddie Reed</td></tr>
<tr><td>8:15–11:00 a.m.</td><td colspan="4">WELCOME AND MOMENT OF SILENCE
PLENARY SESSION: BASIC SCIENCE (Session 27)
Moderator: Gail Prins
Speakers: Thomas Blank, Diane Robins, Angelo DeMarzo, John Isaacs, and Shuk-mei Ho</td></tr>
<tr><td>9:30–9:45 a.m.</td><td colspan="4">BREAK</td></tr>
<tr><td>11:00 a.m.–12:15 p.m.</td><td colspan="4">SPOTLIGHT SYMPOSIUM: The Prostate Cancer Clinical Consortium: Translation of Scientific Discovery to the Clinic (Session 28)
Speakers: Roland Young and Howard Scher</td></tr>
<tr><td>12:30–2:30 p.m.</td><td colspan="4">POSTER SESSION/LUNCH (Session 29)</td></tr>
<tr><td>2:45–4:15 p.m.</td><td colspan="4">PLENARY SESSION: CLINICAL RESEARCH (Session 30)
Moderator: Christopher Logothetis
Speakers: Robert Carey, Stephen Freedland, Laurence Klotz, Fritz Schröder, and Christopher Warlick</td></tr>
<tr><td>4:15–4:30 p.m.</td><td colspan="4">BREAK</td></tr>
<tr><td rowspan="2">4:30 a.m.–6:00 p.m.</td><td colspan="4">SYMPOSIA SESSIONS III</td></tr>
<tr><td colspan="4"><table><tr><td>Session 31
Prevention, Screening, and Early Detection</td><td>Session 32
Developing Immunotherapy for Prostate Cancer</td><td>Session 33
Tumor Suppressors</td><td>Session 34
Targeting Apoptosis</td><td>Session 35
Metastasis</td><td>Session 36
Multicenter Collaborations for Clinical Trials</td></tr></table></td></tr>
<tr><td>7:00–9:00 p.m.</td><td colspan="4">DINNER RECEPTION
Spelman Glee Club
Hal Ackerman Presents a One-man Play:
“Testosterone: How Prostate Cancer Made a Man of Me”</td></tr>
</table>

PROGRAM AT-A-GLANCE

Saturday, September 8, 2007

	EARLY MORNING EDUCATIONAL SESSIONS				Registration
	Session 37	Session 38	Session 39	Session 40	7:00 a.m.–12:00 p.m.
7:00–8:00 a.m.	**Genetic Epidemiology** Speakers: Virgil Simons, Jianfeng Xu, and Henrik Grönberg	**Complementary and Alternative Medicine** Speakers: James McGuinness, Wendy Demark-Wahnefried, and Peter Nelson	**Hormone Refractory Prostate Cancer** Speakers: William Bright, Donald Tindall, and Edward Gelmann	**Treatment and Management of Prostate Cancer** Speakers: Winston Dyer, Daniel Petrylak, Curtis Pettaway, and Richard Valicenti	
8:15–9:30 a.m.	**WELCOME AND MOMENT OF SILENCE** **SPOTLIGHT SYMPOSIUM: Manhattan Project for Targeting the Lethal Phenotypes of Prostate Cancer (Session 41)** Speakers: John Willey and Jonathan Simons				
9:30–9:45 a.m.	**BREAK**				
10:00–11:00 a.m.	**PLENARY SESSION: QUALITY OF LIFE (Session 42)** Speakers: Richard and Desiree Howe, Sara Knight, and David Latini				
11:15 a.m.–12:45 p.m.	**SYMPOSIA SESSIONS IV**				
	Session 43	**Session 44**	**Session 45**	**Session 46**	**Session 47**	**Session 48**
	Treatment, QOL, and Other Health Outcomes	Signal Transduction II	Tumor Microenvironment	Etiology and Novel Biomarkers	Androgen Receptor II	Collaborative Partnership Panel
12:45–2:45 p.m.	**Luncheons (Trainees; HBCU) (Session 48)**					

Wednesday, September 5, 2007

Time	Session	Event	Room
12:00–9:00 p.m.		Registration	
1:00–6:00 p.m.		Poster Board/Exhibitor Setup	Grand Hall
5:00–6:00 p.m.	1	WELCOME AND MOMENT OF SILENCE COL Janet Harris, CDMRP Director USAMRMC Representative PCRP Video Moment of Silence Virgil Simons: The Role of Consumers in Science	Centennial III and IV
6:00–7:30 p.m.	2	KEYNOTE SPEAKERS The Internationalization of Medicine Michael Milken Andrew von Eschenbach Don Coffey	
7:30–9:00 p.m.		Welcome Reception	Regency V and VI

Thursday, September 6, 2007

Time	Session	Event	Room
6:30 a.m.–6:30 p.m.		Registration	
7:00–11:00 a.m.		Poster Board/Exhibitor Setup	Grand Hall
7:00–8:00 a.m.		EARLY MORNING EDUCATIONAL SESSIONS	
	3	Therapeutics Advances Speakers: Charles Rubin, Martin Gleave, and Daniel George	Centennial II
	4	The Business of Medicine Speakers: Westley Sholes, Monica Liebert, and David Penson	Regency V
	5	Immunotherapy Trials Speakers: James Kiefert, Charles Drake, and Susan Slovin	Regency VI
	6	Prostate Cancer 101 Speakers: Larry Junker and Kenneth Pienta	Regency VII
8:15–9:30 a.m.		WELCOME AND MOMENT OF SILENCE	
	7	PLENARY SESSION: HEALTH DISPARITIES Moderator: Lovell Jones Speakers: James Williams, Lovell Jones, and Mack Roach III	Centennial III and IV
9:45–11:00 a.m.	8	SPOTLIGHT SYMPOSIUM: Racial Differences in Prostate Cancer: The North Carolina–Louisiana Prostate Cancer Project (PCaP) Speakers: James Williams and James Mohler	Centennial III- IV
11:00-11:15 a.m.		*Break*	

Thursday, September 6, 2007

Time	Session	Event	Room
11:15 a.m.–12:45 p.m.		SYMPOSIA SESSIONS I	
	9	Genetics of Prostate Cancer Consumer Speaker: Westley Sholes Moderator: Ralph deVere White *Targeted Molecular Analysis of a Prostate Cancer Susceptibility and Metastasis Gene* John A. Martignetti *Genome Wide Profiling of Gene States* Spyro Mousses *Two Prostate Cancer Susceptibility Variants at 8Q24 Identified through a Genome-wide Association Study* Patrick Sulem *DNA Copy Number Alterations in Prostate Cancer* William B. Isaacs *Epigenetic Regulation of Estrogen Receptor-Beta Expression in Prostate Cancer* Shuk-mei Ho	Centennial II
	10	Advocacy and Community Involvement Consumer Speaker: Richard Atkins Moderator: Merel Nissenberg *American Cancer Society's Man to Man Program* Durado D. Brooks *California Prostate Cancer Coalition: Evolution through Advocacy* Sarah E. Connor *Community Engagement for Ensuring Prostate Health and Cancer Survivorship among African Americans* Alan N. Richmond *Multimedia Education on Prostate Cancer in Barbershops: A Novel Paradigm for Community-based Health Education* Virgil Simons	Learning Center
	11	Understanding Angiogenesis Consumer Speaker: Darryl Mitteldorf Moderator: Balakrishna Lokeshwar *Protocadherin-PC (PCDH-PC) in Hormone Refractory Prostate Cancer: Targeting PCDH-PC as a Means to Enhance the Effectiveness of Hormone Therapy* Ralph Buttyan *Interaction of KAI1 on Tumor Cells with DARC on Vascular Endothelium Leads to Metastasis Suppression* Kounosuke Watabe *Calcitriol, Tumor Vasculature and Prostate Cancer: The Role of CYP24* Donald L. Trump *A Novel Function of Angiogenin in Androgen-independent Prostate Cancer* Guo-Fu Hu *Caveolin-1 Uptake and Pro-angiogenic Activities in Prostate Cancer* Timothy C. Thompson	Regency V

AGENDA Thursday

12	**Androgen Receptor I** Consumer Speaker: William Bright Moderator: Gail Prins	Regency VI
	Single Cell Analysis of the Androgen Receptor Mechanism of Action to Identify New Ligands to Treat Prostate Cancer Marco Marcelli	
	Oncogenic VAV3 Promotes Ligand-independent Androgen Receptor Activation via the Rho GTPase, Rac1, in Androgen-independent Prostate Cancer Progression Kerry L. Burnstein	
	Androgen Receptor Decoy Molecules Block the Growth of Prostate Cancer Marianne D. Sadar	
	Prostate Epithelial Androgen Receptor Suppresses Prostate Growth and Tumor Invasion Chawnshang Chang	
	Enhancing Androgen Receptor Recruitment of Corepressor NCoR for the Treatment of Prostate Cancer That Relapses after Androgen Deprivation Therapy Steven P. Balk	
	HIC-5/ARA55: A Regulator of Endocrine and Paracrine Signaling in Prostate Stroma Donald B. Defranco	
13	**Stem Cells** Consumer Speaker: Robert Carey Moderator: Susan Kasper	Centennial III-IV
	Prostate-specific Inactivation of p53 and RB Leads to Metastatic Cancer Arising from the Stem/Progenitor Cell-enriched Proximal Region of Prostatic Ducts Zongxiang Zhou	
	In Vitro-Ex Tempore Model for Cancer Stem Cell-targeted Drug Development Galina I. Botchkina	
	The Identification and Properties of Prostatic Stem Cells E. Lynette Wilson	
	p63 in Development and Maintenance of the Prostate Epithelium Chiara Grisanzio	
	Stem Cells from Human Prostate Cancers: Isolation, Propagation and Phenotypic Analysis Norman J. Maitland	
14	**Novel Therapeutics** Consumer Speaker: Robert Samuels Moderator: Howard Soule	Regency VII
	A Phase I Trial of a G-Quartet Forming Oligonucleotide Aptamer in Patients with Advanced Cancer Donald M. Miller	
	Preclinical Development of Novel Phosphorodiamidate Morpholino Oligomers Based Therapeutics Targeting Insulin-like Growth Factor Axis for Prostate Cancer Gayathri R. Devi	

AGENDA — Thursday

11:15 a.m.–12:45 p.m. — SYMPOSIA SESSIONS I, Novel Therapeutics (cont.) — Regency VII

Molecular Target-based Drug Discovery for Prostate Cancer
Ching-Shih Chen

^{177}Lutetium-Dota-J591, a Radiolabeled Monoclonal Antibody Specific to the Extracellular Domain of Prostate Specific Membrane Antigen (PSMA): Radioimmunotherapy (RIT) Studies in Patients with Prostate Cancer
Neil Bander

Design of Small Molecules Targeting Prostate Specific Membrane Antigen
Xinning Wang

Towards the Induction of Senescence as a Treatment for Advanced Prostate Cancer
David F. Jarrard

1:00–3:00 p.m. — 15 — POSTER SESSION/LUNCH — Grand Hall

P1	Prostate Cancer Advocacy	P9	Animal Models
P2	Quality of Life	P10	Prostate Development
P3	Prostate Cancer Screening	P11	Migration/Invasion/Metastasis
P4	Health Disparities	P12	Bone Metastasis
P5	Cell Cycle Control	P13	Mechanisms of Resistance
P6	Signaling I	P14	Preclinical Therapeutics
P7	Molecular Mechanism of Prostate Cancer Progression	P15	Gene Therapy
		P16	Novel Therapies
P8	Biomarkers I	P17	Radiation Therapy

3:00–3:15 p.m. — *Break*

3:15–5:00 p.m. — 16 — PLENARY SESSION: Translational Research — Centennial III-IV
Moderator: Timothy Ratliff
Speakers: Greg Bolden, Arul Chinnaiyan, James Allison, Shuming Nie, and John Carpten

5:15–6:45 p.m. — SYMPOSIA SESSIONS II

17 — Molecular Pathways of Cancer Progression — Centennial II
Consumer Speaker: Virgil Simons Moderator: Ralph deVere White

Integrative Metabolomics of Pathways to Prostate Cancer Progression
Arun Sreekumar

Jun Expression Is Associated with Recurrence of Prostate Cancer
Xuesong Ouyang

The Role of SUMOylation in Cancer Metastasis
Ling Cai

Integrative Microarray Analysis of Pathways (IMAP) Dysregulated in Metastatic Prostate Cancer
Sunita R. Setlur

Predicting Prostate Cancer Recurrence by Gene Expression Analysis of Formalin-fixed, Paraffin Embedded Tissue
Richard B. Everson

18 — Epidemiology and Biomarkers — Regency V
Consumer Speaker: Benjamin Floyd Moderator: Gary Schwartz

Dietary Fat and Prostate Cancer Risk among African-Americans and Africans: A Case-Control Study
Flora A. Ukoli

A Prospective Study of Plasma Vitamin D Metabolites, Vitamin D Receptor Polymorphisms, and Prostate Cancer
Haojie Li

Is Disparity in Prostate Cancer Rates among Different Ethnic Groups Associated with Well-done Meat Consumption and Specific Acetylator Genotypes
Sangita Sharma

An Investigation of Genetic Risk Factors for Prostate Cancer Using a Large Population-based Cohort
Angela Cox

A Novel Intermediate Endpoint for Predicting Overall Survival in Men with Metastatic Castration-recurrent Prostate Cancer (CRPC): An Analysis of Nine Caleb Studies
Susan Halabi

The Relation between Surrogates of Energy Balance on Hormones Associated with Prostate Cancer Risk in the Health Professionals Follow-up Study
Edward L. Giovannucci

19 Preclinical Drug Discovery — Regency VI
Consumer Speaker: Norwood Sloan Moderator: Deborah Lannigan

More Is Not Always Better: Using the Dog Model to Identify What Dose of Selenium Provides the Best Protection against Prostate Cancer
David J. Waters

Enhancement of Intermittent Androgen Ablation by Off-cycle Maintenance with Finasteride in a Prostate Cancer Xenograft Model
Zhou Wang

The Stretch-activated Calcium Channel as a Central Regulator of Prostate Cancer Cell Migration
Owen P. Hamill

Heparanase in Prostate Tumorigenesis: Potential for Diagnosis and Therapy
Michael Elkin

The Role of the SUMO-specific Protease SENP1 in Prostate Cancer Development
Tasneem Bawa-Khalfe

NSAIDs Therapy in Prostate Cancer: Signaling and Functional Roles of MDA-7/IL-24
Luiz F. Zerbini

20 Prostate Cancer Imaging — Centennial III-IV
Consumer Speaker: George Gamota Moderator: Lucia Languino

Detection of Prostate Cancer with Contrast-enhanced Microflow Imaging
Ethan J. Halpern

Anti-prostate Stem Cell Antigen (PSCA) Antibody Fragments for ImmunoPET Detection of Prostate Cancer
Robert E. Reiter

Non-invasive Detection and Therapeutic Targeting of Cancer in the Prostate Using Fluorine-18 Fluorocholine Positron Emission Tomography
Sandi A. Kwee

Application of PHLIP-Nanotechnology for Specific Delivery of Imaging Probes and Toxin (Phalloidin) to Prostate Cancer Cells
Yana K. Reshetnyak

AGENDA — Thursday

5:15–6:45 p.m.

SYMPOSIA SESSIONS II, Prostate Cancer Imaging (cont.) — Centennial III-IV

Preparation of Radioisotope-doped Iron Oxide Nanoparticles as Dual Modality Imaging Probes
Xiankai Sun

PSMA-based PET Ligands for Prostate Cancer Imaging
Martin G. Pomper

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21 Signal Transduction I — Regency VII

Consumer Speaker: Maurice Denton Moderator: Cindy Miranti

Thioredoxin Reductase 1 Expression Coincides with the Onset of Androgen-independent Growth of Prostate Cancer
Swaroop S. Singh

Cholesterol and Signal Transduction in Prostate Cancer
Michael R. Freeman

Regulation of NKX3.1 by Phosphorylation and Ubiquitination
Charles J. Bieberich

P18INK4C and PTEN Constrain a Positive Regulatory Loop between Cell Growth and Cell Cycle Control
Feng Bai

Protein Arginylation Is a Posttranslational Modification That Is Relevant to Cancer Progression and Provides New Possibilities for Prostate Cancer Treatment
Anna S. Kashina

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22 Training the Next Generation — Learning Center

Consumer Speaker: Quince Fleming Moderator: Marva Price

Increasing Minority Biomedical Researchers in Prostate Cancer Research through Academic Affiliations between UNTHSC and HBCU
Jamboor K. Vishwanatha

Minority Undergraduate Research Scholars Training in Prostate Cancer: The Delaware Consortium Experience
Robert A. Sikes

Prostate Cancer Summer Research Training Program–Collaboration of the University of Iowa and Lincoln University
David M. Lubaroff

Prairie View A&M/Baylor College of Medicine Smart Summer Undergraduate Prostate Cancer Research Project
Nancy L. Weigel

Project Inspire–HBCU Undergraduate Collaborative Summer Training Program to Inspire Students in Prostate Cancer Research
Nagi B. Kumar

Friday, September 7, 2007

Time	Session	Event	Room
7:00 a.m.–9:00 p.m.		Registration	
7:00–8:00 a.m.		EARLY MORNING EDUCATIONAL SESSIONS	
	23	Chemoprevention Speakers: Richard Gillespie, Eric Klein, and Michael Pollak	Regency V
	24	Lifestyle Issues Speakers: Manuel Vasquez, Meir Stampfer, and Edward Giovannucci	Regency VI
	25	Imaging Speakers: William Sproat, Hedvig Hricak, and Mukesh Harisinghani	Regency VII
	26	Prostate Cancer Screening Speakers: Merel Nissenberg, William Catalona, and Eddie Reed	Centennial II
8:15–11:00 a.m.		WELCOME AND MOMENT OF SILENCE	
	27	PLENARY SESSION: BASIC SCIENCE Moderator: Gail Prins Speakers: Thomas Blank, Diane Robins, Angelo DeMarzo, John Isaacs, and Shuk-mei Ho	Centennial III-IV
9:30–9:45 a.m.		*Break*	
11:00 a.m.–12:15 p.m.	28	SPOTLIGHT SYMPOSIUM: The Prostate Cancer Clinical Consortium: Translation of Scientific Discovery to the Clinic Speakers: Roland Young and Howard Scher	Centennial III-IV
12:30–2:30 p.m.	29	POSTER SESSION/LUNCH P18 Nutrition and Prostate Cancer P19 Lifestyle P20 Complementary and Alternative Medicine P21 Chemoprevention P22 Risk P23 Angiogenesis P24 Apoptosis P25 Cancer Stem Cells P26 Biomarkers II P27 Signaling II P28 Androgen Receptor P29 Hormone Refractory Prostate Cancer P30 Imaging P31 Immunotherapy P32 Targeted Therapy P33 Clinical Trials P34 Summer Training Program	
2:45–4:15 p.m.	30	PLENARY SESSION: CLINICAL RESEARCH Moderator: Christopher Logothetis Speakers: Robert Carey, Stephen Freedland, Laurence Klotz, Fritz Schröder, and Christopher Warlick	Centennial III-IV
4:15–4:30 p.m.		*Break*	
4:30–6:00 p.m.		SYMPOSIA SESSIONS III	
	31	Prevention, Screening, and Early Detection Consumer Speaker: Willie Kimmons Moderator: Sherri Sheinfeld Gorin *Predictors of Informed Decision Making in Prostate Cancer Screening* Jeff Riggio *A Behavioral Model of Prostate Cancer Screening for African American Men* Folakemi T. Odedina	Centennial II

AGENDA — Friday

4:30–6:00 p.m.

SYMPOSIA SESSIONS III, Prevention, Screening, and Early Detection (cont.) — Centennial II

Preferences for Prostate Cancer Screening and Treatment: Assessment of Message Framing and Theoretical Underpinnings
Deborah Watkins-Bruner

Systematic Development and Testing of a Web-based Intervention to Primary Care Physicians in African American Communities
Sherri N. Sheinfeld Gorin

Nanoparticle-conjugated Biomarkers for Early Detection of Prostate Cancer in African-American Men
Catherine M. Phelan

32 Developing Immunotherapy for Prostate Cancer — Regency V
Consumer Speaker: Phil Olsen Moderator: Timothy Ratliff

Genetically Engineered T Cells for Adoptive Immunotherapy of Prostate Cancer
Zelig Eshhar

T Cell Responses to Prostate Cancer Antigens
Arthur A. Hurwitz

Dendritic Cell Based Strategies to Treat Cancer of the Prostate: Towards Development of Cancer Vaccine
Prabir K. Chakravarty

Combining Radiation Therapy with Interstitial Radiation-inducible Tumor Necrosis Factor Alpha Expression for Local Regional Cancer Treatment
Mira O. Jung

Defining Novel Molecules to Rescue Immunity against Prostate Cancer: Molecular and Biological Bases for New Therapies
Antonella Viola

Phase I Study of a DNA Vaccine Encoding Prostatic Acid Phosphatase (PAP) in Patients with Non-castrate Non-metastatic Prostate Cancer
Douglas G. McNeel

33 Tumor Suppressors — Learning Center
Consumer Speaker: Richard Gillespie Moderator: Cory Abate-Shen

P57Kip2 Is Downregulated in Human Prostate Cancer and Downregulation Induces Tumorigenesis in Mouse Prostate
Ren J. Jin

Downregulation of Beta1 Integrin in Vivo Delays Prostate Cancer Progression and Increases Radiosensitivity
Hira lal Goel

The FGFR-4 Arg388 Polymorphic Variant Is Associated with Increased Risk of Prostate Cancer and Aggressive Disease
Michael M. Ittmann

Developing a Novel Preclinical Mouse Model for Androgen-independent Prostate Cancer
Yurong Song

Inflammatory Cytokines Induce Ubiquitination and Loss of Prostate Suppressor Protein NKX3.1
Edward Gelmann

Targeting the Cytoprotective Chaperone Protein Clusterin to Enhance Apoptosis in Patients with Prostate Cancer
Kim N. Chi

34 Targeting Apoptosis — Regency VI

Consumer Speaker: Westley Sholes Moderator: Natasha Kyprianou

Increased Expression of Cyclin B1 Sensitizes Prostate Cancer Cells to Apoptosis Induced by Chemotherapy
Carlos Perez-Stable

Testing the Apoptotic Effect of the Potent Pro-apoptotic Small Molecule Smac Mimetics on Prostate Cancer Cell Lines
Chunying Du

The Proapoptotic STE20-like Kinase MST1 Is a Direct Inhibitor of AKT1
Bekir Cinar

Molecular Radiosensitization of Prostate Cancer by Targeting Apoptotic Pathways
Liang Xu

Conversion of Vascular Endothelial Growth Factor into a Death Factor
Timothy P. Quinn

35 Metastasis — Centennial III-IV

Consumer Speaker: Justin Sucato Moderator: Robert Sikes

Membrane Type 1-Matrix Metalloproteinase (MT1-MMP) Induces Epithelial-to-Mesenchymal Transition (EMT) in Less Aggressive Epithelial Cancer Cells
Jian Cao

Prostate Cancer-associated Membrane Type-1 Matrix Metalloproteinase: A Pivotal Role in Bone Response and Intraosseous Tumor Growth
Michael L. Cher

The Adaptor Protein and SRC Substrate AFAP-110 Regulates Prostate Tumor Cell Growth through Focal Adhesion Function
Gary E. Gallick

TGF-Beta Increases Pro-osteolytic Gene Expression and Promotes Bone Metastases from Prostate Cancer
Theresa A. Guise

The Role of the Endothelin-Axis in Androgen Ablation and Progression to Advanced Prostate Cancer
Jason M. D'Antonio

36 Multicenter Collaborations for Clinical Trials — Regency VII

Consumer Speaker: James Kiefert Moderator: Maha Hussain

NCI 7347: Phase I/II Trial of Epothilone Analog BMS-247550 (Ixabepilone), Mitoxantrone, and Prednisone in Hormone Refractory Prostate Cancer Patients Previously Treated with Chemotherapy: Results of a DOD Prostate Cancer Therapy Consortium Trial
Jonathan E. Rosenberg

EMD121974 (NSC 707544, Cilengitide) in Asymptomatic Metastatic Androgen Independent Prostate Cancer (AIPCA) Patients (Pts): A Randomized Trial by the Prostate Cancer Clinical Trials Consortium (NCI 6372)
Maha Hussain

An Open-label, Multicenter, Phase II Study of Single-Agent at-101 in Men with Hormone Refractory Prostate Cancer (HRPC) and Rising Prostate Specific Antigen (PSA) Levels Who Have Not Received Prior Chemotherapy
Glenn Liu

Phase I Study of Sunitinib in Combination with Docetaxel and Prednisone in Metastatic Castrate-resistant Prostate Cancer (CRPC)
Amado J. Zurita

7:00–9:00 p.m.		DINNER RECEPTION Spelman Glee Club Hal Ackerman Presents a One-man Play: "Testosterone: How Prostate Cancer Made a Man of Me"	Centennial I

Saturday, September 8, 2007

Time	Session	Event	Room
7:00 a.m.–12:00 p.m.		Registration	
7:00–8:00 a.m.		EARLY MORNING EDUCATIONAL SESSIONS	
	37	Genetic Epidemiology Speakers: Virgil Simons, Jianfeng Xu, and Henrik Grönberg	Centennial II
	38	Complementary and Alternative Medicine Speakers: James McGuinness, Wendy Demark-Wahnefried, and Peter Nelson	Regency V
	39	Hormone Refractory Prostate Cancer Speakers: William Bright, Donald Tindall, and Edward Gelmann	Regency VI
	40	Treatment and Management of Prostate Cancer Speakers: Winston Dyer, Daniel Petrylak, Curtis Pettaway, and Richard Valicenti	Regency VII
8:15–9:30 a.m.		WELCOME AND MOMENT OF SILENCE	
	41	SPOTLIGHT SYMPOSIUM: Manhattan Project for Targeting the Lethal Phenotypes of Prostate Cancer Speakers: John Willey and Jonathan Simons	Centennial III-IV
9:30–9:45 a.m.		*Break*	
10:00–11:00 a.m.	42	PLENARY SESSION: QUALITY OF LIFE Speakers: Richard and Desiree Howe, Sara Knight, and David Latini	Centennial III-IV
11:15 a.m.–12:45 p.m.		SYMPOSIA SESSIONS IV	
	43	Treatment, QOL, and Other Health Outcomes Consumer Speaker: Alan Simpson Moderator: Folakemi Odedina	Centennial II
		Barriers to Measuring Prostate Cancer (CaP) Quality of Life (QOL) in Underserved Patients Kerry L. Kilbridge	
		Race, Health Insurance and Radical Prostatectomy: Preliminary Data from the North Carolina–Louisiana Prostate Cancer Project (PCaP) Jane C. Schroeder	
		Decreased Cerebral Metabolism from Androgen Suppression Monique M. Cherrier	
		Erbium:YAG Laser Incision of Urethral Strictures after Prostate Cancer Surgery Nathaniel M. Fried	
		Reconstructing Masculine Identity within the Context of Prostate Cancer Treatment-related Symptoms by Low-Income Men Sally L. Maliski	
		The Effect of Hospital and Physician Volume on Racial Differences in Disease Recurrence Following Surgery for Prostate Cancer Louie Ross	

44	**Signal Transduction II** Consumer Speaker: James Williams, Jr. Moderator: James Lillard *Analysis of the Role of the Wnt/Beta-Catenin Pathway in Prostate Development and Tumorigenesis* Bart O. Williams *SIRT1 and Prohibitin Are Recruited to Androgen Response Elements and Are Required for Androgen Antagonist-induced Transcriptional Repression of Androgen-responsive Genes and Cell Growth* Douglas V. Faller *Transcription Factor STAT5 in Progression of Prostate Cancer to Advanced Disease and as a Therapeutic Target Protein for Prostate Cancer* Marja T. Nevalainen *Distinct Role of Annexin A1 in Angiogenesis and Tumor Development–Novel Target for Vascular Immunotargeting of Prostate Tumor* Ming Yi *Targeting Legumain:Integrin Complex in the CaP Microenvironment* Cheng Liu *Investigation of the TCF/LEF Pathway in Human Prostate Cancer Cells* Luca Grumolato	Regency V
45	**Tumor Microenvironment** Consumer Speaker: Manuel Vazquez Moderator: Simon Hayward *A Novel Mechanism of Prostate Cancer Progression Mediated by Alphavbeta6 Integrin* Jing Li *Bone Microenvironment and Androgen Status Modulate Cellular Localization of ERBB3 in Prostate Cancer Cells* Sue Hwa Lin *Hyaluronan/Tumor Cell Interactions in Prostate Cancer Progression* Jennifer H. Carlson *Reactive Stroma as a Mediator of Prostate Cancer Progression* David R. Rowley *Stromal TGF-Beta Signaling Promotes Prostate Cancer* Mingfang Ao	Regency VI
46	**Etiology and Novel Biomarkers** Consumer Speaker: Raul Blasini Moderator: Donald Miller *Biomarkers of Human Prostate Cancer Risk and Prevention* Ercole L. Cavalieri *YAL1 Hyaluronidase: A Prognostic Marker and a Therapeutic Target in Prostate Cancer* Vinata B. Lokeshwar *Evaluating XMRV as an Indicator of Prostate Cancer Risk or Progression* Robert H. Silverman *Small Integrin Binding Proteins as Serum Markers for Prostate Cancer Detection* Alka Jain *Role of Mitochondrial Glycerophosphate Dehydrogenase in Prostate Cancer* Subir Kumar Roy Chowdhury *Novel Protein Microarray Technology to Examine Men with Prostate Cancer* Hans G. Lilja	Centennial III-IV

AGENDA — Saturday

11:15 a.m.–12:45 p.m.

SYMPOSIA SESSIONS IV (cont.)

47 **Androgen Receptor II** — Regency VII

Consumer Speaker: Greg Bielawski Moderator: Donald Tindall

Activated CDC42-associated Kinase Ack1 Promotes Androgen-independent Prostate Cancer Progression via Phosphorylation of Tyr-267 and Tyr-363 Residues of Androgen Receptor
Young E. Whang

Role of Coactivators in Ligand Dependent and Independent Androgen Receptor Action
Irina U. Agoulnik

Determinants of Response to Type 1 Insulin-like Growth Factor Receptor (IGF-IR) Inhibition in Prostate Cancer
Stephen R. Plymate

Novel Murine Prostate Cancer Cell Lines Demonstrate Critical Role of PTEN in Hormone Refractory Prostate Cancer Development
Jing Jiao

Prostate Cancer Cell Proliferation Involves Cell Cycle-dependent Interaction of Androgen Receptor with the Enzymes of DNA Synthesis
Prem Veer G. Reddy

48 **Collaborative Partnership Panel** — Learning Center

Consumer Speaker: Quince Fleming, Jr. Moderator: Shafiq Khan

12:45–2:45 p.m.

Luncheons (Trainees; HBCU)

SPEAKER ABSTRACTS

THERAPEUTICS ADVANCES

The Survivor's Perspective: Therapeutics Advances

Charles Rubin

The Prostate Centre at
Southern Arizona Prostate Cancer Support Group

Progress made in conquering prostate cancer, especially when it has become resistant to hormone therapy, is important to the prostate cancer survivor. While many prostate cancers start as hormone-sensitive tumors, most advanced hormone-sensitive cancers eventually become resistant to hormone treatment and find ways to thrive without hormones. The next line of therapy available then is chemotherapy. The development of new enhancements for standard chemotherapies that target specific pathways or genes involved in resistance to chemotherapy will result in the survival of many more men from this disease.

Improving Chemo and Hormonal Therapies by Targeting Stress Induced Cytoprotective Chaperone Genes

Martin Gleave

The Prostate Centre at Vancouver General Hospital

While advanced prostate cancer responds initially to androgen ablation, tumors recur because surviving cells acquire an androgen-independent (AI) phenotype. This complex process involves adaptive upregulation of stress-induced survival genes and androgen receptor (AR) transactivation in the absence of androgen from mutations or increased levels of co-activators and alternative growth factor pathways, including Her2/neu, EGFR, and IGF-1, leading to dysregulated AR pathways. Improved understanding of specific mechanisms mediating AI progression and new therapeutic strategies designed to inhibit the emergence of this phenotype are needed before additional gains in survival can be realized. *Of special relevance to development of AI progression and HRPC are those survival proteins upregulated after apoptotic triggers like androgen ablation that function to inhibit cell death.* Proteins fulfilling these criteria include antiapoptotic members of the Bcl-2 protein family, clusterin, Hsp27.

i. Clusterin. Clusterin is a cytoprotective chaperone associated with numerous tumors including prostate, breast, lung, and renal cell carcinoma. In human prostate cancer, clusterin levels are low in most untreated hormone-naive tissues but increase significantly within weeks after neoadjuvant hormone therapy. Because clusterin binds to a wide variety of biological ligands and is regulated by transcription factor HSF1 (heat shock factor 1), an emerging view suggests that clusterin functions like a heat shock protein to chaperone and stabilize conformations of proteins at time of cell stress. Experimental and clinical studies associate clusterin with the development of hormone and drug resistance, where clusterin inhibits apoptotic cell death from androgen withdrawal, chemotherapy, and radiation. The second generation antisense drug, OGX-011, decreases cytoprotective sCLU while increasing pro-apoptotic alternative spliced nuclear (nCLU), levels to enhance hormone- and chemo-therapy in many preclinical xenograft models. Such MOE "gap-mer" 2nd generation modifications improve tissue pharmacokinetic profile of ASO. In preclinical models, OGX-011 improves the efficacy of chemotherapy, radiation, and androgen withdrawal by inhibiting expression of clusterin and enhancing the apoptotic response. OGX-011 recently completed two Phase I trials given weekly as a single agent or in combination with docetaxel. The single agent study has a unique design in that patients with localized prostate cancer are treated with the OGX-011 prior to radical prostatectomy. This allows for an assessment of *clusterin* expression and tissue concentrations in prostate tumors from all patients and will permit dose-dependent correlations to be made, allowing for determination of an optimal biologically effective dose and tissue drug levels in addition to the usual parameters of toxicity. Twenty-five patients were enrolled to 6 cohorts with doses of OGX-011 up to 640 mg delivered in combination with LHRH analogue. Toxicity was limited to grade 1 or 2, including fevers, rigors, fatigue, and transient AST and ALT elevations. Prostate tissue concentrations of OGX-011 increased with dose, and tissue concentrations associated with preclinical effect could be achieved. Dose-dependent decreases in prostate cancer cell *clusterin* expression were observed. At 640 mg dosing, *clusterin* mRNA was decreased to a mean of 8% compared with lower dose levels and historical controls as assessed by reverse transcription PCR of microdissected cancer cells. By immunohistochemistry, mean % cancer cells staining with zero intensity for *clusterin* protein at 640 mg dosing was 54% compared with 2%–15% for lower dose levels and historical controls. This Phase I trial demonstrates that OGX-011 is well tolerated and inhibits *clusterin* expression in prostate cancers and confirmed that 640 mg is the Phase II dose based on pk and target regulation data. Phase I combination studies confirmed that 640 mg OGX-011 can be combined with standard doses of the chemotherapy agents. The current status of several Phase II trials in prostate, lung, and breast cancer, including a randomized Phase II trial of 1st and 2nd line chemotherapy +/- OGX-011 in mHRPC will be presented.

ii. Heat Shock Protein 27. Using array analysis to compare gene expression profiles before and after castration, we recently identified Heat Shock Protein 27 (Hsp27) as one of the most highly expressed genes in AI prostate tumors. Hsp27 is a 27-kDa protein highly induced during the stress response to a wide variety of physiological and environmental insults. Various roles have been proposed for Hsp27 to explain its cytoprotective effects during cellular stress, including its role as a molecular chaperone, direct interference with caspase activation, modulation of oxidative stress and regulation of the

cytoskeleton. Higher levels of Hsp27 are commonly detected in various cancers including breast, ovarian and endometrial, and prostate.

Hsp27 expression was low or absent in untreated human prostate cancers but increased beginning 4 weeks after androgen-ablation to become uniformly highly expressed in AI tumors. Forced over-expression of Hsp27 in LNCaP cells suppresses castration-induced apoptosis and confers androgen-resistance while Hsp27 ASO and siRNA potently inhibit Hsp27 expression, increased apoptosis, and decreased PC3 and LNCaP cell growth. Hsp27 ASO also enhanced paclitaxel chemosensitivity in vitro and in vivo. These findings suggest that increased levels of Hsp27 after androgen withdrawal provide a cytoprotective role during development of androgen independence and that ASO-induced silencing can enhance apoptosis and delay tumor progression. A 2nd generation MOE-gapmer ASO targeting Hsp27 has been developed (OGX-427, OncoGenex Technologies Inc.) and will enter clinical trials in 2007.

Selected References

Chi KN, Eisenhauer E, Fazli, L, Jones EC, Goldenberg SL, Powers J, Tu DS, Gleave ME; A Phase I Pharmacodynamic Study of OGX-011, a 2'-Methoxyethyl Antisense Oligonucleotide to Clusterin, in Patients With Localized Prostate Cancer. *J Nat.Canc.Inst.* Vol 97, No. 17, 2005.

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Miyake H, Rennie P, Nelson C, Gleave ME. Acquisition of chemoresistant phenotype by overexpression of the antiapoptotic gene, testosterone-repressed prostate message-2 (TRPM-2), in prostate cancer xenograft models. *Cancer Res* 60:2547-54, 2000.

Rocchi P, So A, Kojima S. Beraldi E, Fazli L and Gleave ME. Heat shock protein 27 increases after androgen ablation and plays a cytoprotective role in hormone refractory prostate cancer. *Cancer Research*, 2004 Sep 15;64(18):6595-602.

Rocchi P, Beraldi E, Ettinger S, Fazli L, Vessella RL, Nelson C, Gleave M. Increased Hsp27 after androgen ablation facilitates androgen-independent progression in prostate cancer via signal transducers and activators of transcription 3-mediated suppression of apoptosis. *Cancer Res*. 2005 Dec 1;65(23):11083-93.

Pathologic, Molecular, and Genomic Evaluation of mTOR Inhibition in Metastatic Hormone Refractory Prostate Cancer

Daniel George

Duke University Medical Center

THE BUSINESS OF MEDICINE

The Business of Medicine

Westley Sholes

California Prostate Cancer Coalition

Medicine is big business in the United States. Total health care expenditures in 2004 was $1.9 trillion, representing 16% of the Gross Domestic Product, and is expected to reach 20% in the next decade.

The process of determining, financing, and executing health policy in the United States is very complicated, and involves many stakeholder groups. Three of the key stakeholders groups are researchers, clinicians, and patient consumer/advocates.

As a 9-year prostate cancer survivor and having spent 8 years volunteering in several advocacy capacities, I have made several observations on the interactions between researchers/clinicians and advocates. This discussion will include observations from my advocacy perspective.

Translational Research Grant Opportunities for Business

Monica Liebert

University of Michigan Medical School

Effective translation of research from bench to bedside should involve product and business development to allow for sustained and continued utilization. The federal government encourages this activity through the Small Business Innovation Development Act of 1982, which has been reauthorized several times, most recently by P.L. 106-554, through September 30, 2008. This law requires that federal agencies with research and development budgets over $100 million must set aside 2.5% of funds for grants to small businesses (under 500 employees) for innovative research with commercialization potential and public benefit. To be eligible, the small business entity must also be 51% owned and controlled by U.S. citizens. Two types of grants are available: the SBIR, Small Business Innovative Research grant, and STTR, the Small Business Technology Transfer Act. The application and review processes for these grants will be discussed, along with other helpful information from the NIH. Recent new developments, including provisions for multiple Principal Investigators and electronic application, will be reviewed.

The Business of Prostate Cancer Care: A Clinician/Researcher's Perspective

David F. Penson

Keck School of Medicine, University of Southern California

Prostate cancer care is expensive. In 2001, Medicare alone spent over a billion dollars on health care related to prostate

cancer. This is in addition to indirect costs incurred by patients, including time away from work for patients and care givers, loss of productivity, and early retirement. Clearly, prostate cancer is big business in the United States. Sadly, while the majority of prostate cancer care is driven by good intentions and science, there is clearly an entrepreneurial element. In this presentation, we will discuss the pharmacoeconomics of prostate cancer care, including recent changes in the use of androgen deprivation therapy in response to changes in Medicare reimbursement, the emergence of robotic surgery, and off-label use of various pharmaceuticals purported to prevent or treat prostate cancer. By reviewing these topics, the audience will garner a better understanding of the various economic forces that shape prostate cancer care and, more importantly, will recognize that many contemporary trends in prostate cancer care are driven as much by the bottom line as they are by good science. This underscores the pressing need for well-designed randomized clinical trials in this common malignancy.

IMMUNOTHERAPY TRIALS

Immunotherapy: A Patient Perspective

James Kiefert

Us TOO International, Inc.

Combination Immunotherapy for Prostate Cancer

Charles G. Drake

Johns Hopkins Sidney Kimmel Comprehensive Cancer Center

Immunotherapy for prostate cancer has reached the point of large, randomized Phase III trials, aimed at improving the survival of men with metastatic, hormone-refractory disease. However, tumors evolve multiple mechanisms to escape immune attack as they progress within an individual patient. To most effectively treat men with prostate cancer, treatment approaches that combine immunotherapy with other treatment modalities will most likely prove necessary. Immunotherapy can be combined with conventional chemotherapy or hormonal therapy, but the most innovative strategies combine active immunotherapy ("vaccination"), with agents that block the biological checkpoints that inhibit a productive immune response. Clinical trials testing these ideas will be introduced and discussed, and the relative risks and benefits of these strategies outlined.

Modulating the Immune Response to Fight Prostate Cancer – How to Maximize a Minimal Outcome

Susan F. Slovin

Memorial Sloan-Kettering Cancer Center

There has been a resurgence of interest in developing noncytotoxic immune therapies for patients with either hormone-naïve biochemically relapsed post-primary therapy or castrate metastatic prostate cancer. These therapies would theoretically make use of the patient's immune system to fight the tumor, particularly if their disease is of reasonably low volume. Many immunotherapeutic approaches have been based on the over-expression and underglycosylation of a wide variety of altered "self" glycolipid and glycoprotein molecules on the tumor cell surface, among which are prostate specific antigen (PSA), acid phosphatase (ACP), prostate stem cell antigen (PSCA), and prostate specific membrane antigen (PSMA), which can serve as targets for immune recognition and attack.

A variety of Phase I, II, and III trials have demonstrated the safety and potential efficacy of immunotherapeutic approaches and have shown that immunologic tolerance could be successfully broken, as evidenced by the development of high antibody titers and T cell responses specific for the tumor. Multiple strategies have been used in these trials to potentiate immune reactivity in vivo and increase antitumor responses. No approach to date has been successful in demonstrating the best way of maximizing immune responsiveness. However, all of these approaches have shown that immunologic tolerance can be successfully broken and that immune responses can occur.

Several limitations to immunotherapeutic approaches were observed in the trials. While vigorous antibody responses can be generated with the immunotherapies, there were little or no antitumor responses in patients with high volume disease, nor have criteria been established that allow better definition as to what should be considered to be a response to the cancer. Another limitation to these approaches is that there has been no easy way to potentiate and quantitate T cell immunity, which is thought to be critical to enhancing and assessing antitumor responsiveness, respectively. There also is no definitive way to quantitate a clinical response to vaccine therapy in patients who have biochemically relapsed following definitive primary therapy such as surgery or radiation. Finally, it remains unclear which antigen(s) is/are the "right target(s)" and which patient population would benefit from these approaches.

This presentation will discuss the strategies currently being used to maximize minimal immunotherapeutic responses to early and late relapsed prostate cancer, including the use of CTLA-4 blockade, using gene transfer of cytokines into tumor cells to enhance tumor cell immunogenicity and manipulating autologous dendritic cells expressing specific tumor peptides. The methods used to assess antitumor effect by the immunotherapeutic approaches also will be discussed.

There is renewed enthusiasm for immune therapies for the treatment of early and late relapsed prostate cancer. The current trends favor the use of multiple immune strategies that are leading not only to our better understanding as to how the immune system functions, but also how it responds to new drug challenges.

PROSTATE CANCER 101

The Patient Perspective

Larry Junker

Us TOO International, Inc.

Prostate Cancer 101

Kenneth J. Pienta

University of Michigan

HEALTH DISPARITIES

Keynote on Health Disparities

James E. Williams, Jr.

Intercultural Cancer Council, Pennsylvania Prostate Cancer Coalition, Alliance for Prostate Cancer Prevention

Cancer health disparities are differences in the incidence, prevalence, mortality, and burden of cancer and related adverse health conditions that exist among specific population groups in the United States. These population groups may be characterized by gender, age, ethnicity, education, income, social class, disability, geographic location, or sexual orientation.[1]

An estimated 30,870 cases of prostate cancer are expected to occur among African American men in 2007, accounting for 37% of all cancers diagnosed in African American men. Between 2000–2003, the average annual prostate cancer incidence rate was 60% higher in African American men than in white men.[2]

Prostate cancer is the second leading cause of cancer death in African American men. It is estimated that 4,240 deaths from prostate cancer will occur in African American men in 2007. African American men have the highest mortality rate for prostate cancer of any racial or ethnic group in the United States. The death rate for prostate cancer is 2.4 times higher in African American men than white men.[3]

The Minority and Underserved Populations Program (MIU Program), originally titled Special Populations Program was established by the Office of the Congressionally Directed Medical Research Programs (CDMRP) to address the significant disparities that exist in the incidence, morbidity, and/or mortality among different ethnic groups in many of the diseases for which the CDMRP provides support.

The CDMRP's efforts to implement the IOM and Minority Health Initiative recommendations have been very effective as evidenced by increases in (1) minority consumers and scientists participating in peer review; (2) the number of proposals received and funded from HBCU/MI; and (3) the number of HBCU/MI and population-specific funding mechanisms advertised and funded. For instance, minority scientist participation in peer review has increased since the establishment of the MIU Program. Minority consumer participation has also increased since the establishment of the MIU Program. Additionally, the percentage of total funds available for research that was spent on proposals from HBCU/MI has consistently surpassed the Department of Army set-aside goal of 5.0%.

A sample supported program can be found at Charles R. Drew University of Medicine and Science in Los Angeles, California (Drew). Researchers at Drew are exploring innovative ways to enhance awareness of prostate cancer research among African Americans by using minority prostate cancer survivors as health educators. Research began by recruiting and training the health educators. Community culturally sensitive educational sessions were developed and then provided by the health educators to the south central Los Angeles African American community. Based on an evaluation of the participants' interest in prostate cancer prevention, the effectiveness of health educators was determined to be positive. The project has the potential to develop more effective prostate cancer research recruitment and educational strategies for African Americans.

The focus of CDMRP to recognize the value of HBCU/MI, support building of infrastructure so good scientific research can be conducted, and further support initiatives that build a pool of minority researchers and consumer participation has led other governmental agencies in addressing health disparities.

References

1. National Cancer Institute, Division of Cancer Control and Population Sciences
2. American Cancer Society, Cancer Facts & Figures for African Americans 2007-2008
3. American Cancer Society, Cancer Facts & Figures for African Americans 2007-2008

The Meaning of Race in Science and Society

Harold Freeman

Ralph Lauren Center for Cancer Care and Prevention

Health Disparity and Prostate Cancer: What Do We Know about Black–White Differences?

Mack Roach III

University of California, San Francisco

African American men experience an excess incidence and mortality from prostate cancer when compared to other American men. Based on the data published by the Radiation Therapy Oncology Group (RTOG) and the preponderance of evidence from other randomized trials, there does not appear to be good evidence to support stratifying men by race or offering different treatment because of race. The RTOG data do not support important biologic differences in the host attributable

to race, such as enhanced to sensitivity to the androgen receptor (e.g., CYP3A4). Thus the participation of underserved populations in prospective randomized trials appears to be the most straightforward strategy for eliminating or reducing health discrepancies. Alternative explanations for the differences in outcome reported elsewhere in the literature include: (1) differences in the extent of disease at diagnosis, (2) poorer access to care, and (3) poor quality care. Thus, research that addresses multifactorial determinants of outcome is required to explain the observed differences in prostate cancer incidence and mortality. However, it is important that these studies should look further beyond the relatively simplistic concept of "race" to identify additional factors that might explain the differences in outcome. For example, recent studies have validated the notion that chronic stress can have important biologic interactions with tumor behavior, which may have implications for the impact of racism on prostate cancer-related outcomes.

SPOTLIGHT SYMPOSIUM: RACIAL DIFFERENCES IN PROSTATE CANCER: THE NORTH CAROLINA–LOUISIANA PROSTATE CANCER PROJECT (PCAP)

Health Disparities of Prostate Cancer: The Survivor's Viewpoint

James E. Williams, Jr.

Intercultural Cancer Council, Pennsylvania Prostate Cancer Coalition, Alliance for Prostate Cancer Prevention

North Carolina–Louisiana Prostate Cancer Project

James Mohler

Roswell Park Cancer Institute

Background: The North Carolina-Louisiana Prostate Cancer Project (PCaP) is a multidisciplinary study of social, individual, and tumor-level causes of racial differences in prostate cancer aggressiveness.

Methods: A population-based sample of incident prostate cancer cases from North Carolina and Louisiana will include 1,000 African Americans and 1,000 Caucasian Americans. Study nurses administer structured questionnaires and collect blood, adipose tissue, urine, and toenail samples during an in-home visit. Clinical data are abstracted from medical records, diagnostic biopsies are reviewed and assayed, and tissue microarrays are constructed from prostatectomy samples. Prostate cancer aggressiveness is classified clinically based on PSA, clinical stage, and Gleason grade and objectively based on tumor growth rate calculated from cell proliferation rate using Ki-67 and apoptosis using ACINUS in immunostained and image analyzed diagnostic prostate biopsies.

Results: As of March 1, 2007, in-home visits have been completed for 768 men in North Carolina and 216 men prior to Hurricane Katrina and 171 men since enrollment resumed September 1, 2006 in Louisiana. Participation exceeds 70% in all groups. Preliminary data analysis demonstrated between- and within-group differences in patient characteristics, use of early detection and treatment by race and state. Prostate cancer aggressiveness has been classified in 667 men as high in 20%, intermediate in 30% and low in 50%. Clinical aggressiveness correlated (means procedure) with prostate cancer growth rate (n =72).

Conclusions: Preliminary data support the feasibility of this comprehensive study to help determine the focus of public health efforts to reduce racial disparities in prostate cancer mortality. (Schroeder JC, Bensen JT, Su LJ, Mishel M, Ivanova A, Smith GJ, Godley PA, Fontham ET, Mohler JL. The North Carolina-Louisiana Prostate Cancer Project (PCaP): Methods and design of a multidisciplinary population-based cohort study of racial differences in prostate cancer outcomes. *Prostate* 2006; 66(11):1162–1176)

TRANSLATIONAL RESEARCH

Translational Research: The Survivor's Viewpoint

Gregory A. Bolden

Metro Atlanta Coalitions for Cancer Awareness

Translational research "translates" basic science discoveries into clinical applications and also uses clinical observations to generate research questions for basic science studies for true "bench-to-bedside" and back again discoveries. Successful translational research relies on communication (part of the translation) between basic science researchers, clinicians, and communities to find the discoveries that can be tested in clinical trials. The researchers and clinicians developing these new targets for therapy need to have active, functional partnerships with academic institutions, government funding agencies, and the biopharmaceutical industry. The community plays an important role in selective recruitment of population-based participants that ensure a varied range of study participants leading to reduced adverse pharmacological interactions in bedside interventions. This type of collaboration could bring new drugs into clinical trials more quickly to benefit the two million men currently battling prostate cancer.

Recurrent Gene Fusions in Prostate Cancer

Arul M. Chinnaiyan

University of Michigan

To date, the great majority of disease-specific, recurrent chromosomal rearrangements have been characterized in hematological malignancies and mesenchymal tumors and not in common epithelial tumors such as breast, lung, colon, or prostate cancer. Here, we employed a bioinformatics approach on a compendium of cancer gene expression data to discover candidate oncogenic chromosomal aberrations based on outlier gene expression. In addition to identifying many gene partners of characteristic rearrangements in human malignancies, this approach identified two members of the ETS family of

transcription factors, *ERG* and *ETV1*, as outliers in prostate cancer. Either *ERG* or *ETV1* was over-expressed in the majority of prostate cancers (50%–70%) and were mutually exclusive across several independent gene expression datasets, suggesting that they may be functionally redundant in prostate cancer development.

By RNA ligase-mediated rapid amplification of cDNA ends (RACE), we identified a recurring gene fusion of the 5' untranslated region of a prostate-specific, androgen-regulated gene *TMPRSS2* to *ERG* or *ETV1* in prostate cancer cases that overexpressed the respective ETS family member. These gene fusions were confirmed using quantitative PCR (QPCR) and sequencing of reverse transcription PCR products. In addition, using fluorescence in situ hybridization (FISH), we demonstrated that 23 of 29 (79%) prostate cancer samples harbor rearrangements in *ERG* or *ETV1*. Furthermore, in vitro cell line studies suggest that the androgen-responsive promoter elements of *TMPRSS2* mediate the aberrant over-expression of *ETS* family members in prostate cancer. Subsequently, we interrogated the expression of all *ETS* family members in prostate cancer profiling studies and identified outlier expression of ETV4 in two of 98 cases. In one such case, we confirmed the over-expression of *ETV4*, and by RACE, QPCR and FISH, we identified fusion of the *TMPRSS2* and *ETV4* loci.

Together, these results suggest a pathogenetically important role for recurrent chromosomal rearrangements in common epithelial tumors and have implications in the molecular diagnosis and treatment of prostate cancer. Importantly, these results identify three molecular subtypes of prostate cancer, *TMPRSS2:ERG*, *TMPRSS2:ETV1*, and *TMPRSS2:ETV4*, and suggest that dysregulation of *ETS* family member expression through gene fusions with *TMPRSS2* may be a generalized mechanism for prostate cancer development.

Immune Checkpoint Blockade in Cancer Therapy

James P. Allison

Howard Hughes Medical Institute
Memorial Sloan-Kettering Cancer Center

While there are exciting examples of success clinical strategies to mobilize the immune system to attack cancer cells, overall the results have not met the promise hoped for in tumor immunotherapy. One reason for less than optimal results is that until recently there was insufficient knowledge of the complex regulatory pathways employed by the immune system to avoid autoimmunity, and therefore insufficient attention has been paid to strategies for avoiding the negative impact of these mechanisms on the effectiveness of immunotherapies. It has become quite clear over the past several years that while T cell responses are initiated by engagement of the antigen receptor, they are shaped by additional signals that act in concert to shape the magnitude, quality, and location of the response to maximize target destruction and minimize harm to normal tissues. The prototype of these regulatory circuits was the CD28/CTLA-4 axis, which regulates early stages of the T cell response. CD28 provides critical costimulatory signals necessary for activation of naïve T cells, while CTLA-4 limits proliferation of the responding T cells. Both CD28 and CTLA-4 bind B7-1 and B7-2 in a complex and dynamic way that can shape the early T cell response.

Our work has provided some insight into the molecular mechanisms whereby CTLA-4 inhibits T cell proliferation in a cell intrinsic manner and can shape the emerging immune response by differential inhibition of individual clones based on the strength of TCR signaling. We have also shown that blockade of CTLA-4 can greatly enhance anti-tumor responses in a number of experimental tumors in mice. As a single agent anti-CTLA-4 can induce the rejection of tumors with inherently high immunogenicity, and in combination with appropriate vaccines can induce rejection of poorly immunogenic tumors. We have also shown that CTLA-4 blockade can synergize with a variety of conventional therapies, including chemotherapy and local radiation.

Anti-CTLA-4 (MDX-010, Ipilimumab) is being co-developed by Medarex, Inc. and Bristol Myers Squibb. Clinical trials have demonstrated objective responses in melanoma, renal, prostate, and ovarian cancer. MDX-010 is now in a pivotal Phase III clinical trial in melanoma.

In the last few years, the number of B7 family members has risen to seven. These fall into four groups and have distinct expression patterns and immunological functions. We recently identified B7x, a molecule that appears to expressed in epithelial tissues rather than by cells in the immune system. By interacting with an as yet unidentified receptor, B7x appears to be capable of inhibiting effector T cell function, including cytolysis. This suggests that B7x may play a role in protecting tissues against damage by aberrantly activated autoreactive T cells. It is of considerable interest that many mouse and human tumor cells express B7x. We are currently seeking to determine whether B7x might represent another checkpoint whose blockade would be of value in tumor immunotherapy.

Finally, recent studies have shown that tumors multiple coding mutations that should result in generation of multiple neoantigens. I will discuss the implications for this to immunologically based as well as conventional cancer therapy.

Quantum Dots: Emerging Nanotechnology for Targeted Prostate Cancer Imaging and Therapy

Shuming Nie

Emory University and Georgia Institute of Technology

Nanotechnology is an enabling technology with broad applications for prostate cancer imaging and treatment at the molecular level. The basic rationale is that nanometer-sized particles such as semiconductor quantum dots (QDs) have novel optical, electronic, magnetic, and structural properties that are not available from either individual molecules or bulk solids. When linked with biotargeting or biorecognition ligands such as monoclonal antibodies, peptides, or small

molecules, these nanoparticles can be used to target tumor antigens (biomarkers) as well as tumor vasculatures with high affinity and specificity. In the mesoscopic size range of 10–100 nm (diameter), quantum dots and polymeric nanoparticles also have more surface areas and functional groups that can be linked to multiple diagnostic (e.g., optical, radioisotopic, or magnetic) and therapeutic (e.g., anticancer) agents. In one example, we have recently developed a new class of self-assembled and biodegradable nanostructures for delivery and targeting of anticancer drugs and have achieved dramatically improved efficacy and reduced toxicities in in-vivo animal models. This type of interdisciplinary research opens new opportunities for biomarker-enabled detection, diagnosis, and individualized therapy of human prostate cancer as well as other malignant tumors.

Genomics and Personalized Medicine

John Carpten

Translational Genomics Research Institute

CHEMOPREVENTION

Chemoprevention – A Patient's Perspective

Richard E. Gillespie

Leader, Us TOO Chapter, Co-sponsored by the Westminster at Lake Ridge and Potomac Hospital

For too many men with advanced or virulent prostate cancer, the disease is seen as a death sentence. They remain distrustful of many treatments, especially chemotherapy treatment. There are reasons for optimism, however. First, men's immune systems are becoming more effective because of improved lifestyle and diet. There also are fewer cases of chemotherapy because of improved hormone therapy regimens reducing the need for chemotherapy. Now, new research into chemoprevention offers a way to war on prostate using treatments that prevent or delay the onset of prostate cancer. For younger men, this is a way to reduce the impact of prostate cancer on their lives.

Chemoprevention of Prostate Cancer

Eric Klein

Cleveland Clinic

Prostate cancer remains the most commonly diagnosed visceral cancer in men in the United States, with more than 230,000 newly diagnosed cases in 2006. Prevention of this disease would have a major impact on disease-associated cost, morbidity, and mortality for a large segment of the population. A major advance in prevention of prostate cancer came in 2003 with the publication of the Prostate Cancer Prevention Trial (PCPT). This overview summarizes the results of that trial, the design of other large-scale trials, and advances in understanding of the molecular mechanisms underlying the effect of other promising agents.

Recent Findings: The PCPT demonstrated that use of finasteride is associated with a 25% reduction in the 7-year period prevalence of prostate cancer in men over age 55 with normal digital rectal exam and initial PSA <3.0 ng/mL. Use of finasteride was associated with a slightly higher risk of Gleason sum 7–10 tumors, some sexual side effects, and fewer urinary symptoms. A substantial body of new molecular evidence supports the existing body of clinical and epidemiological data leading to testing of vitamin E and selenium as preventative agents in men at risk for prostate cancer. Epidemiologic and molecular evidence also makes COX-2 inhibitors, lycopene, soy, and green tea promising agents.

Summary: Results of a population-based, randomized Phase III trial demonstrates that finasteride can prevent prostate cancer. A large amount of data supports the use of other agents as potential preventatives, including selenium, vitamin E, vitamin D, other 5-α reductase inhibitors, COX-2 inhibitors, lycopene, and green tea. Some of these agents are being tested in new large-scale Phase III clinical trials.

Nutrition and Prostate Cancer: Macronutrients and Micronutrients

Michael Pollak

Jewish General Hospital—Lady Davis Institute

In the past two decades, there has been much interest in the influence of micronutrients such as lycopene, silibinin, and selinium on prostate cancer risk and prostate cancer prognosis. In this overview, we will review these data but also explore intriguing new clinical and laboratory observations suggesting important influences of total energy consumption on prostate cancer. We and others have observed that in population studies, hyperinsulinemia is associated with adverse outcome in men with prostate cancer and that this finding can be recapitulated in animal models. In these models, diet-induced hyperinsulinemia is associated with increased tumor growth, increased activation of insulin receptors on neoplastic cells, and increased activation of survival signaling pathways in prostate tumor tissue. These data may be relevant to the reports associating poor prognosis of prostate cancer with obesity, given the well-known association of obesity with hyperinsulinism. Confirmation of the obesity-hyperinsulinism-prostate cancer relationships would have important public health implications: The well-known obesity epidemic would be predicted to lead to increased prevalence of an adverse prognostic factor for prostate cancer in the population and thereby threaten to attenuate recent progress in prostate cancer control. Optimizing diet and lifestyle of men with early prostate cancer as well as in the general population might alleviate this threat. In addition, there is now justification for careful study of insulin-lowering pharmacological agents such as metformin, which may represent useful adjunct therapy for hyperinsulinemic prostate cancer patients.

LIFESTYLE ISSUES

Crossing the Rubicon (*) A Metaphor for Deliberately Proceeding Past a Point of No Return

Manuel Vasquez

Tex Us TOO

The patient is an essential part of his own medical team, and he should choose to be the CEO of this team. The patient's main duty in any disease is to arm himself with up-to-date information from reliable sources that allow the patient to give an informed consent for his treatment(s). In my case, this was the worse part of my road to recovery because all the responsibility for choosing treatment(s) was solely mine. The patient needs to find the best team and the best information to make these important decisions.

In addition to treatment with drugs from pharmaceutical companies, there are other ways a patient can be more proactive with his health. As a sequel to the chosen treatment(s), the impact of diet, exercise, and lifestyle on prostate cancer and other cancers is only now becoming understood. It is well known that men from Asia are less prone to prostate cancer until they move into the Western Hemisphere or adopt Western lifestyles. Although theoretically evident, the effects of diet, exercise, and lifestyle must be proven and replicated with the scientific method. Patients participating in this type of research could be part of exciting results that may impact the scope of cancer prevention and treatments to come.

Life Style Issues in Prostate Cancer Incidence and Progression

Meir Stampfer

Harvard School of Public Health

The huge variation in rates of prostate cancer incidence and mortality around the world, coupled with the relatively rapid changes in disease rates among migrant populations, strongly suggests that powerful nongenetic factors influence prostate cancer occurrence. Genetic factors clearly are important, but much of the variability in prostate cancer is likely due to life style factors. In addition, there is enormous variability in prognosis after diagnosis, with many men living free of any signs of illness even in the absence of therapy, some men having a gradually progressive course, and some men experiencing rapid progression despite aggressive treatment. This variability suggests the possibility that life style factors might influence prognosis after diagnosis.

In this presentation, I will briefly review the evidence for differences in diet, physical activity, adiposity, smoking and other life style factors may alter the risk of prostate cancer. In addition, I will discuss how for some factors, the impact may vary depending upon the genetic endowment of the individual. For most factors, the evidence remains inconclusive, though some appear quite promising, such as selenium and lycopene. I will also review the sparse data regarding the influence of life style factors on disease progression.

Risk Factors for Prostate Cancer in the Health Professionals Follow-up Study

Edward Giovannucci[1], Yan Liu[1], Elizabeth A. Platz[2], Meir J. Stampfer[1], and Walter C. Willett[1]

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The heterogeneous nature of prostate cancers, which range from relatively innocuous to highly aggressive in behavior, may contribute to inconsistent results in epidemiologic studies. Because they may act on different biologic pathways, risk factors may be different for various sub-groups of prostate cancer, such as for "aggressive" and "non-aggressive" cancers, defined by grade, stage, or survival. The premise, usually implicit, that risk factors for initiation of relatively innocuous, well-differentiated prostate cancers should be the same as those that cause death from prostate cancer has little theoretical or empirical basis. Further, although many epidemiologic studies now combine cancers of advanced stage at the time of diagnosis and those with high Gleason grade to characterize "aggressive" prostate cancer, this practice implicitly supposes that grade—which reflects degree of differentiation—carries the same meaning as advanced stage, but a risk factor could influence the progression of a cancer independently of an effect on tumor grade. Thus, results across studies could vary depending on the specific prostate cancer sub-type examined. Using data from the Health Professionals Follow-Up Study, we re-examined ten factors (cigarette smoking history, physical activity, BMI, family history of prostate cancer, race, height, total energy consumption, and intakes of calcium, tomato sauce, and α-linolenic acid) using multivariable Cox regression in relation to multiple subcategories for prostate cancer risk. In this analysis, only four factors had a statistically significant association with incident prostate cancer: African-American race, positive family history, higher tomato sauce intake (inversely), and α-linolenic acid intake. In contrast, for fatal prostate cancer, recent smoking history, taller height, higher BMI, family history, and high intakes of total energy, calcium, and α-linolenic acid and lower vigorous physical activity level were associated with a statistically significant increased risk. For these risk factors, advanced stage at diagnosis was a good surrogate for fatal prostate cancer, but high-grade (Gleason ≥7 or Gleason ≥8) was not. Only for high calcium intake was there a close correspondence for associations among high-grade cancer, advanced, and fatal prostate cancer. Tomato sauce (inversely) and α-linolenic acid (positively) intakes were strong predictors of advanced cancer among those with low-grade cancers at diagnosis. Although the proportion of advanced stage cancers was much lower after prostate-specific antigen (PSA) screening began, risk factors for advanced stage prostate cancers were similar in the pre-PSA and PSA era. Results of many studies have been conflicting, especially in the PSA era. Studies relying solely on incidence may have

limited applicability to identifying means to prevent dying from prostate cancer. Using Gleason sum to characterize aggressiveness may be informative in some contexts but is always not useful in identifying risk factors that influence disease progression. Fatal and advanced-stage prostate cancer may be informative endpoints, although advanced stage should be based on clear indicators, such as invasion into the seminal vesicle or other regional structures or metastasis to the lymph nodes, bone, or other organs. The complexity of the clinical and pathologic manifestations of prostate cancer must be considered in the design and interpretation of studies.

IMAGING

Perspectives on Physical Imaging as a Prostate Cancer Patient, Reviewer of Imaging Proposals, and as a Researcher

William H. Sproat

Us TOO International, Inc.

My Palladium 103 isotope brachytherapy was deferred until a new Bruel & Kjar ultrasonic instrument was acquired by the hospital of my choice and offered myself as a subject for staff training with the device. The phased array transducer and 3 dimensional color image synthesis provided an excellent model for dosimitry and prescribed isotope distribution. Hormonal ablation therapy with the Lueteinizing Hormone - Releasing Hormone (LH-RH) agonist Zoladex, first administered for the brachytherapy deferral, was also necessary to reduce the prostate volume below 50 cubic centimeters. Clinical depression was experienced following hormonal ablation; testosterone deprivation suspected on my part as the trigger for brain chemistry aberrations. Suspect cause/effect was validated on review of a proposal for magnetic resonance imaging of the hippocampus and frontal lobes of patients treated with testosterone deprivation agents.

My career research in nondestructive testing/inspection was the industrial equivalent of medical imaging. Many modalities are common to both and “cross-pollination” in exchanging imaging concepts can be most beneficial. Further, with nanoparticles emerging as transport platforms for detectors, the research community can tailor these platforms into “guided missiles” against errant cells. They can also be transducers, converting energy in one form to another. For example, eddy currents induced in electrically resistive nanoparticles clustered at disease sites could both detect and thermally eradicate the disease. Let’s have a “cross-pollination” seminar; industrial researchers meet medical researchers.

Prostate Cancer Imaging: The Progress Achieved and the Challenges Ahead

Hedvig Hricak

Memorial Sloan-Kettering Cancer Center

Given the biological heterogeneity of prostate cancer (PCa), noninvasive evaluation of tumor prognostic variables is of great clinical interest. Imaging is becoming increasingly important for the management of prostate cancer from diagnosis, to treatment selection, treatment planning, and follow-up.

No consensus exists regarding the use of imaging for evaluating primary prostate cancer. Ultrasound is mainly used for biopsy guidance and brachytherapy seed placement. When PSA is elevated but biopsies are negative, MRI may be useful for directing targeted biopsy. MRI has been shown to be significantly better than DRE in detecting cancer throughout the prostate and significantly better than TRUS-guided biopsy in detecting cancer in the mid-gland and base. Furthermore, MRI is helpful for assessing local stage (e.g., extracapsular extension and seminal vesicle invasion). The use of pre-operative MRI has been shown to improve the surgeon’s decision to preserve or resect the neurovascular bundles during radical prostatectomy.

MR spectroscopic imaging (MRSI) can be performed in the same examination and on the same scanner as MRI, using commercially available software. PCa identification on MRSI is based on the detection of an increased choline+creatine to citrate ratio and a decrease in polyamines. MRSI can improve PCa detection and localization on MRI, including identification of extracapsular extension and seminal vesicle invasion. As the ratio of choline+creatine to citrate in PCa correlates positively with Gleason grade, MRSI may also provide information about tumor aggressiveness.

One of the biggest challenges in managing PCa is the identification of low-risk disease. Inclusion of MRI or combined MRI/MRSI findings has been shown to significantly increase the prognostic value of clinical nomograms for predicting low-risk PCa.

Computed tomography (CT) is reserved for the evaluation of advanced disease. The use of combined positron emission tomography/CT is limited in assessing primary PCa but is gaining acceptance in treatment follow-up.

Lymphotrophic Nanoparticles in Prostate Cancer

Mukesh Harisinghani

Massachusetts General Hospital

Accurate pretreatment localization of metastastic lymph nodes is important to ensure optimal therapy in primary prostate cancer. Conventional cross-sectional imaging relies on anatomical nodal morphology and size as the primary yard stick for differentiating benign from malignant lymph nodes.

Using these nodal parameters, it is challenging to detect minimal tumor burden in normal size nodes. Lymphotropic nanoparticle-enhanced magnetic resonance imaging (LNMRI) has been recently evaluated and has proven to be an accurate technique to reliably determine nodal status in patients with various primary genitourinary cancers. LNMRI relies on the use of highly optimized MRI pulse sequences (dual echo gradient echo sequences), the administration of lymphotropic magnetic nanoparticles (e.g., Combidex; Advanced Magnetics Inc., Cambridge, Massachusetts; Sinerem; Guerbet, Paris, France), and sophisticated image analysis and comparison to enhancement databases. The strength of this imaging technique lies in its ability to provide high sensitivity (detecting minimal tumor burden) without compromising on the specificity. Owing to its ability to reliably detect metastastic nodes independent of their size, LNMRI has shown to be an effective presurgical and pretreatment planning tool. Information from patients scanned with LNMRI can allow one to create a comprehensive and composite map of nodal locations to define pelvic nodal regions at highest risk for harboring occult disease.

PROSTATE CANCER SCREENING

Prostate Cancer Screening: The Consumer Perspective

Merel Grey Nissenberg

National Alliance of State Prostate Cancer Coalitions

Prostate cancer is the most common non-skin cancer affecting American men and remains the second leading cause of cancer-related death for them. The current gold standard for screening is a combination of the Digital Rectal Examination and the PSA blood test, which while not perfect, remain the best hope for early detection of prostate cancer and thus a greater chance for curative, rather than palliative care. More research is needed to develop even better early detection tools. In the meantime, the DRE and PSA should be a regular part of men's health exams and all men should be aware of their PSA levels and more importantly any *changes* in their PSA. There is a glaring need for increased education and awareness of prostate cancer for as-yet undiagnosed men. Further, the debate about prostate cancer "screening" identifies critical challenges and opportunities for health care educators and clinicians to help men and their families make informed decisions regarding detection and treatment choices.

Prostate Cancer Screening with PSA

William Catalona

Northwestern University, Feinberg School of Medicine

Numerous medical organizations have developed a broad, and sometimes conflicting, range of recommendations for prostate cancer screening. The American Cancer Society recommends that both the prostate-specific antigen (PSA) blood test and digital rectal examination (DRE) should be offered annually, beginning at age 50, to men who have at least a 10-year life expectancy. African American men and men with a one or more first-degree relatives diagnosed before age 65 with prostate cancer should begin testing at age 45, while men with multiple first-degree relatives affected at an early age could begin testing at age 40. On the opposite end of the spectrum, the U.S. Preventive Services Task Force of the Agency for Healthcare Research and Quality has concluded that, "the evidence is insufficient to recommend for or against routine screening for prostate cancer using PSA testing or DRE."

Based on research in the changes in levels of PSA over a man's lifetime, the National Comprehensive Cancer Network has recommended that PSA measurements and prostate examinations should be performed annually for average-risk men starting at 40 years of age, and the results should be recorded so the PSA velocity can be calculated over time and be used as a marker of curable disease. The median PSA values for men without clinical evidence of prostate cancer is 0.7 ng/mL for men in their 40s, 0.9 ng/mL for men in their 50s, and 1.4 ng/mL for men 60 years of age or older. If a man's PSA level is at the median value or less for his age group, then the risk for prostate cancer is very low. If a man's PSA is higher than the median for his age group, his risk of having prostate cancer is higher and the risk that the cancer is aggressive is higher. An initial biopsy is recommended if the PSA is persistently higher than 2.5 ng/mL following a course of antibiotics and repeat PSA measurements to rule out prostatitis, which also raises PSA values. Although reducing the PSA threshold for biopsy could theoretically increase the number of unnecessary biopsies, many of these biopsies would be required later as the PSA level continued to rise. Also, some unnecessary biopsies could be eliminated through the use of PSA derivatives. While it is known that conditions such as benign prostatic hyperplasia can cause PSA elevations, the time course of such PSA increases typically differs from that of prostate cancer. Thus, measurements of PSA kinetics, particularly PSA velocity, may be useful to increase the specificity for prostate cancer screening.

Because the likelihood of curable prostate cancer correlates with the PSA level at diagnosis, we recommend the expansion of PSA screening to begin at age 40 to establish a baseline to measure PSA velocity, using a lower total PSA threshold, and the adjunctive use of PSA-based parameters such as PSA velocity to increase the specificity of screening for aggressive prostate cancer.

PSA Screening

Eddie Reed

Director, Division of Cancer Prevention and Control
Centers for Disease Control and Prevention

BASIC SCIENCE

Why and How I and My Fellow Survivors Need Basic Science

Thomas Blank

University of Connecticut

As almost 200,000 more men are added each year to the ranks of prostate cancer survivors, the search for answers to how to prevent, manage, and, ultimately, cure prostate cancer becomes ever more urgent. Clinical applications for primary treatment and for secondary treatments and longer-term management when the initial treatment is not sufficient are obviously crucial for all of us, perhaps especially men like me who are managing recurrences or advanced disease. But clinical applications do not appear de novo. Rather, they must be built on the firm foundations of solid basic science, from molecular and cellular to organ and organism levels. Theories, models, and empirical research on the mechanisms that underlie the development and growth of cancer cells and tumors are the keys to their defeat. Research on the processes that promote or inhibit growth of cancer cells or that create an environment less compatible with tumor growth is urgently needed so that it can form the basis for the next steps of impact on the prevalence and persistence of the disease. Basic research such as exemplified in this session can and must provide the building blocks of new generations of treatments. They are the essential underpinnings of progress in prostate cancer that fosters our hopes for a future without prostate cancer in our lifetimes.

Androgen Receptor Variants and Prostate Cancer in Humanized AR Mice

Diane M. Robins, Megan Albertelli, Orla A. O'Mahony, Kirk Wojno, and Michele Brogley

University of Michigan

The androgen receptor (AR) is involved in prostate cancer initiation, transition to androgen independence, and resistance to hormone therapy. Therefore genetic variation in AR may be informative for prognosis and treatment. The length of an N-terminal glutamine (Q) tract (CAG repeat) correlates inversely with AR transcriptional strength and has been implicated in prostate cancer risk. The epidemiological data, however, is confounded by ascertainment differences and complex gene-gene and gene-environment interactions. To interrogate AR and the Q tract, we converted the mouse AR to the human sequence by germline gene targeting, creating humanized alleles with 12, 21, or 48 Qs. These h/mAR mice are physiologically normal but vary subtly in some androgen-dependent traits and target gene expression.

To examine Q tract effects in prostate cancer, h/mAR mice were crossed with the transgenic adenocarcinoma of the mouse prostate (TRAMP) model. At 12 weeks of age, prostatic intraepithelial neoplasia (PIN) was most prevalent in 12Q mice, and although tumors were detected at similar ages for 12Q and 21Q, 12Q mice survived substantially longer. This suggests that the stronger 12Q AR drives earlier oncogene activation but slower disease progression. PIN, tumor detection, and death occurred later in 48Q mice, as predicted. Within each genotype, the time from tumor detection until death (disease length) was variable but correlated with stage of tumor differentiation. Tumors with longer disease length were more differentiated and expressed more AR than tumors that progressed rapidly, which were poorly differentiated. To test a role of Q tract length in androgen-independent disease, mice were castrated at 12 weeks. Remarkably, this resulted in Q tract length effects opposite to those in intact mice. This was most pronounced in the castrated 12Q mice where tumor detection and death were significantly delayed relative to other groups. The marked difference following castration suggests that Q tract length affects androgen-independent as well as -dependent progression, perhaps via an influence of the Q tract on ligand-independent AR activation. Moreover, the response to androgen ablation varied significantly for each allele. While all 12Q mice survived longer following castration, only half of the 21Q group benefited from treatment and 48Q mice apparently did worse. This suggests that differences in AR activity may direct distinct pathways of progression following androgen depletion.

IMPaCT: The h/mAR allelic series in a homogeneous mouse background reveals Q tract associations difficult to discern in man. We find that Q tract length affects both initiation and progression of prostate cancer, as well as response to hormonal therapy, highlighting significant risk associated with alterations in the androgen axis. This genetic paradigm should prove useful for dissecting mechanisms of androgen resistance, modeling response to treatment and testing therapies targeted specifically to the human AR.

Prostate Cancer and Inflammation

Angelo M. DeMarzo, Yasutomo Nakai, Elizabeth A. Platz, and William G. Nelson

Johns Hopkins Medical Institutions

Prostate cancer is the most common non-cutaneous malignant neoplasm in men in Western countries, responsible for the deaths of approximately 30,000 men per year in the United States[1], and the number of afflicted men is increasing rapidly as the population of males over the age of 50 grows worldwide. Thus, finding strategies for prevention of prostate cancer is a critical medical challenge. Since men in South East Asian countries have a low incidence of prostate cancer that increases rapidly upon immigration to the West, this disease is not an intrinsic feature of aging. The pathogenesis of prostate cancer reflects both hereditary and environmental components. What are the environmental exposures and genetic variations that have produced such an epidemic of prostate cancer?

Recent estimates indicate that approximately 20% of all human cancers in adults result from chronic inflammatory states/chronic inflammation[2, 3] that are triggered by infectious agents or environmental exposures, or by a combination

thereof. There is also emerging evidence for a role of inflammation in the etiology of prostate cancer. This evidence stems primarily from epidemiological, histopathological, and molecular pathological studies[4-8].

Histologically, the vast majority of lesions containing either acute or chronic inflammatory infiltrates in the prostate are associated with atrophic epithelium or focal epithelial atrophy[9-13]. Perhaps correspondingly, focal areas of epithelial atrophy are exceedingly common in the aging prostate[9, 14, 15] often encompassing a large fraction of the peripheral zone, where it most often appears[14, 16, 17]. Compared with normal epithelium, there is an increased fraction of epithelial cells that are proliferating in focal atrophy[11, 12, 18, 19], and we have suggested the term proliferative inflammatory atrophy (PIA) for most of these atrophic lesions[12, 20]. Not all focal prostate atrophy shows increased inflammatory cells and for these the term proliferative atrophy (PA) may be used. In morphological studies, we and others observe transitions at times between atrophic epithelium and adenocarcinoma[9, 21-23] and frequent transitions between areas of PIA/PA with high-grade PIN[12, 24].

Epidemiological studies have revealed a link between prostate cancer incidence and mortality and the consumption of red meat and animal fats[25-29]. One mechanism by which meats may stimulate cancer may relate to the formation of heterocyclic amines (HCAs)[30-32] that occur by cooking meats at high temperatures. HCAs can be metabolized to biologically active metabolites that can adduct to DNA and lead to mutations. 2-amino-1-methyl-6-phenylimidazo[4,5-b]pyridine (PhIP) is the most abundant HCA present in meats cooked at high temperatures[33, 34]. Exposure of laboratory rats to dietary PhIP results in carcinomas of the intestine in both sexes, in the mammary gland in females, and in the prostate in males[32, 35-37]. In a recent study we exposed laboratory rats to PhIP and found a similar increase in the mutation frequency in all lobes of the prostate, yet the ventral lobe selectively responded with increased cell proliferation and cell death[38]. Thus PhIP acts as both a lobe-specific classical tumor "initiator" as well as a tumor "promoter." We also found that only the ventral lobe showed an increase in stromal mast cells and stromal and intraepithelial macrophages[38]. At 12 weeks of PhIP exposure, the ventral lobe developed widespread epithelial atrophy; later PIN and intraductal carcinomas were observed to develop directly from the atrophic epithelium (AM DeMarzo, Y Nakai, WG Nelson, manuscript in process). Others have recently reported similar findings in that PhIP treatment was found to induce inflammation and atrophy prior to inducing PIN and intraductal cancers[39].

Our integrative model predicts that prostate injury occurs, in the correct genetic background, either as a result of diet, inflammation or both, and that this leads to regenerative lesions referred to collectively as PIA/PA. Given their frequency and extent, if only a small fraction of these atrophic lesions progress to PIN and or carcinoma, they may be highly clinically significant.

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Prostate Cancer Stem Cells

John Isaacs

Johns Hopkins Medical Institutions

Recent research has found the involvement of embryonic proteins and pathways in cancer and tumorigenesis associated with embryonic pathway activation may result from misdirection of cells toward stem cell or stem cell-like phenotypes. These more primitive, transformed versions of the tissue's normal cells also may be the cells that can metastasize to form new tumors in distant locations. Stem cells also may be responsible for not only maintaining the tumor but also the development of androgen-independent disease. An important aspect of prostate cancer treatment is that androgen ablation kills tumor cells, easing the symptoms associated with tumor burden. However, it is possible that androgen-independent cancer stem cells survive treatment and continue to multiply. Over time, the stem cells may acquire further mutations that allow its progeny to become androgen independent, and the tumor returns in a more advanced form. Recent studies will be presented showing the interrelationship between prostate cancer stem cells and androgen-independent tumors.

Estrogens/Antiestrogens in Prostate Cancer Etiology and Treatment

Shuk-mei Ho

University of Cincinnati

Estrogens, in synergy with androgens, are now known to play a key role in the pathogenesis of prostate cancer (PCa). New experimental evidence suggests that the estrogenic influence may begin as early as prenatal life. During adulthood, estrogen carcinogenicity is mediated by the combined effects of hormone-induced, unscheduled cell proliferation, oxidative stress, and generation of genotoxic intermediates. Increased bioavailability of estrogen through age-dependent increases in conversion from androgen may also be an important contributing factor. Individual variations and race-/ethnic-based differences in circulating or locally formed estrogens, working in conjunction with disparity in tissue responsiveness may explain differential PCa risk amongst individuals or different populations. Estrogen receptor (ER)α and ERβ are the main mediators of estrogen action in the prostate. However, ERβ is the first ER subtype expressed in the fetal prostate. Intriguely, ERβ is also the predominant receptor subtype found in normal and malignant epithelial cells of the prostate, and in bone and lymph node metastases. During cancer development, ERβ expression is first lost as PCa progresses into high grade in the primary site. Yet, the receptor is strongly expressed in all metastatic PCa. DNA methylation has been shown to regulate transcriptional silencing and reactivation of the *ERβ* gene. A variety of estrogenic/antiestrogenic/selective estrogen receptor modulator (SERM)-like compounds has been tested for their efficacies in treating PCa. Their modes of action have been shown to be mediated by classical and non-canonical signaling pathways. New therapeutics targeting ERβ signaling pathways may open new therapeutic avenues. A new generation of estrogen-based PCa therapies with maximal pro-apoptotic action but few or no side effects may be forthcoming in the near future.

SPOTLIGHT SYMPOSIUM: THE PROSTATE CANCER CLINICAL CONSORTIUM: TRANSLATION OF SCIENTIFIC DISCOVERY TO THE CLINIC

Life with Prostate Cancer

Roland Young

Us TOO International, Inc.

Forming and Operating a Multi-institutional Research Consortium

Howard I. Scher

Memorial Sloan-Kettering Cancer Center

In 2006, the DOD began funding the Prostate Cancer Clinical Trials Consortium (PCCTC), a collaborative that consists of clinical investigators at 10 academic research centers throughout the United States. By bringing these researchers together, the goal of this clinical consortium was to streamline clinical trial execution by leveraging economies of scale and consolidating the cross-institutional expertise of basic scientists, clinical researchers, and database managers. To date, such consortia are uncommon because traditional trial funding mechanisms account for costs on a per-trial, per-patient, or per-agent basis. Creating a common infrastructure across institutions to implement multi-institutional trials presents challenges, but the experience of the PCCTC is encouraging for other groups that want to establish an infrastructure that will advance research in their fields.

Multi-institutional clinical trials can accrue patients from many centers simultaneously and therefore deliver results of novel therapies earlier than traditional single-institutional studies. The pooled expertise of scientists and clinicians broadens the experience with an agent sooner while offering cross-institutional uniformity of design and outcome measures. Larger studies with uniform measures of clinical effects are easier to interpret in terms of deciding whether to advance or abandon a given regimen. In addition, they provide a framework to evaluate biomarkers in clinically relevant contexts. Large data sets that are both consistent and of high quality would result.

Challenge 1: Sharing data among different institutions while protecting patient privacy and institutional and sponsor intellectual property. The PCCTC sought a database solution that could work across institutional systems. By mutual agreement, consortium members adopted a database that allows each member to propose and sign up for trials through a secure online letter-of-intent process.

Challenge 2: Guaranteeing consistent cross-institutional sample collection, sample handling, data acquisition, and data reporting. The PCCTC planned to collect samples and clinical data consistently so that they could be mined across trials to test new ideas. Through a consensus process, consortium members developed common data elements and vocabulary to address these issues before samples or data were gathered. They also developed standard operating procedures that codified the standards for consistency and language. Finally, they developed a protocol template so that these same standards would be presented to and approved by the sponsors of the trials and the institutional review boards (IRBs) of the institutions.

Challenge 3: Expediting patient accruals to consortium trials. Multi-institutional trials face multiple IRBs, which could hamper the process of opening trials to patients. Protocols were tracked from concept design to scientific review to IRB review across institutions. In addition, a common protocol template that met both local IRB, Cancer Therapy Evaluation Program, and U.S. Food and Drug Administration requirements enhanced the likelihood of efficient and expeditious review.

Challenge 4: Creating a business plan with provisions for revenue after the grant funding runs out. Because industry-sponsored trials on a registration track offer the highest per-case reimbursement, and investigator-initiated (i.e., non–registration-track) studies remain difficult to fund, the PCCTC has started to consider the market value of its rarefied scientific activities, such as consulting, developing protocols, defining endpoints, discovering and validating biomarkers, and providing clinical insights by experienced investigators. Some potential clients could include the pharmaceutical industry, biotech companies, venture capitalists, as well as nonprofit organizations and individual philanthropists.

The utility of an infrastructure that removes obstacles and creates efficiencies for researchers has been demonstrated. More experience from this and other consortia is necessary to determine which specific approaches provide the best research environment, minimize cost, and most effectively speed the research.

CLINICAL RESEARCH

Expectant Management of Prostate Cancer: A Survivor's Viewpoint

Robert Carey

Georgia Prostate Cancer Coalition and Men Coming Together

If the option is available to him, a man's choice between expectant management or immediate treatment and surgery after a diagnosis of prostate cancer is a difficult decision to make. There are questions and there are fears. There are emotional and psychological challenges to overcome. There is much to learn about prostate cancer and there might be limited time to learn. There are choices to be made. There will be changes in his life and there could be changes in his quality of life. Where does he go? Who does he talk with? There are many life-altering and life-changing possibilities that could take place that must be considered in making this decision. The presentations made by the scientists in this session will emphasize the importance of this decision, the consequences that result from the decision, and other aspects that should be taken into consideration when expectant management is chosen for prostate cancer. Also important are the new ways that doctors can tell when expectant management needs to be replaced with active treatment of the cancer.

Dietary and Lifestyle Adjuncts to Expectant Management

Stephen Freedland

Duke University Medical School

The majority of men diagnosed with prostate cancer will die of causes other than prostate cancer. This fact coupled with the fact that even men who die of their disease, the clinical course is often slow with a long extended period of time until death, has given rise in an interest in expectant management. While for some men, expectant management may be better termed deferred curative therapy, some will never need to undergo curative therapy. For all of these men, non-prostate cancer mortality remains the greatest threat to overall health. Specifically, for these men, heart disease is the number one cause of death. Given that dietary and lifestyle interventions are known to reduce the risk of heart disease, these therapies for men with low-risk disease are likely to have a greater impact on improving overall survival than any prostate cancer-related treatment. Moreover, many patients are uncomfortable about doing "nothing" for their cancers. Therefore, by including dietary and lifestyle modifications, the patient can take an active role in their care and gain some sense of control. Finally, while all prostate cancer treatments can worsen quality of life, particularly sexual function, dietary, and lifestyle interventions can actually *improve* overall quality of life and have been shown to improve erectile function. Therefore, for men with low-risk disease undergoing expectant management, no single intervention can have as much of a positive impact of quality and quantity of life as dietary and lifestyle intervention. The difficulty is determining the optimal intervention. While reducing calories, exercising, and losing weight are the best advice, this is not always easy to follow. Unfortunately, the best approach for weight loss is unclear. Increasing evidence suggests avoidance of red meat and simple carbohydrates and increased intake of fruits, vegetables, and fish may be beneficial. However, given that heart disease is the number one cause of death among these men, we must constantly be thinking heart first and prostate second. However, it is highly likely that heart healthy is also prostate healthy.

Active Surveillance for Favorable Risk Prostate Cancer

Laurence Klotz

Sunnybrook Health Sciences Centre

Identification and Results of Management of Potentially Indolent Prostate Cancer

Fritz H. Schröder and the Rotterdam ERSPC Study Group

Erasmus University Medical Center

The application of screening tests for prostate cancer driven by men in the age groups at risk is increasing in Western countries. Reported rates in Europe run between 20 and 40, in the United States between 24% and 75% depending on region and age distribution of the different studies. The value of screening in terms of reducing prostate cancer specific mortality at an acceptable impact on quality of life and cost has not yet been established. The European Randomised Study of Screening for Prostate Cancer (ERSPC), the basis for this report, is set to contribute to the resolution of this issue.

One of the prominent features of prostate-specific antigen (PSA) based screening for prostate cancer is the identification of men who otherwise would never experience the presence of prostate cancer by symptoms or by disease progression leading to death (overdiagnosis). If screening within a few years would indeed be shown to be effective in terms of lowering prostate cancer mortality, quality of life adjustments will be carried out; overdiagnosis and specifically the treatment of men who are not at risk of experiencing prostate cancer as a disease (overtreatment) will be important issues. It is for this reason that within ERSPC, major effort was taken to identify the rate of overdiagnosis (Draisma et al, *JNCI* 2003) and to reduce overdiagnosis by attempting to identify cases that are not at risk. The latter issue has led the group to validate a nomogram (Kattan, *J Urol* 2003) for the identification of indolent disease in a setting of clinically diagnosed cases. A rate of indolence of 20% was found by Kattan in 1,022 radical prostatectomy specimens. In using exactly the same criteria for identification of indolent cases on a screen-detected series of 287 men who had cancers identified by screening and who underwent radical prostatectomy, a rate of insignificant cancer in those surgical specimens of 49% was found. In validating the Kattan nomogram (Steyerberg et al; 2007), a new nomogram for the prediction of indolent disease in screen-detected prostate cancer could be developed. Using a cut-off for the probability of having indolent disease of 70% (score 21) 133 of 142 men suffering of potentially aggressive disease were correctly classified and treated (49%). Six percent of advanced cases is incorrectly classified with this cut-off value. The same probability cut-off will identify 43 of 136 indolent cases (32%). This means that 68% of the potentially indolent cases would still be actively treated.

With this conservative cut-off for the probability, indolent in mind, an internal validation has been carried out (Roemeling et al, 2007, submitted). In applying the same criteria, 30% of all screen-detected cancers in the first round and a higher proportion at repeat screening can be classified as indolent.

Obviously further validation will have to take place. External validation of the nomogram is planned in two different settings: A United States cohort at the University of Cleveland, Ohio, and a screen-detected cohort in Sweden. Furthermore, internal validation on all screen-detected cases in Rotterdam that might be suitable for active surveillance according to the identified criteria (PSA range, ultrasound volume of the prostate, biopsy Gleason score, millimeter of cancer, and millimeter of non-cancerous tissue) has been carried out. Since in the setting of ERSPC cancers are treated according to regional preference (n=291) these men had a choice to be treated by radical prostatectomy, radiotherapy, or active surveillance. Four men developed metastatic disease

and 3 died of prostate cancer within a follow-up period of more than 8 years. None of the 64 men who had chosen active surveillance developed metastatic disease or died of prostate cancer (Roemeling et al., *Eur Urology* 2006). These data are in line with other observations obtained from prospective active surveillance studies.

The newly developed algorithm can serve to identify prostate cancer cases that are eligible for management by active surveillance, preferably in protocols evaluating trigger points for treatment and outcomes. Furthermore, once screening becomes a formal health care policy, the possibility to avoid overtreatment in 30% of screen-detected cases will decrease the impact of screening on quality of life in a significant fashion and help to convince health authorities to establish national screening programs.

Expectant Management of Prostate Cancer

Christopher Warlick

University of Minnesota Medical School

Men screened for prostate-specific antigen (PSA) are diagnosed an estimated 10 years earlier in the natural history of prostate cancer than men diagnosed without PSA screening. This has led to the diagnosis of some cancers that would not have been detected in the absence of screening (i.e., overdiagnosis). Overdiagnosis leads to overtreatment since more than 90% of men diagnosed with prostate cancer today undergo active treatments. Expectant management (or active surveillance) with delayed curative intent (i.e., surgery or radiation therapy) has been proposed as an alternative to immediate surgery for carefully selected men with newly diagnosed prostate cancer that is low grade and low stage as an approach that could reduce unnecessary treatment. Selection of those men for whom surveillance would be safe, and defining the criteria that lead to intervention during a window of curability for those whose disease progresses during surveillance, is an important area for future research.

We have 407 men (median age 66 years, range 46, 82) with stage T1c prostate cancer in our expectant management program who are thought to have small-volume prostate cancer based on needle biopsy findings and PSA density. A total of 382 subjects have been followed for more than 1 year with semiannual PSA and digital rectal examination, and annual surveillance prostate biopsies (median follow-up 3 years, range 1,13). A recommendation for treatment has been made if progression of disease is suggested by unfavorable follow-up needle biopsy findings (Gleason pattern 4 or 5, >2 biopsy cores with cancer, >50% involvement of any core with cancer). A total of 239 of 407 (59%) of the men remain on active surveillance with a median follow-up of 3 years (range = 0.4,13), and 103 of 407 (25%) have undergone curative intervention at a median follow-up of 2 years (range = 1, 7). Age at diagnosis (p=0.012), PSA density (PSA divided by prostate volume) at diagnosis (p= 0.037), and date of diagnosis (p=0.002) were significantly associated with curative intervention in a multivariate risk analysis (two-sided chi square test) while PSA velocity based on all PSA measurements was not (p= 0.479).

Recognizing the indolent nature of many prostate cancers diagnosed today, a program of active surveillance for carefully selected men who are thought to harbor small-volume disease seems rational.

GENETIC EPIDEMIOLOGY

The Importance of Genetic Epidemiology

Virgil Simons

The Prostate Net

Genetic Epidemiology: Search for Prostate Cancer Genes

Jianfeng Xu

Wake Forest University School of Medicine

Genetic susceptibility to prostate cancer is well established. Genetic linkage studies have been widely used to identify chromosomal regions harboring major prostate cancer genes among familial prostate cancer families. Genetic association studies are effective approaches for the identification of specific sequence variants that are directly or indirectly associated with prostate cancer risk among prostate cancer case and control subjects. With rapid advances in high-throughput genotyping technology and the great increase in information available on SNPs throughout the genome, high density fine mapping of linkage regions, systematic pathway analysis, and even genome-wide searches have now become feasible. We will discuss several examples of these approaches, including genetic linkage studies among families of the International Consortium for Prostate Cancer Genetics (ICPCG), linkage and association studies of germline DNA copy number changes, systematic searches for sequence variants in the inflammation pathway, and genome-wide association.

Prostate Cancer Genetics Today and Tomorrow

Henrik Grönberg

Karolinska Institutet

COMPLEMENTARY AND ALTERNATIVE MEDICINE

Alternative Herbal Treatment in Conjunction with Conventional Treatment for Prostate Cancer

James McGuinness

American Cancer Society, Brevard Office, and Man to Man

Do herbals treatments work? Yes, and I will detail my treatment with them from the date I was diagnosed in 1997 until today. I will explain how I came to the decision to use herbal treatments working closely with my oncologist.

Does conventional therapy work in conjunction with the alternate treatment? Yes, it did until the conventional treatment started to cause kidney damage. I will detail the treatments that we used, what caused the kidney damage, and how and why we changed plans after several years.

What happened to "PC SPES?" My personal feelings are that this was a tragic loss of a good product that needed more quality control. Its loss affected thousands of hormone refractory men that were being treated with it, and they were left floundering for a substitute, with the eventual loss of a lot of lives needlessly. My suggestions on this subject have been culled from the thoughts of many prostate cancer survivors.

Where do we go from here? Some suggestions from many of my fellow prostate cancer survivors and some serious discussion on this subject time permitting. Finally a "thank you" to IMPaCT for allowing me to attend and discuss this important subject at this session.

The Impact of Flaxseed Supplementation on Prostate Cancer

Wendy Demark-Wahnefried

Duke University Medical Center

Flaxseed is a unique food distinguished by its high lignan content and high levels of plant-based omega-3 fatty acids in the form of alpha-linolenic acid. These characteristics make it a potentially compelling, yet controversial functional food with regard to cancer control. This presentation will review the epidemiologic evidence regarding lignan and omega-3 fatty acids (including alpha-linolenic acid) in relation to prostate cancer and will cover results of studies in cell culture and animal models that have explored the impact of flaxseed supplementation or flaxseed-derived components on the growth of prostate cancer. Finally, findings of a recent Phase II trial that was designed to ascertain the comparative effects of flaxseed supplementation in contrast to a fat-restricted diet on prostate cancer proliferation rates and related biomarkers will be discussed.

Approaches for Characterizing Cellular Responses to Complementary/Alternative Medicine Therapies

Peter Nelson

Fred Hutchinson Cancer Research Center

Complementary and alternative medicine (CAM) comprises a wide variety of interventions that are often used for the prevention and treatment of cancer and other diseases but are not presently considered to be part of conventional medicine. It has been estimated that as many as 80% of adult cancer patients use at least one form of CAM during or after conventional treatment. Prostate cancer, which is characterized by a long disease latency period, limited treatment strategies for advanced disease, and strong dietary influences, is a model disease for CAM therapies. Many men turn to CAM with the belief that it represents a viable therapeutic option that is free of adverse side effects.

Herbal extracts are one component of complementary medicine frequently taken by men with advanced disease. While some herbal extracts may have anti-cancer activities, their modes of action are poorly characterized, and the potential side effects are often unrecognized. One herbal extract mixture, PC-SPES, was used by many men because it had shown efficacy in patients with androgen-dependent and androgen-independent prostate carcinoma. The mixture contained extracts from eight common herbs that had been used medicinally as treatments for a variety of conditions. Studies of individual herbal constituents in PC-SPES have been shown to inhibit tumor growth, although the mechanisms of these activities are poorly defined. Though clinical benefits were observed, random testing of PC-SPES in the laboratory showed that its capsules also contained warfarin and diethylstilbestrol (DES), which might be providing the clinical benefit observed in the patients who took the supplement.

More study is needed to define the efficacy and modes of action of CAM therapies. My laboratory used gene expression profiling methods to determine the mechanism(s) by which PC-SPES exerted its cytotoxic effect. PC-SPES treatment of prostate cancer cells altered the expression of several cytoskeletal genes that included microtubule proteins. Although paclitaxel (a microtubule-stabilizing drug) stabilized and PC-SPES treatment disrupted microtubule architecture in LNCaP cells, the combination of both agents had an intermediate effect. PC-SPES inhibited tubulin polymerization in vitro, even in the presence of paclitaxel. The in vivo effects of PC-SPES and paclitaxel were assessed using androgen-independent prostate cancer xenografts. Compared with tumors in control mice, tumors were statistically significantly smaller in mice that received PC-SPES, paclitaxel, or the combination of PC-SPES and paclitaxel. However, tumor responses to the combination of paclitaxel and PC-SPES were less than either agent alone, indicating that PC-SPES could antagonize the cytotoxic effects of paclitaxel. This activity has implications for the clinical management of patients with advanced prostate cancer who may be taking PC-SPES concurrently with microtubule-modulating chemotherapeutic agents.

As learned with PC-SPES, herbal compounds contain bioactive substances that may enhance or inhibit the effects of conventional therapies. More oversight is needed in the manufacturing and component testing of herbal and dietary supplements. A recent U.S. Food and Drug Administration announcement establishing regulations to require current good manufacturing practices for dietary supplements will assist in providing assurances to patients and caregivers that the products they use contain the identified components at the purity, strength, and composition stated on the label.

HORMONE REFRACTORY PROSTATE CANCER

The Survivor's Journey

William Bright

Us TOO International, Inc.

The hormone refractory prostate cancer (HRPC) survivor is at the end of the treatment options line. Of the various HRPC treatment options available, there are varied survivor perspectives of these options. This presentation will address the issues and dilemmas these men have in: sorting among and choosing the appropriate treatment options; adjusting to the treatment chosen; and confronting the ensuing physical, mental health, and relationship issues. Attention will be directed to the aging process and the choice of treatment options. The journey of a survivor will be presented.

Hormone Refractory Prostate Cancer

Donald J. Tindall

Mayo Clinic and Foundation

Prostate cancer is a significant cause of morbidity and mortality worldwide. Normal prostate tissue is regulated by androgens, which activate the androgen receptor, a nuclear receptor transcription factor. Most prostate tumors retain androgen dependence. Therefore, current therapies for advanced prostate cancer either reduce androgen levels or prevent binding to the androgen receptor. Despite this regimen, prostate cancer invariably progresses to a fatal, androgen-refractory state. Although these relapsed tumors are androgen independent, they are still dependent on the androgen receptor for their growth and survival. The focus of this talk will be to highlight our current understanding of the mechanisms of androgen receptor activation in androgen-refractory prostate cancer. How these mechanisms of androgen receptor activation could be targeted in this advanced stage of the disease is also discussed.

Mechanisms Underlying Hormone-refractory Prostate Cancer

Edward Gelmann

Columbia University College of Physicians and Surgeons

Resistance to androgen ablation marks the last phase of prostate cancer and occurs after a highly variable period of clinical response. In both experimental models and translational studies, hormone-refractory prostate cancer is characterized by activation of androgen receptor and its downstream pathways despite the presence of castrate levels of circulating androgens. Activation of androgen receptor signaling can result from gene amplification or overexpression of mRNA. Activation of oncogenes such as SRC, can result in androgen receptor phosphorylation that can activate the receptor in a ligand-independent manner. In some cases, particularly after prolonged exposure to antiandrogens, androgen receptor mutations arise that cause an agonistic response to antiandrogens and broaden the ligand specificity of the receptor to confer responsiveness to adrenal androgens and other steroids. In some tissues activation of androgen synthetic enzymes and production of androgens locally appear to mediate tumor progression. Lastly, increased expression of steroid receptor coactivators and activation of proteins that can enhance androgen receptor signaling such as β-catenin may affect androgen receptor activation in the presence of castrate levels of circulating androgens. The common denominator of all these mechanisms is the sensitization of androgen receptor to subphysiologic levels of androgen and other steroids or activation of androgen receptor in a ligand-independent manner.

TREATMENT AND MANAGEMENT OF PROSTATE CANCER

Winston Dyer

New York Prostate Cancer Community Outreach Project

Daniel Petrylak

Columbia University College of Physicians and Surgeons

Curtis Pettaway

University of Texas M.D. Anderson Cancer Center

Richard Valicenti

Associate Professor of Radiation Oncology
Thomas Jefferson University Hospital

SPOTLIGHT SYMPOSIUM: MANHATTAN PROJECT FOR TARGETING THE LETHAL PHENOTYPES OF PROSTATE CANCER

Prostate Cancer Research Consortium – A Patient's Perspective

John L. Willey

Pennsylvania Prostate Cancer Coalition and Intercultural Cancer Council

New targets are needed for treating the lethal phenotypes of hormone refractory prostate cancer. Modeling his group of investigators after the Manhattan Project, Dr. Simons chose the best scientific investigators in their fields, and these scientists and physicians dedicated themselves to developing "weapons" that could eliminate the threat of hormone refractory prostate cancer. Like General Grove, Dr. Simons coordinated and supplied the necessary materiel and infrastructure for this Consortium and, as Groves was described by Los Alamos scientist Robert Bacher, Dr. Simons proved to be "a genius at getting things done under very adverse circumstances." Despite initial funding cuts, he and his investigators showed that bringing together people from a variety of disciplines against a common enemy could solve problems that are too complex to be solved by any one person or any one discipline.

Manhattan Project for Targeting the Lethal Phenotypes of Prostate Cancer

Jonathan Simons

Prostate Cancer Foundation

QUALITY OF LIFE

Life after Prostate Cancer

Richard and Desiree Howe

Tex Us TOO

Improving Patient Decision Making in Prostate Cancer Care

Sara J. Knight

University of California, San Francisco

Shared decision making in prostate cancer treatment depends on an accurate understanding of patient values, goals, and preferences for care. Previous assessments of patient preferences for prostate cancer care have focused on well-defined, clinically derived attributes such as urinary and sexual function. In contrast, recent investigations have identified a broad range of personal and interpersonal considerations that influence treatment choice, such as anxiety and self-esteem. This presentation will highlight advances in conceptual models and methods that incorporate these complex considerations in prostate cancer treatment decisions, with an emphasis on using these innovative frameworks to contribute to improved individual decision making in prostate cancer care.

Quality of Life for Men with Prostate Cancer: Targets for Intervention

David M. Latini

Baylor College of Medicine

Numerous descriptive studies have examined clinical and quality of life outcomes for men with prostate cancer over the last 10 years. These advances in our understanding of prostate cancer outcomes have resulted from work by investigators on large disease registry studies, particularly CaPSURE™ and the Prostate Cancer Outcomes Study (PCOS). These and other studies suggest points of intervention where clinical researchers can focus their efforts to improve quality of life for men with prostate cancer and their partners and caregivers. Previous quality of life interventions for men with prostate cancer and directions for future research will be discussed.

POSTER SESSIONS

Prostate Cancer Advocacy

Sep 6 1:00 p.m.-3:00 p.m.
Odd-numbered: 1:00 p.m.-2:00 p.m.
Even-numbered: 2:00 p.m.-3:00 p.m.

P1-1 The Arkansas Prostate Cancer Foundation Campaign against Prostate Cancer
Helen Baldwin
Arkansas Prostate Cancer Foundation

P1-2 Massachusetts Prostate Cancer 10th Annual Symposium: A Model for Prostate Cancer Education
Ingolf Tuerk[1], Matthew Smith[2], Richard Babayan[3], Glenn Bubley[4], A. Oliver Sartor[5], and Judy L. Green[6]
[1]Lahey Clinic, [2]Massachusetts General Hospital, [3]Boston Medical Center, [4]Beth Israel Deaconess Medical Center, Boston, [5]Dana-Farber Cancer Institute, [6]Massachusetts Prostate Cancer Coalition, Inc.

P1-3 The Florida Prostate Cancer Network: A State-wide Instrumentation for Comprehensive Delivery of Prostate Cancer Education and Outreach
B. Lee Green and Brian M. Rivers
H. Lee Moffitt Cancer Center and Research Institute at University of South Florida

P1-4 A Survivor Network for Outreach and Education
Thomas A. Farrington
Prostate Health Education Network (PHEN)

P1-5 Unique Treatment Issues for Gay Men Diagnosed with Prostate Cancer
Darryl Mitteldorf[1], Gerald Perlman[1], and Vincent M. Santillo[2]
[1]Malecare, Inc.
[2]Columbia University Medical School

Quality of Life

Sep 6 1:00 p.m.-3:00 p.m.
Odd-numbered: 1:00 p.m.-2:00 p.m.
Even-numbered: 2:00 p.m.-3:00 p.m.

P2-1 Decisions and Outcomes in Early Prostate Cancer: Stories Men Tell of Meeting the Challenge of Choosing Their Treatment
Jack A. Clark[1] and James A. Talcott[2]
[1]Boston University School of Public Health, [2]Massachusetts General Hospital

P2-2 Does It Make a Difference How Hot Flashes in Prostate Cancer Survivors Are Measured?
Steven C. Palmer, Liisa Hantsoo, David J. Vaughn, James C. Coyne, and Laura J. Hanisch
University of Pennsylvania

P2-3 Patient-reported Outcomes in Quality Assessment and Quality Improvement: Extending Technology Assessment of a Novel Prostate Brachytherapy Technique to Medical Treatments with Side Effects That Affect Quality of Life
R.C. Chen[1], J. Manola[2], A.V. D'Amico[3], A.L. Zietman[1], I. Kaplan[4], J.A. Clark[5], and J.A. Talcott[1]
[1]Massachusetts General Hospital, [2]Dana-Farber Cancer Institute, [3]Brigham and Women's Hospital, [4]Beth Israel Deaconess Medical Center, Boston, [5]Boston University School of Public Health

P2-4 Adaptation to Prostate Cancer
Mary Harper[1], Jonathan Decker[1], Karen Edmonson[1], Deborah Moore[1], Gloria Velez[1], Allison Edmonds[1], Sandy Knapp[1], Candace Eden[1], Laurie Stark[1], and Lorrie L. Powel[2]
[1]University of Central Florida, [2]University of Texas Health Science Center at San Antonio

P2-5 Androgen Deprivation Therapy for Prostate Cancer Is Not Associated with Cognitive Impairment
London C. Butterfield[1], Rosette C. Biester[2], David J. Vaughn[2], James C. Coyne[2], and Pamela J. Shapiro[1,3]
[1]Fox Chase Cancer Center, [2]University of South Florida, [3]University of Pennsylvania, School of Medicine

P2-6 Novel Dietary Indole Analogs for Prostate Cancer Treatment: Managing Prostate Cancer with Quality of Life Considerations
Dawn Yean, Wan-ru Chao, Khalid Amin, Carol Green, James Bakke, and Ling Jong
SRI International

P2-7 Variation in Indirect Costs of Newly Diagnosed Prostate Cancer Patients
Sumedha Chhatre, Richard Whittington, Alan J. Wein, S. Bruce Malkowicz, and Ravishankar Jayadevappa
University of Pennsylvania

P2-8 A Patient–Spouse Centered Intervention to Facility Treatment Decision Making for Localized Prostate Cancer: Results from a Randomized Trial
Nihal Mohamed[1], Eric Horwitz[2], Robert Uzzo[1], Richard Greenberg[1], Michael A. Diefenbach[1], and Alan Pollack[2]
[1]Mount Sinai School of Medicine, New York, [2]Fox Chase Cancer Center

P2-9 Variations in Quality of Care for Men with Early-stage Prostate Cancer
David C. Miller[1], Mark S. Litwin[1], Jamie D. Ritchey[2], Andrew K. Stewart[2], Rodney L. Dunn[3], E. Greer Gay[2], Howard M. Sandler[3], John T. Wei[3], and Benjamin A. Spencer[4]
[1]University of California, Los Angeles School of Public Health, [2]American College of Surgeons Commission on Cancer, [3]University of Michigan, [4]University of California, Los Angeles

P2-10 The Brain Basis of Memory Loss with Androgen Deprivation Therapy: Methods Development and Preliminary Findings
Mark A. Krause, Tomasz M. Beer, and Jeri S. Janowsky
Oregon Health and Science University

P2-11 Evaluation of Long-term Outcome among Men with Conservatively Treated Localized Prostate Cancer
Ove AndrÉn[1], Sven Perner[2], Eberhard Varenhorst[3], Jan-Erik Johansson[1], Hans-Olov Adami[3], Katja Fall[3], Lorelei Mucci[4], and Mark Rubin[2]
[1]Örebro University Hospital, Sweden, [2]Brigham and Women's Hospital, [3]Karolinska Institute, [4]Harvard Medical School

P2-12 The Prostate Interactive Education System (PIES), a Multimedia Education and Decision Aid for Prostate Cancer Patients: Results from a Randomized Controlled Trial
Brian P. Butz[1], Amanda C. McCulley[2], Elissa A. Kolva[2], Michael A. Diefenbach[2], and Simon J. Hall[2]
[1]Temple University, [2]Mount Sinai School of Medicine, New York

P2-13 The Role of Historical Treatment Patterns in Current Prostate Cancer Mortality Trends
Ruth Etzioni and Steven B. Zeliadt
Fred Hutchinson Cancer Research Center

Prostate Cancer Screening

Sep 6 1:00 p.m.-3:00 p.m.
Odd-numbered: 1:00 p.m.-2:00 p.m.
Even-numbered: 2:00 p.m.-3:00 p.m.

P3-1 Pharmacists as Health Educators and Risk Communicators in the Early Detection of Prostate Cancer
Helene Vilme[1], Steven Young[1], Folakemi T. Odedina[1], and Cynthia Warrick[2]
[1]Florida A&M University, Tallahassee, [2]Elizabeth City State University

P3-2 Detection and Diagnosis of Prostate Cancer Via Internet and Wireless Communication Networks
Yu-Dong Yao
Stevens Institute of Technology

P3-3 Evaluation of an Innovative Partner-focused Mail-home Intervention to Facilitate Prostate Cancer Risk Assessment among High-risk Men
Joanne Buzaglo, Regina Coles, Kuang-Yi Wen, Michael A. Diefenbach, and Suzanne M. Miller
Fox Chase Cancer Center

P3-4 Internet-based Education for Prostate Cancer Screening
Kathryn L. Taylor, Marc D. Schwartz, Kimberly M. Davis, Paula Goldman, David Dawson, Mary Fishman, Carmella Cole, Alex Krist, Steven Woolf, and Janet Ohene-Frempong
Georgetown University

P3-5 Barriers to Prostate Cancer Screening: Focus Group Findings
Calvin Atchison, Michelle C. Reece, Baqar A. Husaini, and Pamela C. Hull
Tennessee State University

P3-6 Informed Decision Making for Prostate Cancer Screening: An African American and Hispanic Perspective
Theresa L. Byrd[1], John R. Ureda[2], Heather M. Brandt[3], Jessica A. Calderon[1], Myriam E. Leyva[4], and Evelyn C.Y. Chan[1]
[1]University of Texas Health Science Center at Houston, [2]Insights Consulting, Inc., [3]University of South Carolina, [4]University of Texas at El Paso

Health Disparities

Sep 6 1:00 p.m.-3:00 p.m.
Odd-numbered: 1:00 p.m.-2:00 p.m.
Even-numbered: 2:00 p.m.-3:00 p.m.

P4-1 Study of Prostate Cancer Screening, Co-morbidity and Mortality in Blacks and Whites
W. Mkanta[1], Y. Ndjakani[1], F. Bandiera[2], J. Yongsung[1], D. Blumenthal[2], U. Nseyo[3], and N.R. Asal[1]
[1]University of Florida, [2]Morehouse School of Medicine, Atlanta, [3]VA Medical Center

P4-2 Variation in Return to Baseline Values of Patient Reported Outcomes across Racial/Ethnicity in Elderly Prostate Cancer Patients
Jerry C. Johnson, Sumedha Chhatre, Alan J. Wein, S. Bruce Malkowicz, and Ravishankar Jayadevappa
University of Pennsylvania

P4-3 Analysis of Variations in Longitudinal Prostate-specific Antigen Levels in a Community Screening Population
Cary N. Robertson, Seronda Arlette Jackson, and Marva Mizell Price
Duke University

P4-4 Racial Variation in the Matrix Metalloproteinase: E-Cadherin Ratio in Localized Prostate Cancer
Renduo Song, Xuemei Wang, Jun Liu, Peter C. Black, Ina N. Prokhorova, Cindy Soto, Patricia Troncoso, Timothy J. McDonnell, Curtis A. Pettaway, and Sara S. Strom
M.D. Anderson Cancer Center, University of Texas

P4-5 Altered Drug Sensitivity Profile of MDA PCa 2B, an African American Derived Prostate Carcinoma Cell Line
Aglaia Pappa, Yuka Nakanishi, David J. Kroll, and Nicholas H. Oberlies
Research Triangle Institute

P4-6 Cathepsin B Expression Indicates That Prostate Cancer Is Similar in African American and Caucasian Men
Jenifer L. Morgan, Michael J. Wilson, and Akhouri A. Sinha
University of Minnesota, Twin Cities

P4-7 Different Chromosomal Alterations Correlate with Gene Expression in African American (AA) versus Caucasian American (CA) Prostate Cancer (PC) Patients
Jerome Jean-Gilles[1], Jaya Satagopan[2], Christine Zhou[2], Atreya Dash[2], Jessie Yu[1], Peng Lee[1], William Gerald[2], Iman Osman[1], and Howard Scher[2]
[1]New York University School of Medicine, [2]Memorial Sloan-Kettering Cancer Center

P4-8 The Influence of Patient Race and Social Vulnerability on Urologist Treatment Recommendations in Localized Prostate Carcinoma
Fernando J. Kim[1], Robert C. Flanigan[2], Diane Fairclough[1], Brenda L. Beaty[1], John F. Steiner[1], Richard M. Hoffman[3], and Thomas D. Denberg[1]
[1]University of Colorado Denver, Health Sciences Center, [2]Loyola University, Chicago, [3]University of New Mexico, Albuquerque

P4-9 Trends and Racial Differences in the Utilization of Androgen Deprivation Therapy for Advanced Prostate Cancer
April P. Carson[1], William R. Carpenter[2], Yhenneko B. Jallah[1], Paul A. Godley[2], Daniel L. Howard[1], and Kyna M. Gooden[1]
[1]Shaw University, [2]University of North Carolina at Chapel Hill

P4-10 Racial Trends in Prostate Cancer Incidence Rates for Illinois and the United States, 1986–2000
Katrine Wallace Candidate, Sylvia E. Furner, Faith M. Davis, and Vincent Freeman
University of Illinois, Chicago

P4-11 Telomere Length Polymorphisms: A Potential Factor Underlying Increased Risk of Prostate Cancer in African American Men and Familial Prostate Cancer
Yuko Konishi[1], William B. Isaacs[1], Alan K. Meeker[1], and Elizabeth A. Platz[2]
[1]Johns Hopkins University School of Medicine, [2]Johns Hopkins University, Bloomberg School of Public Health

P4-12 Interrogating Chromosome 12 for Prostate Cancer Susceptibility Genes in African Americans Using an Admixture Mapping Approach
Carolina Bonilla[1] and Rick Kittles[2]
[1]Ohio State University, [2]University of Chicago

Cell Cycle Control

Sep 6 1:00 p.m.-3:00 p.m.
Odd-numbered: 1:00 p.m.-2:00 p.m.
Even-numbered: 2:00 p.m.-3:00 p.m.

P5-1 Disruption of Fibroblast Growth Factor Receptor (FGFR) Signaling as an Approach to Prostate Cancer Therapy
Shantu Dixit, Michael Ittmann, and Mustafa Ozen
Baylor College of Medicine

P5-2 p53 Promotes Prostate Tumor Cell Survival due to the Reversibility of Its Cell Cycle Checkpoints: Implications for Prostate Tumor Responsiveness to Chemotherapy
David Beck, Dana J. Lukin, Luis A. Carvajal, Wen-jun Liu, Lois Resnick-Silverman, and James J. Manfredi
Mount Sinai School of Medicine, New York

Signaling I

Sep 6 1:00 p.m.-3:00 p.m.
Odd-numbered: 1:00 p.m.-2:00 p.m.
Even-numbered: 2:00 p.m.-3:00 p.m.

P6-1 G Protein Signaling in Prostate Cancer Progression
Jae Kim and Yehia Daaka
Medical College of Georgia

P6-2 Role of IKKs and Transcription Factor NF-kappaB in Prostate Tumorigenesis
Alexander Yemelyanov[1], Alexander Gasparian[2], and Irina Budunova[1]
[1]Northwestern University Medical School, [2]Cleveland Biolabs, Inc.

P6-3 Prostate Tumor Suppression by the ERBB4 Receptor Tyrosine Kinase
Richard M. Gallo, Eric E. Williams, Laurie J. Trout, Ianthe N. Bryant, Christopher Mill, Desi J. Penington, and David J. Riese II
Purdue University

P6-4 Mechanisms of HSP90 Inhibition: Implications for Telomere Biology and Prostate Cancer Chemotherapy
Sarah A. Compton[1], Binh N. Nguyen[2], Kimberly Haydu[2], Colleen K. Jackson-Cook[2], Lynne W. Elmore[2], and Shawn E. Holt[2]
[1]University of North Carolina at Chapel Hill, [2]Virginia Commonwealth University

P6-5 Anti-apoptotic Signaling Network in Prostate Cancer
Konduru S. R. Sastry, Adrienne J. Smith, Sergey Prokopovich, Yelena Karpova, and George Kulik
Wake Forest University

P6-6 Prostasin Serine Protease Is a Proteolytic Modulator of EGFR Signaling in the Prostate
Mengqian Chen[1], Li-Mei Chen[1], Chen-Yong Lin[2], and Karl X. Chai[1]
[1]University of Central Florida, [2]Georgetown University

P6-7 Prostate-specific G-Protein Coupled Receptors (PSGRs) in Prostate Cancer Development and Diagnostics
Jinsheng Weng, Xuhong Cheng, Jianhua Wang, Mingyao Liu, and Michael Ittmann
Texas A&M University System Health Sciences Center Research Foundation

P6-8 Role of Tyrosine Kinase C-Kit Receptor and Stem Cell Factor Ligand in Prostate Cancer Proliferation and Apoptosis
LaTonia D. Taliaferro-Smith, Shanti Oyenuga, Soma Sannigrahi, Shafiq A. Khan, and Myron N.V. Williams
Clark Atlanta University

P6-9 K-*Ras* Mutation Leads to Downregulation of Epidermal Growth Factor Receptor (EGFR) Expression, Constitutive Activation of the MAPK/ERK Pathway, and Increased Sensitivity of AKT Activation in Prostate Cancer Cells
Jamil Haider, Soma Sannigrahi, Shafiq A. Khan, and Myron N.V. Williams
Clark Atlanta University

P6-10 Inositol Hexaphosphate Represses Telomerase Activity in Prostate Cancer Cells
Partha P. Banerjee
Georgetown University Medical Center

P6-11 A Functional Epo–Epor Axis Contributes to the Growth and Survival of Prostate Cancer Cells
Jee-Yeong Jeong, Arthur J. Sytkowski, Yuxun Wang, and Laurie Feldman
Beth Israel Deaconess Medical Center, Boston

P6-12 Regulation of Telomerase by the AKT Pathway in Prostate Cancer
Shivani Ruparel, Williams Friedrichs, Richard Montellano, Robert Marciniak, and Linda deGraffenried
University of Texas Health Science Center at San Antonio

P6-13 GSK3/RBL2 Pathway Is an Important Mediator of the Tumor Suppressing Effect of Rapamycin in Prostate Cancer Cells
Rebecca Toddings, James A. DeCaprio, and Larisa Litovchick
Dana-Farber Cancer Institute

P6-14 Crosstalk between p53 and NF-kappaB in Prostate Cancer Cells: Possible Role in Racial Disparity and Clinical Outcome
P. Sankar[1], Q. Yang[1], A.B. Abdel-Mageed[2], and S.P. Kale[1]
[1]Xavier University of Louisiana, New Orleans, [2]Tulane University

P6-15 Functional Analysis of the PTEN Tumor Suppressor Gene
Rosalia Rabinovsky, Ronny I. Drapkin, Jonathan S. Duke-Cohen, and William R. Sellers
Dana-Farber Cancer Institute

P6-16 Herstatin, an Alternative Product of the HER-2 Gene Expressed in the Prostate, Differentially Regulates Insulin-like Growth Factor and Insulin Signaling
Julie M. Carroll, Scott Kuhn, Gail M. Clinton, and Charles T. Roberts, Jr.
Oregon Health & Science University

P6-17 Molecular Studies on Microphage Inhibitory Cytokine (MIC-1) in Prostate Cancer
Siu-Ju Chen, Ajay P. Singh, Sonny L. Johansson, Surinder K. Batra, Ming-Fong Lin, Murielle Meamault, and Kunal Chaudhary
University of Nebraska

P6-18 Expression of p66Shc Protein Correlates with Proliferation of Human Prostate Cancer Cells
Tsukasa Igawa, Ta-Chun Yuan, Fen-Fen Lin, Ming-Shyue Lee, Jamie S. Lin, Sonny L. Johansson, Ming-Fong Lin, and Suresh Veeramani
University of Nebraska Medical Center

P6-19 Involvement of H2 Relaxin in the Progression of Androgen-independent Prostate Cancer
Ralph W. deVere White, Ruth L. Vinall, Shangqin Liu, Clifford Tepper, Xu-Bao Shi, Lynn Xue, Regina Gandour-Edwards, and Hsing-Jien Kung
University of California, Davis

P6-20 Investigating the Differential Roles of FOXO Transcription Factors in Regulating the TRAIL Gene
Ramon Parsons and Megan Keniry
Columbia University College of Physicians and Surgeons

P6-21 Prostate-specific Activation of IKKbetta/NFkappaB in a Transgenic Mouse Prostate Carcinogenesis Model
Tak Wak Mak, Wen-Chen Yeh, and Wen-Jye Lin
University of Toronto

P6-22 Regulation of Cadherin-11 in Cancer Cells
Robert J. Lechleider[1], Stephen W. Byers[2], and Anne K. Farina[2]
[1]National Cancer Institute, [2]Georgetown University

P6-23 Regulation of Protein Tyrosine Kinase 6 in Prostate Cancer Cells
Angela L. Tyner and Patrick M. Brauer
University of Illinois, Chicago

P6-24 P66Shc, A Novel Redox Protein, Regulates Androgen-stimulated Prostate Cancer Cell Proliferation
Ta-Chun Yuan, Fen-Fen Lin, Ming-Fong Lin, and Suresh Veeramani
University of Nebraska Medical Center

P6-25 Cub and Sushi Multiple Domains1 in Prostate Cancer
Aurora Dibner, Levi Garraway, Judit Jane-Valbuena, and William R. Sellers
Dana-Farber Cancer Institute

P6-26 The Two-pore Domain K+ Channel (K2P), TREK-1, Is Overexpressed in Human Prostate Cancer
Iryna Voloshyna, Alessandra Besana, Mahesh Mansukhani, Richard B. Robinson, Steven J. Feinmark, and I. Bernard Weinstein
Columbia University

P6-27 Role of the WW Domain-binding Motif in Regulation of the Transactivation Function of EGR-1 in Prostate Cancer
Anna Reeves[1], Marius Sudol[1], Mark Bedford[2], Mohammed M. Shareef[1], Mohammed Mohiuddin[1], and Mansoor M. Ahmed[1]
[1]Geisinger Clinic, [2]M.D. Anderson Cancer Center, University of Texas

Molecular Mechanism of Prostate Cancer Progression

Sep 6 1:00 p.m.-3:00 p.m.
Odd-numbered: 1:00 p.m.-2:00 p.m.
Even-numbered: 2:00 p.m.-3:00 p.m.

P7-1 Prostate Hypoxia in Patients and the Impact of Androgen Withdrawal: Implications for Disease Progression and Radiation Response
Tony Panzarella, Ants Toi, Joan Sweet, Charles Catton, Padraig Warde, Richard Hill, Robert Bristow, and Michael Milosevic
University Health Network, Toronto

P7-2 STAT5B and Prostate Cancer
Alexander V. Kazansky
Baylor College of Medicine

P7-3 Identifying Candidate Tumor Suppressor Genes for Prostate Cancer Using NMD Inhibition
Maria Johnson, Igor Ivanov, Michael Rossi, and Yurij Ionov
Roswell Park Cancer Institute, Buffalo

P7-4 RSK and Oncogene Addiction
Jeffrey A. Smith, David E. Clark, Josefa Andrade, Michael J. Weber, and Deborah A. Lannigan
University of Virginia

P7-5 Chip-on-Chip Reveals Egr1 Target Genes in Prostate Cancer Cells upon UV Stimulation Using Promoter Arrays
Eileen D. Adamson[1], Yipeng Wang[2], Dan Mercola[1], Shilpi Arora[1], and Michael McClelland[2]
[1]University of California, Irvine, [2]Sidney Kimmel Cancer Center

P7-6 Novel Oncogenic Functions of Delta-Catenin in Prostate Cancer Progression
Larry J. Dobbs, Yan Zeng, Tao Wang, Beverly Jeansonne, Sarah E. James, Yan-Hua Chen, and Qun Lu
East Carolina University

P7-7 Functional Analysis of the Prostate-specific and Androgen-regulated PRLZ Gene
Fray F. Marshall, Haiyen E. Zhau, Leland W.K. Chung, and Ruoxiang Wang
Emory University

P7-8 Prostate-specific Membrane Antigen Drives Prostate Cancer Initiation and Progression in a Tissue Recombinant Model
Dean J. Bacich[1], Veronica Yao[1], Anil Parwarni[1], and Christoph Maier[2]
[1]University of Pittsburgh, [2]Indiana University of Pennsylvania

P7-9 The Helix-Loop-Helix Id-1 Regulates Cell Phenotypes and PSA Expression in Prostate Cancer Cells
Misako Kawahara, Jean-Philippe Coppe, Sylvia Fong, Heidi Feiler, and Pierre-Yves Desprez
California Pacific Medical Center

P7-10 MicroRNA Profile in Prostate Cancer Cells and Response to Androgen Depletion
Yong Sun Lee, Hak Kyun Kim, and Anindya Dutta
University of Virginia

P7-11 Evidence of Decreased NRF2 Expression and Increased ROS Damage in Prostate Cancer
Michael T. McCabe[1], Rebecca Arnold[1], Mark L. Day[2], and Dean A. Frohlich[2]
[1]Emory University, [2]University of Michigan

P7-12 Identification of Human Prostate and Bladder Stromal Factors by Quantitative Transcriptome and Proteomics Analysis
Alvin Y. Liu, David R. Goodlett, and Young Ah Goo
University of Washington

P7-13 Absence of Endogenous Aromatase Activity and Estrogen Results in Reduced Susceptibility to Hormonal Induction of Prostate Malignancy in Adulthood
John S. Pedersen[1], Stephen J. McPherson[2], and Gail P. Risbridger[2]
[1]Tissupath Pty, Ltd., Australia, [2]Monash University

P7-14 Increased Expression and Differential Phosphorylation of Stathmin May Promote Prostate Cancer Progression
Guangyu Gu[1], Erin Tillman[1], Jialing Yuan[1], Yongquing Wang[1], David Friedman[1], Ladan Fazlli[2], Paul S. Rennie[2], Susan Kasper[1], and Ritwik Ghosh[1]
[1]Vanderbilt University, [2]Vancouver General Hospital

P7-15 Proteomic Approach for Identification of Lecithin:Retinol Acyltransferase Interacting Complexes from Mammalian Cells
Lorraine J. Gudas and Moo-Jin Suh
Cornell University, Weill Medical College

P7-16 Genetic Analysis of Prostate Cancer with Kinome-wide RNAi Screens
Ian Dunn[1], Kara Repich[2], David Chun[1], David Root[1], William C. Hahn[1,2], So Young Kim[1,2], and Isil Guney[2]
[1]Broad Institute of MIT and Harvard, [2]Dana-Farber Cancer Institute

P7-17 Hypo-methylation of the Genome Marks a Prostate Cancer Field-defect
William R. Green[1], Christoph Maier[2], Federico A. Monzon[1], and Denise S. O'Keefe[1]
[1]University of Pittsburgh, [2]Indiana University of Pennsylvania

P7-18 Haploinsufficiency of the Maspin Tumor Suppressor Gene Leads to Hyperplastic Lesions in Prostate
Ming Zhang
Northwestern University Feinberg School of Medicine

Biomarkers I

Sep 6 1:00 p.m.-3:00 p.m.
Odd-numbered: 1:00 p.m.-2:00 p.m.
Even-numbered: 2:00 p.m.-3:00 p.m.

P8-1 Molecular Analysis of Androgen-metabolic Genes in Prostate Cancer: Current Successes and Future Needs
Juergen Reichardt
University of Sydney

P8-2 Integrating Functional Genomics and Experimental Models to Identify Targets in Hormone-manipulation Resistant Prostate Cancer
Maria Nieto, David Strochlic, Raanan Berger, William C. Hahn, and Isil Guney
Dana-Farber Cancer Institute

P8-3 Frequent Primary Prostate Tumor Mutations in ABI1/HSSH3BP1 Gene Suggest General Mechanism of Prostate Tumorigenesis Involving C-ABL Tyrosine Kinase
Xiaoling Xiong[1], Yogindra Vedvyas[1], Sajjad Hossain[1], Cyrus Hedvat[2], Guozhen Xu[1], and Leszek Kotula[1]
[1]New York Blood Center, [2]Memorial Sloan-Kettering Cancer Center

P8-4 A Common Deletion at YP11.2 in Prostate Cancer Involves the TSPY Gene
Sapna Vijayakumar, Devon C. Hall, Xavier T. Reveles, Robin J. Leach, Dean A. Troyer, Teresa L. Johnson-Pais, and Susan L. Naylor
University of Texas Health Science Center at San Antonio

P8-5 Frequent Alterations of the ETS Related Gene (ERG) in Prostate Cancer
Gyorgy Petrovics, Chen Sun, Bungo Furusato, Ahmed Mohamed, Chun Ling Gao, Govindan Vaidyanathan, Hongyun Li, Joseph R. Sterbis, Jun Miki, Taduru Sreenath, David G. McLeod, Johng S. Rhim, Isabell A. Sesterhenn, Shiv Srivastava, Albert Dobi, and Jennifer Cullen
Uniformed Services University of the Health Sciences

P8-6 Discovery of Protein Biomarkers for Prostate Cancer in Seminal Plasma by SELDI Mass Spectrometry
Martha K. Terris, James A. Brown, Ronald W. Lewis, and Bao-Ling Adam
Medical College of Georgia

P8-7 Androgen-Regulation of the Bone Marrow Microenvironment Targets IGFBP-5
Chang Xu[1], Lynn F. Graf[1], Ladan Fazli[2], Michael E. Cox[2], Beverly J. Torok-Storb[1], Beatrice S. Knudsen[1], Peter S. Nelson[1], Stephen R. Plymate[3], and Martin Gleave[2]
[1]Fred Hutchinson Cancer Research Center, [2]Vancouver General Hospital, [3]University of Washington

P8-8 Expression Profiling of K-Ras Transformed Human Prostatic Cell Lines
David Téa Okou, K. Sean Kimbro, Jamil Haider, Jodi-Ann Moore, and Myron N.V. Williams
Clark Atlanta University

P8-9 Down Regulation of hZIP1 Zinc Transporter Is a Critical Early Event in Prostate Cancer Development and Progression
Omar Bagasra
Claflin University

P8-10 Characterization of the Phosphoproteome in Prostate Cancer Cells
Francesco Giorgianni, Yingxin Zhao, Bin Fang, and Sarka Beranova-Giorgianni
University of Tennessee Health Science Center

P8-11 Arginase: A Novel Proliferative Determinant in Prostate Cancer
David B. Seligson[1], Stephen D. Cederbaum[1], Anthony E. Pegg[2], Wayne W. Grody[1], and Shannon M. Mumenthaler[1]
[1]University of California, Los Angeles, [2]Pennsylvania State University

P8-12 The Fusion of TMPRSS2: ERG and an Intronic Deletion Is Associated with Hereditary Prostate Cancer
Matthias D. Hofer[1], Rainer Kuefer[2], Sven Perner[1], Christiane Maier[2], Kathleen Herkommer[2], Thomas Paiss[2], Francesca Demichelis[1], Walther Vogel[2], Josef Hoegel[2], Arul M. Chinnaiyan[3], and Mark A. Rubin[1]
[1]Brigham and Women's Hospital/Harvard Medical School, [2]University of Ulm, Germany, [3]University of Michigan Medical School

P8-13 Validation of Method to Identify DNA Markers of Aggressive Prostate Cancer
Nandita Barnabas, Lihua Xu, Adnan Savera, and Evelyn R. Barrack
Henry Ford Health System

P8-14 Elevated Levels of the Mismatch Repair Protein PMS2 Are Associated with Prostate Cancer
Alixanna M. Norris, Ralph D. Woodruff, Ralph B. D'Agostino, Jr., Jill E. Clodfelter, and Karin D. Scarpinato
Wake Forest University Health Sciences

P8-15 Prostate Adenocarcinoma: Candidate Genomic Signatures Prognostic of Recurrent Disease
C. Elisa Oquendo, Priti Lal, Michelle Korenblit, Kurt D'Andrea, Li Ping, Weigen Shang, Phyllis A. Gimotty, S. Bruce Malkowicz, John E. Tomaszewski, and Katherine L. Nathanson
University of Pennsylvania School of Medicine

P8-16 CpG Island Methylation Screening Reveals More Aberrant Methylation in Castration-recurrent Than Androgen-stimulated Prostate Cancer
Srimoyee Ghosh, James Mohler, Dominic J. Smiraglia, and Donald Trump
Roswell Park Cancer Institute, Buffalo

P8-17 Activity of Cyclooxygenase Isoforms in the Murine Prostate
Nicole Janeba, James Loos, and Mark Garzotto
Oregon Health and Science University

P8-18 Recurrent Gene Fusions in Prostate Cancer
Arul M. Chinnaiyan
University of Michigan

P8-19 Expression of Cutaneous Fatty Acid-binding Protein (C-FABP) in Prostate Cancer: Prognostic Significance and Therapeutic Potential
Elwin A. Morgan, Shiva S. Forootan, Janet Adamson, Hiroshi Fujii, Christopher S. Foster, and Youqiang Ke
Liverpool University

P8-20 Role of Human Polyomavirus BKV in Prostate Cancer
Dweepanita Das and Michael J. Imperiale
University of Michigan

P8-21 Glycobiology of Prostate Cancer Cells
Jun Xue, Joseph T.Y. Lau, E.V. Chandrasekaran, Khushi L. Matta, James Mohler, and Gary Smith
Roswell Park Cancer Institute, Buffalo

P8-22 The Wilms' Tumor Suppressor Gene, WT1, Modulates Androgen-induced VEGF Expression in LNCaP Prostate Tumor Cells
Gail Fraizer, Julie Hanson, Kylie Graham, Jennifer Reese, Katie Brown, Jennifer Cash, Jacquelyn Gorman, and Rachel Leahy
Kent State University

P8-23 Telomere DNA Content in Prostate Biopsies Predicts Early Rise in Prostate Specific Antigen Following Radical Prostatectomy for Prostate Cancer
Eric G. Treat[1], Larry Massie[2], Anthony Y. Smith[1], Michael S. Davis[1], Jeffrey K. Griffith[1], Christopher M. Heaphy[1], and Marco Bisoffi[1]
[1]University of New Mexico, Albuquerque, [2]VA Medical Center, Albuquerque, NM

Animal Models

Sep 6 1:00 p.m.-3:00 p.m.
Odd-numbered: 1:00 p.m.-2:00 p.m.
Even-numbered: 2:00 p.m.-3:00 p.m.

P9-1 Roles of Steroid Receptor Coactivator-1 and Transcriptional Intermediary Factor 2 in Androgen Receptor Activity in Mice
Xiangcang Ye[1], Sang Jun Han[2], Sophia Y. Tsai[2], Francesco J. DeMayo[2], Jianming Xu[2], Ming-Jer Tsai[2], and Bert W. O'Malley[2]
[1]University of Texas, M.D. Anderson Cancer Center, [2]Baylor College of Medicine

P9-2 Fibroblast Growth Factor Receptor 2 Tyrosine Kinase Is Required for Prostatic Morphogenesis and Acquisition of Strict Androgen Dependency for Adult Tissue Homeostasis
Yongshun Lin and Fen Wang
Institute of Biosciences and Technology, Texas A&M University System Health Sciences Center

P9-3 The Prostate Cancer Metastasis Gene, KAI1/CD82, Suppresses Tumor Cell Invasion and Metastasis through Regulation of the Receptor Tyrosine Kinase c-Met
Suganthi Sridhar, Sharon Moshkovitz, Kristen M. Saari, Lia Tesfay, Mathew J. Edick, and Cindy K. Miranti
Van Andel Research Institute

P9-4 A Novel Regulatory Pathway for Targeting Invasive Prostate Cancer Identified through Selective Inactivation of Connexin 26 (Gjb2) Gene
Thomas Ott[1], Klaus Willecke[1], and Moulay Alaoui-Jamali[2]
[1]University of Bonn, Germany, [2]Sir Mortimer B. Davis Jewish General Hospital

P9-5 Deletion of Platelet-activating Factor Acetylhydrolase (Phospholipase A2 Group VII) Increases the Severity of Prostate Cancer and Reduces Life Span in the TRAMP Model of Prostate Carcinogenesis
Ethan Reichert, Alison Gardner, Wayne Meikle, Lyska Emerson, and Diana M. Stafforini
University of Utah

P9-6 An Epigenetic Link to Prostate Cancer
Danny Reinberg and Raphael F. Margueron
University of Medicine and Dentistry of New Jersey, Robert Wood Johnson Medical School

P9-7 Transposon-mediated Mutagenesis for Prostate Cancer Gene Discovery
Eric P. Rahrmann, Lara S. Collier, Laura E. Green, David A. Largaespada, and Paul C. Marker
University of Minnesota, Twin Cities

P9-8 STAT3 Activation and Prostate Cancer Progression
Jorge Mario Blando, Manolis Demetriou, Steve Carbajal, Linda Beltran, and John DiGiovanni
University of Texas, M.D. Anderson Cancer Center

P9-9 Establishment and Molecular Characterization of Mouse Xenografts of Organ Confined Human Prostate Tumors
Carmen Priolo, Michelle Agostini, Stephen Finn, Eyoung Shin, Azra Ligon, Diana Donovan, Ewa Sicinska, and Massimo Loda
Dana-Farber Cancer Institute

P9-10 Inflammation and Prostate Cancer Progression
Alex Garcia, Shunyou Wang, and Hong Wu
University of California, Los Angeles

Prostate Development

Sep 6 1:00 p.m.-3:00 p.m.
Odd-numbered: 1:00 p.m.-2:00 p.m.
Even-numbered: 2:00 p.m.-3:00 p.m.

P10-1 Interaction between Bone Morphogenetic Protein 7 (BMP7) and Notch-mediated Progenitor Cell Fate Selection in the Prostate Epithelium. Implications for Prostate Development and Tumorigenesis
Christopher Ferrara[1], Sun Yup Kim[1], Helen Makarenkova[2], Herbert Lepor[1], Irina Grishina[1], and Paul Walden[1]
[1]New York University School of Medicine, [2]Neurosciences Institute

P10-2 The Function of Rex1 in Human Prostate Epithelial Cells
Lorraine J. Gudas and Chunyang Zheng
Cornell University, Weill Medical College

Migration/Invasion/Metastasis

Sep 6 1:00 p.m.-3:00 p.m.
Odd-numbered: 1:00 p.m.-2:00 p.m.
Even-numbered: 2:00 p.m.-3:00 p.m.

P11-1 CXCR5-CXCL13 Expression Regulates Cellular Mechanisms Involved in Prostate Cancer Cell Invasion and Correlates with Prostate Cancer Progression
Rajesh Singh[1], William E. Grizzle[2], Sean K. Kimbro[3], Leland W.K. Chung[3], James W. Lillard, Jr.[1], and Shailesh Singh[1]
[1]University of Louisville, [2]University of Alabama at Birmingham, [3]Emory University

P11-2 Tumor Microenvironment as a Driving Factor for E-Cadherin Loss and Re-expression during Prostate Cancer Metastasis
Clayton Yates, Christopher Shepard, and Alan Wells
University of Pittsburgh

P11-3 Reduced PDEF Ets Transcription Factor Expression Increases Prostate Cancer Cell Invasion and Expression of Mesenchymal Genes
Xuesong Gu[1], Hasan H Otu[1], Manoj Bhasin[1], Quanli Yang[2], Marie G. Joseph[1], Franck Grall[1], Tomi Onatunde[1], Ricardo Correa[3], Towia A. Libermann[1], and Luiz F. Zerbini[1]
[1]Beth Israel Deaconess Medical Center, Boston, [2]National Institute of Family Planning, Beijing, China, [3]Salk Institute

P11-4 Endometase/Mtrilysin-2/Matrix Metalloproteinase-26 as a Putative Biomarker for Early Stage of Human Prostate Cancer
Yonghao Jin[1], Yun-Ge Zhao[1], Hyun I. Park[1], Ai-Zhen Xiao[1], Robert G. Newcomer[1], Tiebang Kang[1], Seakwoo Lee[1], Haiyen E. Zhau[2], Martin A. Schwartz[1], Leland W. K. Chung[2], and Qing-Xiang Amy Sang[1]
[1]Florida State University, [2]Winship Cancer Institute, Emory University

P11-5 Monocyte-Prostate Cancer Cell Intercellular Communication: A New Approach to the Effects of Inflammation on Prostate Cancer Invasion and Progression
Neela Sivapurapu, Yi Lu, Youngsun Hwang, Borko Jovanovic, and Paul F. Lindholm
Northwestern University

P11-6 Androgen-stimulated PAK6 Activation Promotes Prostate Cancer Progression
Steven P. Balk[1], Michael L. Lu[2], and Xin Yuan[2]
[1]Florida Atlantic University, [2]Beth Israel Deaconess Medical Center, Harvard Medical School

P11-7 Characterization of a Novel Protein, Lyric, and Its Potential Role as a Mediator of Prostate Tumor Cell Migration and Invasion
Steven Ash, DongQin Yang, and Deborah E. Britt
Brown University

P11-8 The Role of RDC1/CXCR7 as a Chemokine Receptor for CXCL12 (SDF-1) in Prostate Cancer
Jianhua Wang, Jincheng Wang, Yu Wang, Rohit Mehra, Robert Loberg, Kenneth J. Pienta, and Russell S. Taichman
University of Michigan

P11-9 TGF-Beta Signaling in Prostate Stromal Cells Promotes Prostate Carcinoma Growth by Stimulating Stromal Genes Related to Tissue Remodeling and Growth
Erik V. Verona[1], Abdel Elkahloun[2], Junhua Yang[1], Abhik Bandyopadhyay[1], I-Tien Yeh[1], and LuZhe Sun[1]
[1]University of Texas Health Science Center at San Antonio, [2]National Human Genome Research Institute

P11-10 SOX9 Enhances Prostate Cancer Xenograft Establishment and Invasion
Hongyun Wang[1], Irwin Leav[2], Steven .P Balk[1], Michael L. Lu[3], and Xin Yuan[1]
[1]Beth Israel Deaconess Medical Center, Boston, [2]University of Massachusetts Medical Center, [3]Florida Atlantic University

P11-11 Complete Restoration of Cell Surface Activity of Transmembrane-truncated Membrane-type Matrix Metalloproteinase-1 by a Glycosylphosphatidylinositol Anchor: Implications for MT1-MMP Activity in Cell Invasion in Three-dimensional Matrix
Jing Nie[1], Jing Pei[1], Malcolm Blumenthal[2], and Duanqing Pei[1]
[1]University of Minnesota, Twin Cities, [2]University of Minnesota Medical School

P11-12 Hyaluronan Synthesis and Turnover Induce Metastasis of Prostate Tumor Cells
Alamelu G. Bharadwaj[1], Joy L. Kovar[2], Katherine Metz[1], Eileen Loughman[1], and Melanie A. Simpson[1]
[1]University of Nebraska, [2]LI-COR Biosciences, Inc.

P11-13 A Novel Role of Hedgehog Signaling in Prostate Cancer Tumor Metastasis
Kai Chen, Sumin Chi, Zoran Gatalica, Jingwu Xie, and Xiaoli Zhang
University of Texas Medical Branch, Galveston

P11-14 SLIT2 Suppresses Tumor Development and Metastasis
Hee Kyung Kim[1], Hong Zhang[2], Hui Li[1], Tsung-Teh Wu[2], Stephen Swisher[2], Donggou He[1], Lizhi Wu[1], Craig Elmets[1], Xiaochun Xu[2], and Hui Xu[1]
[1]University of Alabama at Birmingham, [2]M.D. Anderson Cancer Center, University of Texas

P11-15 Truncated Beta 3 Integrins May Represent an Alternative Motility Mechanism in Prostate Cancer
Mohit Trikha, Yinlong Cai, David Grignon, Kenneth V. Honn, and Rongxian Jin
Wayne State University

P11-16 Human Prostate Primary Xenografts: A Model of Human Angiogenesis in an Intact Human Tissue Microenvironment
Alejandro S. Godoy, Viviana Montescinos, and Gary J. Smith
University of North Carolina at Chapel Hill

P11-17 The Molecular and Cellular Mechanism of Prostate Cancer Metastasis Suppressor KAI1/CD82
Xin Zhang
University of Tennessee Health Science Center

P11-18 The Role of the SSECKS/GRAVIN/AKAP12 Gene in Prostate Cancer Progression, Metastasis, and Neovascularization
Bing Su, Yahao Bu, and Irwin H. Gelman
Roswell Park Cancer Institute, Buffalo

P11-19 Receptor Activator of NF-kappaB Ligand (RANKL) Expression, Promoted by Interaction with Bone Microenvironment, Growth Factors, and Snail Transcription Factor Is Associated with Epithelial to Mesenchymal Transition in Human Prostate Cancer Cells
Valerie Odero-Marah
Clark Atlanta University

P11-20 MicroRNAs in Prostate Cancer Tumor Progression
Cheng Lu, Wenzhong Wang, Pamela J. Green, Blake Meyers, Daniel D. Carson, Mary C. Farach-Carson, and Chu Zhang
University of Delaware

P11-21 Macrophage Inhibitory Cytokine-1 Up-regulates Toll Like Receptor 9 Expression in Prostate Cancer Cells
Savita Wakchoure[1], Telisha Millender Swain[1], Xu Feng[1], Kevin W. Harris[1], Samuel Breit[2], and Katri S. Selander[1]
[1]University of Alabama at Birmingham, [2]University of New South Wales

P11-22 LIM Kinase 1 Induced Invasion of Prostate Epithelial Cells Involves Matrix Metalloproteinases and Is Independent of Its Kinase Activity
Tenekua Tapia and Ratna Chakrabarti
University of Central Florida

P11-23 Rap1 Promotes Invasion and Migration in Prostate Cancer Metastasis
Patrick Kelly, Patrick J. Casey, and Candice L. Bailey
Duke University Medical Center

P11-24 The Disintegrin-Metalloproteinase, ADAM15, Supports the Metastatic Progression of Prostate Cancer
Kathleen C. Day, Erin E. Sargent, Casey W. Wright, Mark L. Day, and Abdo J. Najy
University of Michigan

P11-25 Transcription Factor STAT3 Promotes Metastatic Progression of Prostate Cancer
Junaid Abdulghani[1], Lei Gu[1], Jacqueline Lutz[1], Gloria Bonucelli[1], Ayush Dagvadorj[1], Tuomas Mirtti[2], Tapio Visakorpi[3], Lukas Bubendorf[4], and Marja Nevalainen[1]
[1]Thomas Jefferson University, [2]University of Turku, Finland, [3]University of Tampere, [4]University of Basel, Switzerland

P11-26 Cleaved Laminin-alpha5beta1gamma1 Fragment Activates EGFR in Prostate Cancer Cells
Elisabeth L. Bair, G. Tim Bowden, Anne E. Cress, Raymond B. Nagle, and Sangita C. Pawar
University of Arizona, Tucson

P11-27 Mitogen-activated Protein Kinase and Prostate Cancer: Connections through Adhesion
Ashok K. Pullikuth, Evangeline M. McKinnon, Andrew D. Catling, and Electa R. Park
Louisiana State University Health Sciences Center

P11-28 Quantifying the Role of TGF-beta1 in Prostate Cancer Metastasis: Computer Modeling and Experimental Studies
Fayth L. Miles, Seung-Wook Chung, Babatunde Ogunnaike, Carlton Cooper, and Robert A. Sikes
University of Delaware

P11-29 Role of Prostate Specific Membrane Antigen in Suppressing Prostate Cancer Invasion and Metastasis
Angelo Baccala, Kelley M. Harsch, Arundhati Ghosh, and Warren D.W. Heston
Cleveland Clinic Foundation

P11-30 FGFR4 Downregulation of Cell Adhesion in Prostate Cancer
Kristy Drafahl, April N. Meyer, and Daniel J. Donoghue
University of California, San Diego

P11-31 Investigating the Functional Role of Prostate-specific Membrane Antigen and Its Enzymatic Activity in Prostate Cancer Metastasis
Vincent Navarro, He Liu, Neil H. Bander, and Sharron X. Lin
Cornell University, Weill Medical College

P11-32 Focal Degeneration of Aged or Injured Basal Cells and Resultant Auto-immunoreactions Are Trigger Factors for Prostate Tumor Invasion
Yan-gao Man
Nanocrystal Imaging Corporation (NIC)

P11-33 Stromal c-FLIP Function in Prostate Cancer
Yirong Li[1], Huihui Ye[1], Jonathan Melamed[1], Jianjun Wei[1], Peng Lee[1], Michael J. Garabedian[1], Iman Osman[1], Patrice S. Pearce[1], and Zhengxin Wang[2]
[1]New York University School of Medicine, [2]M.D. Anderson Cancer Center, University of Texas

P11-34 Targeting the Epithelial-Mesenchymal Transition in Hormone Refractory Prostate Cancer
Joyce Yamashiro, Scott Hahm, Zhennan Gu, Robert E. Reiter, and Zev A. Wainberg
University of California, Los Angeles

Bone Metastasis

Sep 6 1:00 p.m.-3:00 p.m.
Odd-numbered: 1:00 p.m.-2:00 p.m.
Even-numbered: 2:00 p.m.-3:00 p.m.

P12-1 Hypogonadism Causes Bone Loss and Increased Bone Metastases in a Model of Mixed Osteolytic/Osteoblastic Metastases: Prevention by Zoledronic Acid
Michele Carreon[1], Barry G. Grubbs[1], John C. Chirgwin[2], Theresa A. Guise[2], and Susan S. Padalecki[1]
[1]University of Texas Health Science Center at San Antonio, [2]University of Virginia

P12-2 PSA Regulates Bone Metastases through PTHrP Proteolysis: Preclinical Assay Development
John M. Chirgwin[1], Samuel R. Denmeade[2], and Theresa A. Guise[1]
[1]University of Virginia, [2]Johns Hopkins University School of Medicine

P12-3 Combining Conditional Replication-competent Adenovirus and Integrin-targeting siRNAs to Target Prostate Cancer-Bone Stroma Interaction Effectively Inhibits Tumor Growth in Bone
Kristen Bisanz[1], Leland W. K. Chung[1], Magnus Edlund[1], Chia-Ling

Hsieh[1], and Mien-Chie Hung[2]
[1]Emory University, [2]M.D. Anderson Cancer Center, University of Texas

P12-4 CXCL12/CXCR4 Transactivates Her2 in Lipid Rafts, Induces AKT Activation, MMP-9 Expression in Prostate Cancer Cells and Promotes Growth of Metastatic Deposits in Bone
Sivasakthy Sivalogan, Zhong Dong, R. Daniel Bonfil, Michael L. Cher, and Sreenivasa R. Chinni
Wayne State University

P12-5 Bone Targeting Peptide Binds to Nucleolin, Inhibits Rac-GTPase Signaling and Prostate Cancer Cell Adhesion
Vedavathi Madhu[1], Gina L. Beck[1], Deqing Huang[1], Michael H. Kagey[1], Quanjun Cui[1], Shaun K. Khosla[1], Jay W. Fox[1], Gary Balian[1], and Robert A. Sikes[2]
[1]University of Virginia, [2]University of Delaware

P12-6 Novel Roles for Cathepsin K in Metastasis of Prostate Cancer to the Skeleton
Bruce E. Linebaugh, Deborah L. Rudy, Mary B. Olive, Kamiar Moin, Izabela Podgorski, and Bonnie F. Sloane
Wayne State University School of Medicine

P12-7 Bone Remodeling Sites Are Preferred Targets for Metastatic Colonization by Prostate Cancer Cells
Lauren J. Silbert[1], Mitchell B. Schaffler[1], Irwin H. Gelman[2], and Robert J. Majeska[1]
[1]Mount Sinai School of Medicine, New York, [2]Roswell Park Cancer Institute, Buffalo

P12-8 Potential Roles for RHOC and Rac GTPases in Prostate Cancer Bone Metastasis
Christopher Hall[1], Robert Loberg[1], Linda Sequeira[2], Kenneth L. Van Golen[2], and Carlton Cooper[2]
[1]University of Michigan, [2]University of Delaware

P12-9 Effects of Cyclooxygenase-2 and Prostaglandin E2 on Prostate Cancer Bone Metastases
Xin-Hua Liu and Alice C. Levine
Mount Sinai School of Medicine, New York

P12-10 The Role of DKK-1 in Prostate Cancer Bone Metastases
Zhi Gang, Li Jun Yang, Diana Rose, Funda Vakar-Lopez, Paul Mathew, Adriana Lopez, Christopher J. Logothetis, Sue-Hwa Lin, and Nora M. Navone
M.D. Anderson Cancer Center, University of Texas

P12-11 The Role of Interleukin-6/GP130 Signaling in Prostate Cancer Progression and Its Contribution to Bone Metastasis Morbidity
Daniel R. McCulloch, Elizabeth D. Williams, Richard P. Redvers, and Erik W. Thompson
University of Melbourne

P12-12 Anti-hypoxic Inhibition of Prostate Cancer Bone Metastases
Khalid S. Mohammad[1], Vu Dong[1], Lauren A. Kingsley[1], Larry J. Suva[2], John M. Chirgwin[1], and Theresa A. Guise[1]
[1]University of Virginia, [2]University of Arkansas for Medical Sciences

P12-13 Induction of Osteogenesis in Osseous and Non-osseous Sites by an Androgen Receptor-negative Human Prostate Cancer Xenograft
Paul Mathew, Zhi Gang Li, Jie Liu, Jun Yang, Michael W. Starbuck, Charles Sikes, Asha S. Multani, Jing Wang, Tina V. Fanning, Victor G. Prieto, Patricia Troncoso, Austin K. Raymond, Sue-Hwa Lin, and Nora M. Navone
M.D. Anderson Cancer Center, University of Texas

Mechanisms of Resistance

Sep 6 1:00 p.m.-3:00 p.m.
Odd-numbered: 1:00 p.m.-2:00 p.m.
Even-numbered: 2:00 p.m.-3:00 p.m.

P13-1 Acquired TRAIL Resistance in Prostate Cancer: Side Effects of TRAIL-induced Anti-apoptotic Nature
Jee Y. An, Yong T. Kwon, Yong J. Lee, and Jae J. Song
University of Pittsburgh School of Medicine

P13-2 Targeting Mechanisms of Resistance to Taxane-based Chemotherapy
Celestia Higano[1], Paul H. Lange[2], Lawrence True[2], Tomasz M. Beer[1], Mark Garzotto[1], Chung-Ying Huang[2], Peter S. Nelson[2], and Robert Vessella[2]
[1]Oregon Health & Science University, [2]Fred Hutchinson Cancer Research Center

Preclinical Therapeutics

Sep 6 1:00 p.m.-3:00 p.m.
Odd-numbered: 1:00 p.m.-2:00 p.m.
Even-numbered: 2:00 p.m.-3:00 p.m.

P14-1 An Efficient Synthesis of Bullatacin and Related Analogs
Hongda Zhao[1], Jeffrey S.T. Gorman[1], and Brian L. Pagenkopf[1,2]
[1]University of Texas at Austin, [2]University of Western Ontario

P14-2 Cellular Uptake and Tissue Distribution of an RNA Aptamer to Prostate-specific Membrane Antigen
Liuqing Yang[1], Amy B. Foraker[1], Peter W. Swaan[2], and Thomas D. Schmittgen[1]
[1]Ohio State University, [2]University of Maryland School of Pharmacy

P14-3 PAM Inhibitors
Jeffrey Sarver, Wieslaw Klis, Mugunthu Dhananjeyan, Jill Trendel, Nicole Ellis, Crystal Bykowski, Ritesh Mittal, Jidong Liu, Rahul Khupse, Mohammad El-Dakdouki, and Paul Erhardt
University of Toledo

P14-4 Cytotoxic Paclitaxel Analogues for Conjugation to Targeted Scaffolds as Novel Prostate Cancer Therapeutics
Waldemar Priebe, Jim Klostergaard, and Dean Tang
M.D. Anderson Cancer Center, University of Texas

P14-5 Ying Yang 1 (YY1) and Raf Kinase Inhibitor Protein (RKIP) Regulate TRAIL Sensitivity and Are Prognostic Markers in Prostate Cancer
Sara Huerta-Yepez, Mario I. Vega, Alina Katsman, Stavroula Baritaki, and Benjamin Bonavida
David Geffen School of Medicine, Jonsson Comprehensive Cancer Center, University of California, Los Angeles

P14-6 Development of an Integrated System with Feedback Control for Interstitial Photodynamic Therapy
Jun Li, Xiaodong Zhou, Jarod C. Finlay, Andreea Dimofte, Martin D. Altschuler, Gary Kao, Bruce S. Malkowicz, Keith A. Cengel, Neha

Vapiwala, Stephen M. Hahn, and Timothy C. Zhu
University of Pennsylvania

P14-7 Cytotoxicity of Lipoxygenase Inhibitors toward Prostate Cancer Cells
Renshu Zhang, Tiffiney Greer, Alemayehu Kassa, Yanfei Zhou, Xinbin Gu, Ebrahim Ashayeri, and Rajagopalan Sridhar
Howard University, Washington

P14-8 Indole-3-Acetic Acid Analogues as RXR Ligands and Effects on Prostate Cancer Cells
Mao Ye, Qiong Ying Hu, Zebin Xia, Xihua Cao, Marcia I. Dawson, and Xiao-kun Zhang
Burnham Institute

P14-9 Discovery of Antiandrogen Activity of Nonsteroidal Scaffolds of Marketed Drugs
W.H. Bisson, N. Bruey-Sedano, J. Chen, N. Goldberger, L.T. May, A. Christopolous, J.T. Dalton, P.M. Sexton, X.K. Zhang, R. Abagyan, and A.V. Cheltsov
Scripps Research Institute

P14-10 Inhibition of Tumor Growth and PSA Secretion by Selenite in the LAPC-4 Human Prostate Cancer Xenograft Model
Bryan Husbeck, Rumi S. Bhattacharyya, David Feldman, and Susan J. Knox
Stanford University Medical Center

P14-11 Enhancing the Efficacy of Beta-lapachone in Prostate Cancer Therapy Using Millirod Delivery System
Ying Dong, Shook-Fong Chin, Wareef Kabbani, Elvin Blanco, Erik A. Bey, Jinming Gao, and David A. Boothman
University of Texas Southwestern *Medical Center at Dallas*

P14-12 X-ray Crystal Structures of ErbB2 Tyrosine Kinase Inhibitors (TKIs)
Cheryl L. Klein Stevens, Peter Tran, and Naijue Zhu
Xavier University, Louisiana

P14-13 A Genetic Screen to Identify Novel Therapeutic Targets in Hormone Refractory Prostate Cancer
Nicola J. Clegg[1], David H. Leung[1], Chris Tran[1], Thomas F. Westbrook[2], Stephen J. Elledge[2], Jennifer C. King[1], and Charles L. Sawyers[1]
[1]Memorial Sloan-Kettering Cancer Center, [2]Harvard Medical School and Howard Hughes Medical Institute

P14-14 Sensitizing Hormone-refractory Prostate Cancer Cells to Drug Treatment by Targeting 14-3-3Sigma Using Structure-based Drug Design
Baoguang Han, Hui Peng, Zhaomin Li, Han Xie, Qun Chen, and Jian-Ting Zhang
Indiana University School of Medicine

P14-15 Chemical Ablation of Androgen Receptor in Prostate Cancer Cells with Histone Deacetylase Inhibitors
Jie Wu[1], Liwei Chen[1], Hai Wang[1], Songshu Meng[1], Kapil B. Bhalla[1], and Peter Atadia[2]
[1]H. Lee Moffitt Cancer Center & Research Institute and University of South Florida, [2]Novartis Institutes of Biomedical Research

P14-16 Discovery of Natural Product-derived Small Molecules That Inhibit Hypoxia-inducible Factor-1 (HIF-1) Activation in Prostate Tumor Cells
Yu-Dong Zhou, Yang Liu, and Dale G. Nagle
University of Mississippi

P14-17 Development of the C-terminal Inhibitors of Heat Shock Protein 90 in the Treatment of Prostate Cancer
Chris Avila[1], George Vielhauer[1], Brian Blagg[2], and Jeffrey M. Holzbeierlein[1]
[1]University of Kansas Medical Center, Kansas City, [2]University of Kansas, Lawrence

P14-18 Synthesis of Apoptolidin Analogs as Potential Therapeutics for the Treatment of Prostate Cancer
Gary A. Molander and Elizabeth A. Jurica
University of Pennsylvania

P14-19 Total Synthesis of Nemorosone, A Natural Product Displaying Selective Cytotoxicity against Prostate Cancer Cells
Tsukano Chihiro, Samuel J. Danishefsky, and Dionicio R. Siegel
Memorial Sloan-Kettering Cancer Center

P14-20 Design of DNA Alkylating Agents That Block DNA Repair and Disrupt Cancer-specific Cellular-signaling Programs
John M. Essigmann and Robert G. Croy
Massachusetts Institute of Technology

P14-21 Computational Modeling and Experimental Evaluation of a Novel Radioactive Prodrug for Targeting the Extracellular Space of Prostate Tumors
Pavel Pospisil, Ketai Wang, Ayman F. Al Aowad, Yongliang Yang, Houari Korideck, S. James Adelstein, and Amin I. Kassis
Harvard Medical School

P14-22 A Novel Vitamin D Compound for Prostate Cancer
James Lambert[1], Sibaji Sarkar[2], Kelly S. Persons[2], and Rahul Ray[2]
[1]University of Colorado Denver, Health Sciences Center, [2]Boston University School of Medicine

P14-23 RNA Isolation for Prostate Cancer Targeting
Andrej Luptak[1], Frank Alexis[2], Benjamin A. Teply[2], Judy Cheng[2], Jack W. Szostak[1], Robert Langer[2], Omid C. Farokhzad[2], and Etgar Levy-Nissenbaum[2]
[1]Massachusetts General Hospital, [2]Brigham and Women's Hospital

P14-24 Preclinical Development and Mechanistic Evaluation of Parthenolide and Its Water Soluble Analogue, Dimethylaminoparthenolide (DMAPT) as a New Treatment for Prostate Cancer
Rajasubramaniam Shanmugam[1], Praveen Kusumanchi[1], Marc Mendonca[1], Peter Crooks[2], Harikrishna Nakshatri[1], and Christopher Sweeney[1]
[1]Indiana University, Indianapolis, [2]University of Kentucky

P14-25 Structural Studies of the PAR-4 Protein
Gabriel Birrane, Michael Durney, Aditi Soni, and John A.A. Ladias
Beth Israel Deaconess Medical Center, Boston

P14-26 Progress in the Synthesis of Taxol-like Chemotherapeutic Agent for Prostate Cancer
Hyunil Jo and Jeffrey D. Winkler
University of Pennsylvania

P14-27 Testing Candidate Novel Therapies in a New Model of Osteosclerotic Bone Metastasis
Elizabeth D. Williams[1], Christine L. Chaffer[1], Dhanya Sreedharan[1], Nigel Brooks[2], Timothy P. Green[2], and Erik W. Thompson[3]
[1]Monash Institute of Medical Research, Australia, [2]Astra Zeneca, [3]University of Melbourne

P14-28 Development of Cytotoxamers Targeting PSMA for Treatment of Prostate Cancer
Robert E. Hamlin, Jr., McKnight Garner, Sarah F. Quinlan, and William H. Gmeiner
Wake Forest University School of Medicine

Gene Therapy

Sep 6 1:00 p.m.-3:00 p.m.
Odd-numbered: 1:00 p.m.-2:00 p.m.
Even-numbered: 2:00 p.m.-3:00 p.m.

P15-1 Targeted BikDD Expression for Curing Both Androgen-dependent Prostate Cancer (ADPC) and Androgen-independent Prostate Cancer (AIPC) in Xenograft and Transgenic Models
Weiya Xia, Mien-Chie Hung, and Xiaoming Xie
M.D. Anderson Cancer Center, University of Texas

P15-2 Development of Genetically Modified Adenoviruses, Cancer Terminator Viruses, to Treat Resistant and Metastatic Human Prostate Tumors
Devanand Sarkar, Irina V. Lebedeva, Zao-zhong Su, and Paul B. Fisher
Columbia University

P15-3 Ad5-TRAIL Gene Therapy for Prostate Cancer
Thomas S. Griffith
University of Iowa

P15-4 Potent Antitumor Activity of a Novel Oncolytic Herpes Simplex Virus against Orthotopic and Metastatic Prostate Cancer
Mikihito Nakamori, Xinping Fu, and Xiaoliu Zhang
University of Texas Medical Branch, Galveston

P15-5 Anti-tumor and Radio-sensitization Activity of Ad-U2, a Prostate-specific Replication-competent Adenovirus Armed with TRAIL, in a Tri-modal Therapy for High-risk Prostate Cancer
Yousef Mohammadi, Kyung Hee Bae, Yan-Ping Zhang, Xiong Li, Matthew Mellon, Thomas A. Gardner, Chinghai Kao, and Juan A. Jimenez
Indiana University-Purdue University, Indianapolis

P15-6 Efficient, Specific Propagation of a Transcriptionally Targeted Oncolytic Adenovirus to Treat Prostate Cancer
Makoto Sato[1], Russell Powell[1], Michael Carey[1], Sanjiv S Gambhir[2], and Lily Wu[1]
[1]David Geffen School of Medicine, University of California, Los Angeles, [2]Stanford University School of Medicine

P15-7 Safe, Focused Delivery of Viral Vectors to Target Tumors Using Virus-Microbead Conjugates for Gene Therapy of Cancer
Alan Jerusalmi, Samuel J. Farlow, Mark W. Pandori, and Takeshi Sano
Beth Israel Deaconess Medical Center, Boston

P15-8 A Signal-Smart Oncolytic Herpes Virus for Targeting Prostate Cancer
Faris Farassati, Weihong Pan, Farnaz Yamoutpoor, and Tuba Esfandyari
University of Minnesota, Twin Cities

P15-9 Early Growth Gene Response-1 Functional Activation, Signaling and Radiation Response in Prostate Cancer
Marianna Sultanov-Zagurovskaya[1] and Mansoor M. Ahmed[2]
[1]University of Kentucky, [2]University of Kentucky Research Foundation, Inc.

P15-10 Systemic Delivery of AAV-based AR siRNAs Eradicate Prostate Cancer Xenografts in Nude Mice
Yan Hong, J. Brantley Thrasher, and Benyi Li
University of Kansas Medical Center, Kansas City

P15-11 Targeted Eradication of Prostate Cancer Mediated by Engineered Mesenchymal Stem Cells
Luhong Sun, Peilin Zhao, and Yan Cui
Louisiana State University Health Sciences Center

P15-12 Engineering Polypeptide-coated Adenoviral Gene Delivery Vectors for Systemic Therapy of Prostate Cancer
Sok Boon Shuwen Koh, Timothy Deming, and Lily Wu
University of California, Los Angeles

P15-13 Development of a Gene Therapy Trial for Metastatic Prostate Cancer
Xhong Li, Thomas A. Gardner, Chinghai Kao, and Juan A. Jiménez
Indiana University-Purdue University, Indianapolis

Novel Therapies

Sep 6 1:00 p.m.-3:00 p.m.
Odd-numbered: 1:00 p.m.-2:00 p.m.
Even-numbered: 2:00 p.m.-3:00 p.m.

P16-1 Peroxisome Proliferator-activated Receptor-delta Antagonism as a Therapeutic Strategy for Prostate Cancer
Nurulain T. Zaveri, Barbara G. Sato, Faming Jiang, and Brian J. Murphy
SRI International

P16-2 Anticancer Activity of G-Rich Oligonucleotides That Target Nucleolin
John O. Trent, Damian A. Laber, William M. Pierce, Jr., Jon B. Klein, Shirish S. Barve, Yun Teng, Allicia C. Girvan, Simone Juliger, Lavona K. Casson, Paula J. Bates, and Donald M. Miller
University of Louisville Research Foundation, Inc.

Radiation Therapy

Sep 6 1:00 p.m.-3:00 p.m.
Odd-numbered: 1:00 p.m.-2:00 p.m.
Even-numbered: 2:00 p.m.-3:00 p.m.

P17-1 Enhanced Cell Killing by Very Low Dose-rate Irradiation: Possibilities for Better Prostate Cancer Radiotherapy
Sarah A. Krueger[1], George D. Wilson[1], Brian Marples[2], and Michael C. Joiner[1]
[1]Wayne State University, [2]Karmanos Cancer Institute

P17-2 Dosimetric Characteristics of a Newly Designed RadioCoil™ ^{103}Pd Sources for Prostate Interstitial Implant
Ali Soleimani-Meigooni, Shahid Bashir Awan, Sharifeh Azam Dini, and Kai Dou
University of Kentucky

P17-3 Antigen-independent Methods to Improve Radioimmunotherapy of Prostate Cancer
Janina Baranowska-Kortylewicz, Michio Abe, Jessica Nearman, R. Lee Mosley, Gabriela Pavlinkova, and Charles A. Enke
University of Nebraska

P17-4 Adaptive IMRT for Improved Prostate Cancer Treatment
Adam de la Zerd, Ming Chao, Benjamin Armbrush, Yong Yang, Steve Hancock, Christopher King, Tianfang Li, and Lei Xing
Stanford University School of Medicine

P17-5 Curcumin, a Potent Radiosensitizer for Prostate Cancer
Chendil Damodaran
University of Kentucky

P17-6 Interaction of Isoflavones and Vitamin E with Irradiation in Androgen Independent PC-3 Human Prostate Cancer Cells
Lori Rice, Renita Handayani, Theresa Medrano, Yamil Selman, Brandon Mauldin, Charles J. Rosser, and Kathleen T. Shiverick
University of Florida

P17-7 Dose Escalation of Dominant Intra-prostatic Lesion Defined by Magnetic-resonance Spectroscopy Imaging Using Inverse Planning for HDR Prostate Brachytherapy
I-Chow Hsu, Etienne Lessard, Yongbok Kim, Susan Moyher Noworolski, John Kurhanewicz, and Jean Pouliot
University of California, San Francisco

P17-8 A Genetically Determined Dose Volume Histogram Predicts for Rectal Bleeding among Patients Treated with Prostate Brachytherapy
Richard G. Stock, David P. Atencio, Sheila Peters, Christopher A. Peters, Ryan J. Burri, Nelson N. Stone, Barry S. Rosenstein, and Jamie A. Cesaretti
Mount Sinai School of Medicine, New York

P17-9 Radiosensitizing Effects of Histone Deacetylase Inhibitors in Prostate Cancer Cell Lines: Identification of Novel Mechanisms
Ching-Shih Chen[1], Seema Gupta[2], and Mansoor M. Ahmed[2]
[1]Ohio State University, [2]Geisinger Clinic

P17-10 Early Results of a Feasibility Study of Registered Fluoroscopy and Ultrasound for Permanent Interstitial Prostate Brachytherapy
Anton Deguet, Yi Le, Jack Blevins, Iulian Iordachita, E. Clif Burdette, Gabor Fichtinger, Elwood Armour, Ameet Jain, and Danny Song
Johns Hopkins University School of Medicine

P17-11 PARP-1 Hyperactivation Mediates Synergy between Beta-lapachone and Ionizing Radiation in Human Prostate Cancer Cells That Express Endogenously Elevated NQO1 Levels
Ying Dong, Erik A. Bey, Melissa Bentle, Kathryn Reinicke, and David A. Boothman
Case Western Reserve University

P17-12 Late Tissue Effects Following Radiotherapy of the Prostate Measured with Quantitative Magnetic Resonance Imaging
Lucy E. Kershaw[1], Charles E. Hutchinson[1], Noel W. Clarke[2], John P. Logue[2], and David L. Buckley[1]
[1]University of Manchester, Manchester, United Kingdom, [2]Christie Hospital, Manchester, United Kingdom

Nutrition and Prostate Cancer

Sep 7 12:30 p.m.-2:30 p.m.
Odd-numbered: 12:30 p.m.-1:30 p.m.
Even-numbered: 1:30 p.m.-2:30 p.m.

P18-1 Vitamin D Metabolism and Its Implications for Prostate Cancer Chemoprevention
Tai C. Chen
Boston University Medical Campus

P18-2 Regulation of Prostate Gene Expression, Prostate Growth and Prostate Tumorigenesis by Dietary Fat and Phytochemicals
Li-Qun Cai[1], Wendy Wu[1], Jianyou Tan[2], Julianne Imperato-McGinley[1], and Yuan-Shan Zhu[1]
[1]Cornell University, Weill Medical College, [2]Bio-Reference Laboratories, Inc.

P18-3 Reactivation of Silenced Pi-class Glutathione S-Transferase Gene and Repression of Androgen Receptor by an Isothiocyanate
L.G. Wang[1], X. M. Liu[2], J. Feng[1], D. Liu[1], and J. W. Chiao[1]
[1]New York Medical College, [2]New York University

P18-4 Androgen Signaling Axis as Targets of Selenium Anticancer Action
Shuang Liu and Yan Dong
Roswell Park Cancer Institute, Buffalo

P18-5 The Citrus Flavonoid Naringenin Stimulates DNA Repair in Prostate Cancer Cells
Kun Gao[1], Anlong Xu[2], David Heber[3], and Susanne M. Henning[1]
[1]University of California, Los Angeles, [2]Sun Yat-sen University, [3]David Geffen School of Medicine, University of California, Los Angeles

P18-6 Selenium Molecular Mechanisms in Prostate Cancer: Regulation of the Tumor Suppressor p53
Sivalokanathan Sarveswaran, Sathish Sundaram, Joshua Liroff, and Jagadananda Ghosh
Henry Ford Health System

P18-7 Effects of Dietary Saw Palmetto on the Prostate of Transgenic Adenocarcinoma of the Mouse Prostate Model (TRAMP)
Teri L. Wadsworth[1], Teresa R. Worstell[1], Norman M. Greenberg[2], and Charles E. Roselli[1]
[1]Oregon Health and Science University, [2]Fred Hutchinson Cancer Research Center

P18-8 Curcumin Regulates FOXO Transcription Factor Activity and Apoptosis in Prostate Cancer
Sharmila Shankar, Suthakar Ganapathy, Quinghe Chen, and Rakesh K. Srivastava
University of Texas Health Center at Tyler

P18-9 Long-term Treatment with Vitamin D Selects for Prostate Cancer Cells with Altered Antiproliferative and Gene Transcriptional Response
Guangzhou Han, Michael T. Moser, Candace S. Johnson, Adebusola A. Alagbala, Barbara A. Foster, and Donald L. Trump
Roswell Park Cancer Institute, Buffalo

P18-10 Dietary Influences on Alpha-Methylacyl-CoA Racemase (AMACR) Expression in the Prostate
Ryan J. Deaton, Richard Van Breemen, Gayatri Borthakur, Erika Enk, Peter H. Gann, and Vijayalakshmi Ananthanarayanan
University of Illinois at Chicago

P18-11 The Dietary Charred Meat Carcinogen PhIP Causes Inflammation and Atropy in the Rat Prostate prior to the Development of Prostatic Intraepithelial Neoplasia
Y. Nakai, W.G. Nelson, G. Palapattu, and Angelo M. DeMarzo
Johns Hopkins University School of Medicine

P18-12 Paracrine Factors from Mouse Bone Marrow Cells Inhibit the Growth of Human Prostate Cell Lines, but This Is Modulated by Effects of Dietary Fat and Cytochrome P4501B1
Michele C. Larsen and Colin R. Jefcoate
University of Wisconsin, Madison

P18-13 A No-carbohydrate Diet Significantly Delays Prostate Cancer Growth in an Animal Model
John Mavropoulos[1], Amy Wang[2], Medha Darshan[2], William Aronson[3], David Hwang[3], Bercedis Peterson[1], Timothy Fields[1], Salvatore Pizzo[1], Pinchas Cohen[3], Stephen J. Freedland[1], William B. Isaacs[2], and Wendy Demark-Wahnefried[1]
[1]Duke University Medical Center, [2]Johns Hopkins University School of Medicine, [3]University of California, Los Angeles

P18-14 Selenium Inhibits Growth of LNCaP Human Prostate Tumor Accompanied by a Decrease in the Expression of Androgen Receptor and Prostate-specific Antigen (PSA)
Soo Ok Lee, Jae Yeon Chun, Nagalakshmi Nadiminty, and Allen C. Gao
Roswell Park Cancer Institute, Buffalo

P18-15 Neurotensin Growth Signaling Involves the PKC and Lipoxygenase Pathways, and NT Receptor Function Is Subject to Feedback Regulation
Sazzad Hassan, Paul R. Dobner, and Robert E. Carraway
University of Massachusetts Medical School

P18-16 Modulation of Prostate Cancer Genetic Risk by Omega-3 and Omega-6 Fatty Acids
Isabelle M. Berquin, Younong Min, Ruping Wu, Jiansheng Wu, Donna Perry, J. Mark Cline, Mike J. Thomas, Todd Thornburg, Adrienne Smith, Iris J. Edwards, Yong Q. Chen, and George Kulik
Wake Forest University

P18-17 Dietary Modulation of Polyunsaturated Fatty Acids in Prostate Cancer Affects Response to Hormone Ablation Therapy
Carol Ziegler[1], Xiaoou Li[1], Danielle Reel[1], Ahmed Shoieb[1], Kenneth Tomer[2], Jay Whelan[1], and Michael F. McEntee[1]
[1]University of Tennessee, Knoxville, [2]National Institute of Environmental Health Sciences

Lifestyle

Sep 7 12:30 p.m.-2:30 p.m.
Odd-numbered: 12:30 p.m.-1:30 p.m.
Even-numbered: 1:30 p.m.-2:30 p.m.

P19-1 Centralized Obesity, a Component of the Metabolic Syndrome, Is Associated with Increased Risk of High-grade Prostatic Intraepithelial Neoplasia (PIN)
Saundra S. Motley, Marcia Wills, Michael S. Cookson, Raoul S. Concepcion, Sam S. Chang, Joseph A. Smith Jr., and Jay H. Fowke
Vanderbilt University Medical Center

P19-2 Obesity as a Predictor of Prostate Cancer Mortality in Puerto Rican Men
Ellen Smit[1], Mario R. Garcia-Palmieri[2], Nayda Figueroa-Valle[2], Jo Freudenheim[1], Barbara Fuhrman[1], I-Min Lee[3], and Carlos J. Crespo[1]
[1]State University of New York, Buffalo, [2]University of Puerto Rico, San Juan, [3]Harvard University, Cambridge

P19-3 Lifestyle Behaviors in White and African American Prostate Cancer Survivors: A Qualitative Study
Jessie A. Satia
University of North Carolina at Chapel Hill

P19-4 Elevated Prostate-specific Antigen in African American Men with High Meat-carcinogen Intake: A Prospective Clinic-based Study
Kenneth T.Bogen[1], Leslie J. Paine[2], Ernest L. Simms[2], Elizabeth A. Holly[3], June Chan[3], James S. Felton[1], and Garrett A. Keating[1]
[1]Lawrence Livermore National Laboratory, [2]Alta Bates Summit Medical Center, University of California, San Francisco

P19-5 Role of Obesity at Different Ages in Prostate Cancer Development in TRAMP Mice
Melissa J.L. Bonorden, Olga P. Rogozina, Nancy K. Mizuno, and Margot P. Cleary
Hormel Institute, University of Minnesota

Complementary and Alternative Medicine

Sep 7 12:30 p.m.-2:30 p.m.
Odd-numbered: 12:30 p.m.-1:30 p.m.
Even-numbered: 1:30 p.m.-2:30 p.m.

P20-1 Isolation and Characterization of Anti-angiogenic Constituents of PC-SPES, a Clinically Effective Therapy for Advanced Prostate Cancer
A.A. Leslie Gunatilaka[1], Luke Whitesell[1], Linda Meade-Tollin[1], E.M. Kithsiri Wijeratne[1], Vanimireddy L.N. Reddy[1], Deborah Cooper[1], Mischa Guild[1], Edlyn Jon[1], Marilyn T. Marron[1], Anna M. Burns[1], Manping X. Liu[1], and Jing-yu Liang[2]
[1]University of Arizona, Tucson, [2]China Pharmaceutical University, Nanjing

P20-2 A Diet, Physical Activity, and Meditation Intervention in Men with Rising Prostate-specific Antigen (PSA)
Thomas G. Hurley[1], Jamie Ritchey[1], Brook E. Harmon[1], Philip P. Cavicchia[1], Elizabeth A. Fallon[2], Wendy B. McKenzie[1], Linzhi Xu[1], Sue Heiney[1], and James R. Hebert[1]
[1]University of South Carolina, [2]Kansas State University

P20-3 Phytoestrogens Inhibit Hedgehog Signaling in Prostate Cancer Cell Lines
Anna Slusarz, Nader Shenouda, Mary S. Sakla, Byron J. Bernabe, Katherine M. Beck, Charles A. Parker, Cynthia L. Besch-Williford, and Dennis B. Lubahn
University of Missouri, Columbia

Chemoprevention

Sep 7 12:30 p.m.-2:30 p.m.
Odd-numbered: 12:30 p.m.-1:30 p.m.
Even-numbered: 1:30 p.m.-2:30 p.m.

P21-1 Resveratrol Suppresses Poorly Differentiated Prostate Cancer in Transgenic Mice
Curt E. Harper, Brijesh B. Patel, Jun Wang, Isam A. Eltoum, and Coral A. Lamartiniere
University of Alabama at Birmingham

P21-2 Mono-methyl Selenium: Superior Second-generation Agents Than Selenomethionine for Prostate Cancer Chemoprevention
Guang-Xun Li, Hyo-Jeong Lee, Zhe Wang, Hongbo Hu, Lei Wang, Cheng Jiang, Sung-Hoon Kim, Jennifer Watts, Gerald F. Combs Jr., and Junxuan Lü
University of Minnesota, Austin

P21-3 Boron Inhibits Prostate Cancer Cell Proliferation in the Laboratory and Reduces the Risk of Prostate Cancer in Texas
Wade T. Barranco, Danny H. Kim, Kimberly A. Henderson, and Curtis D. Eckhert
University of California, Los Angeles

P21-4 Silibinin Inhibits Growth of PC-3 Prostate Tumor Xenograft Involving Upregulation of Cyclin-dependent Kinase Inhibitors and IGFBP-3, and Downregulation of Survivin
Gagan Deep, Rajesh Agarwal, and Rana P. Singh
University of Colorado Health Sciences Center, Denver

P21-5 Molecular Role of I3C/DIM in Prostate Cancer Cells
Fazlul H. Sarkar and Yiwei Li
Wayne State University

P21-6 Complementary Roles in Cancer Prevention: Soy Protease Inhibitors Protect the Chemopreventive Lunasin Peptide from Digestion and Make It Bioavailable
Ben O. de Lumen[1], Hyun J. Jeong[1], Jae Ho Park[2], Chang-su Lim[3], Terri Sutherland-Bozzo[1], Mark Fitch[1], and Gurpreet Ratra[4]
[1]University of California, Berkeley, [2]Andong University, Korea, [3]Virginia Polytechnic Institute and State University, [4]Northview Pacific Laboratories, Inc.

P21-7 Prostate Cancer Cell Proliferation Inhibition and Alterations in Retinol Esterification Induced by Phytanic Acid and Docosahexaenoic Acid
Lorraine J. Gudas, Rong Li, Moo-Jin Suh, and Xiao-Han Tang
Cornell University, Weill Medical College

P21-8 Antioxidant Prophylaxis for Prostatic Intraepithelial Neoplasia
Addanki P. Kumar[1], Nicole E. Arevalo[2], Gretchen E. Garcia[1], Keya De[1], Maxwell L. Smith[2], M. Scott Lucia[2], Daniel E. Chan[2], and Rita Ghosh[1]
[1]University of Texas Health Science Center, San Antonio, [2]University of Colorado Health Sciences Center, Denver

P21-9 Prostate Cancer Prevention by Resveratrol: Purification and Analysis of Cellular Protein Targets of Resveratrol Using a Ligand-captured Bioaffinity Strategy
Joseph M. Wu
New York Medical College

P21-10 Molecular Targets of N-3 PUFAs for Prostate Cancer Prevention
Yuan Qiao and Huseyin Aktas
Harvard Medical School

P21-11 MAP Kinase Phosphatase 5 Mediates Anti-inflammatory Activities of Dietary Chemopreventive Agents in Primary Cultures of Prostate Cells
Larisa Nonn and Donna M. Peehl
Stanford University School of Medicine

P21-12 Soy Protein Isolate Increases Urinary Estrogens and the Ratio of 2:16Alpha-hydroxyestrone in Men at High Risk of Prostate Cancer
S.A. Rebello, W. Thomas, J.W. Slaton, J.M. Hamilton-Reeves, and M.S. Kurzer
University of Minnesota, Twin Cities

P21-13 Prostate Cancer Chemoprevention: A Combination Strategy Targeting Androgen Signaling
Dian Yao and Haitao Zhang
Roswell Park Cancer Institute, Buffalo

P21-14 Prostate Cancer Chemopreventive Efficacy of Silibinin: Bench to Bedside
Rajesh Agarwal
University of Colorado Health Sciences Center, Denver

P21-15 Ketosamines: Food-related Glycoaminoconjugates with a Prostate Cancer Prevention Potential
Thomas P. Mawhinney and Valeri V. Mossine
University of Missouri-Columbia

Risk

Sep 7 12:30 p.m.-2:30 p.m.
Odd-numbered: 12:30 p.m.-1:30 p.m.
Even-numbered: 1:30 p.m.-2:30 p.m.

P22-1 Genetic Variation in the IGF Pathway and Prostate Cancer Risk
Yuan Chun Ding[1], Martha Slattery[1], Arthur Brothman[1], Susan L. Neuhausen[2], and Li-Hao Chu[2]
[1]University of California, Irvine, [2]University of Utah

P22-2 The Role of IGF-1 and IGFBP-3 Gene Polymorphisms on Serum Levels and Prostate Cancer Risk in African Americans
Wenndy Hernandez[1], Cassandra Grenade[1], Eunice R. Santos[1], Chiledum Ahaghotu[2], Carolina Bonilla[3], and Rick A. Kittles[1]
[1]University of Chicago, [2]University of Oxford, [3]Howard University

P22-3 Genetic Polymorphisms in CYP17, CYP3a4, CYP19, and SRD5a2 and Prostate Cancer Risk in African American Men: The Flint Men's Health Study
Leslie A. Lange[1], Anna Ray[2], Ethan M. Lange[1], Rodney L. Dunn[2], Kathleen A. Cooney[2], and Aruna V. Sarma[2]
[1]University of North Carolina at Chapel Hill, [2]University of Michigan

P22-4 Acrylamide and Prostate Cancer Risk
Kathryn Wilson[1], Katarina Balter[2], Yudi Pawitan[2], Henrik Gronberg[2], Margereta Tornqvist[3], Hans-Olov Adami[2], and Lorelei Mucci[1]
[1]Harvard School of Public Health, [2]Karolinska Institutet, [3]Stockholm University, Sweden

P22-5 RNASEL/HPC1 and Macrophage Scavenger Receptor 1 in Asian-Indian Advanced Prostate Cancer
Hanna Rennert[1], Charnita M. Zeigler-Johnson[2], Rama Mittal[3], Caren Sadowl[2], Joshua Edwards[2], Matthew J. Finely[2], Ying-cai Tan[1], Anyl Mandhani[3], Balraj Mital[3] and Timothy R. Rebbeck[2]
[1]Weill Medical College of Cornell University, [2]University of Pennsylvania, [3]Sanjay Gandhi Postgraduate Institute of Medical Sciences

P22-6 Association between Past Urinary Tract Infections and Current Symptoms Suggestive of Chronic Prostatitis/Chronic Pelvic Pain Syndrome
Carol L. Link[1], Michael J. Barry[2], John B. McKinlay[1], and Nicholas A. Daniels[3]
[1]New England Research Institute, Waterton, [2]Massachusetts General Hospital, [3]University of California, San Francisco

P22-7 Gene-Environment Interactions between Three Key Endogenous Antioxidant Enzymes and Dietary Antioxidants for Prostate Cancer Risk and Survival
Fredrick R. Schumacher[1], Kathryn Penney[2], Meir J. Stampfer[1], Haojie Li[1], and Jing Ma[1]
[1]Brigham and Women's Hospital, [2]Harvard University School of Public Health

P22-8 Molecular Signatures of Lethal Prostate Cancer
Meir J. Stampfer[1], Yudi Pawitan[2], Francesca Demichelis[1], Jennifer R. Stark[1], Swen-Olof Andersson[3], Ove AndrÉn[3], Lars Holmberg[4], Wei Huang[5], Philip W. Kantoff[1], Robert Kim[1], Sven Perner[1], Jan-Erik Johansson[3], Hans-Olov Adami[6], Katja Fall[2], Lorelei A. Mucci[1], and Mark A. Rubin[1]
[1]Brigham and Women's Hospital, [2]Karolinska Institute, [3]University of Örebro, Sweden, [4]Uppsala University, [5]University of Wisconsin, Madison, [6]Harvard Medical School

P22-9 Prostate Cancer Gene Identification by Admixture Mapping in African American Men
Ann G. Schwartz[1], David Reich[2], Susan J. Land[1], Benjamin A. Rybicki[3], Cathryn H. Bock[1], and Rick A. Kittles[4]
[1]Wayne State University, [2]Broad Institute, [3]Henry Ford Health System, [4]University of Chicago

P22-10 Prostate Cancer Risk Associated with Ambient Pesticide Exposure in California's Central Valley
Paul Mills[1], Xinbo Zhang[2], John Zadnick[2], Jennifer Marusek[2], Beate Ritz[3], and Myles Cockburn[2]
[1]Public Health Institute, Oakland, [2]University of Southern California, Keck School of Medicine, [3]University of California, Los Angeles

P22-11 Simultaneous Analysis of Germline CNPs and SNPs in Prostate Cancer Risk among Hereditary Prostate Cancer Families
Wennuan Liu[1], Jishan Sun[1], Latchezar Dimitrov[1], Siqun Lilly Zheng[1], Bao-Li Chang[1], William B. Isaacs[2], and Jianfeng Xu[1]
[1]Wake Forest University School of Medicine, [2]Johns Hopkins University School of Medicine

P22-12 Neighborhood Composition and Nutritional Biomarkers for Prostate Cancer Risk
Carlos A. Reyes Ortiz, Karl Eschbach, Hyunsu Ju, Yong-Fang Kuo, and James S. Goodwin
University of Texas Medical Branch, Galveston

P22-13 Assessing the Feasibility and Efficiency of Friend Control Recruitment in an African American Case Control Study
Aubrey R. Turner, Tamara S. Adams, Bao-Li Chang, and Jianfeng Xu
Wake Forest University School of Medicine

Angiogenesis

Sep 7 12:30 p.m.-2:30 p.m.
Odd-numbered: 12:30 p.m.-1:30 p.m.
Even-numbered: 1:30 p.m.-2:30 p.m.

P23-1 Effects of Stable Endostatin and Angiostatin Gene Therapy on Angiogenesis-related Gene Expression in Multistage Prostate Cancer in TRAMP Model
Tatyana Isayeva, Diptiman Chanda, Dongquan Chen, and Selvarangan Ponnazhagan
University of Alabama at Birmingham

P23-2 Targeting of Pericytes Diminishes Neovascularization and Lymphangiogenesis in Prostate Cancer
Ugur Ozerdem
La Jolla Institute for Molecular Biology

P23-3 Prostate Restricted Replicative Adenovirus Expressing Human Endostatin–Angiostatin Fusion Gene Exhibiting Dramatic Antitumor Efficacy
Xiong Li, You-Hong Liu, Sang-Jin Lee, Meei-Huey Jeng, Thomas A. Gardner, and Chinghai Kao
Indiana University, Indianapolis

P23-4 Vitamin D Receptor Agonist Treatment Alters Secretion of Angiogenic and Autocrine Growth Regulatory Factors That Inhibit Prostate Cancer Cell Growth and Angiogenesis
Muralimohan Yepuru and Nancy L. Weigel
Baylor College of Medicine

P23-5 Pigment Epithelial-derived Factor: Design and Development of Anti-angiogenic Peptides for the Therapy of Prostate Cancer
Arin Aurora, Olga V. Volpert, and Yelena Mirochnik
Northwestern University Medical School

P23-6 COX-2 and Prostate Cancer Angiogenesis
Xin-Hua Liu and Alice C. Levine
Mount Sinai School of Medicine, New York

Apoptosis

Sep 7 12:30 p.m.-2:30 p.m.
Odd-numbered: 12:30 p.m.-1:30 p.m.
Even-numbered: 1:30 p.m.-2:30 p.m.

P24-1 Zinc Targets Prostate Tumorigenicity by Inducing Apoptosis and Altering Metabolism in Prostate Cancer Cells
Tieluo Li, Zhixing Guan, Renty B. Franklin, Leslie C. Costello, and Pei Feng
University of Maryland, Baltimore

P24-2 A Novel Molecular Insight on Nucleotide Regulation of the Apoptosome Formation and Caspase Activation: Implications in Prostate Cancer Therapeutics
Shawn B. Bratton[1], Maria D. Person[1], Yanan Tian[2], Angel G. Martin[3], Mary Ayres[4], Howard O. Fearnhead[3], Varsha Gandhi[4], Dhyan Chandra[4], and Dean G. Tang[4]
[1]University of Texas at Austin, [2]Texas A&M University, College Station, [3]National Cancer Institute, [4]M.D. Anderson Cancer Center, University of Texas

P24-3 The Molecular Basis for Maspin-sensitized Prostate Epithelial Cell Apoptosis
Jiayou Liu, Xiaohua Li, Yonghong Meng, Neelima Reddy, Shijie Sheng, and Shuping Yin
Wayne State University School of Medicine

P24-4 Pretreatment of Acetyl Salicylic Acid Promotes TRAIL-induced Apoptosis by Downregulating BCL-2 Gene Expression
Ki M. Kim, Jee Young An, Yong Tae Kwon, Yong J. Lee, and Jae J. Song
University of Pittsburgh

P24-5 Concurrent Activation of PI3K/AKT and JNK, upon PKC Suppression, Induces Apoptosis in Prostate Cancer Cells
Jinjin Guo, LeaAnn Collins, and Chang-Yan Chen
Boston University School of Medicine

P24-6 The Role of FLIP Gene Transcription in Androgen-withdrawal Induced Apoptosis of the Prostate
Kent L. Nastiuk, Kiwon Yoo, Jennifer Davis, Andrew Cornforth, and John J. Krolewski
University of California, Irvine

P24-7 Apolipoprotein L1 and L6, Two Novel BH3-only Pro-death Proteins, Induce Two Different Types of Programmed Cell Death in Prostate Cancer Cells
Guanghua Wan, Zhaorigetu Siqin, Zhihe Liu, Zeyu Jiang, and Chien-an A. Hu
University of New Mexico Health Sciences Center

P24-8 C-FOS Promotes TRAIL-induced Apoptosis by Repressing C-FLIP(L)
Xiaoping Zhang, Liang Zhang, Hongmei Yang, Xu Huang, Hasan Otu, William C. DeWolf, Roya Khosravi-Far, Towia A. Libermann, and Aria F. Olumi
Massachusetts General Hospital, Harvard Medical Center

P24-9 New Paradigm for Antitumor Action of IGF Binding Protein-3 (IGFBP-3): Identification of a Novel IGFBP-3 Receptor and Its Proapoptotic and NF-kappaB Inhibitory Effects in Prostate Cancer
Jinfeng Han, Sherryline Jogie-Brahim, and Youngman Oh
Virginia Commonwealth University

P24-10 Enhancing the Apoptotic Potential of IGFBP-3 in Prostate Cancer by Regulation of Phosphorylation
Satomi Koyama, Laura J. Cobb, and Pinchas Cohen
University of California, Los Angeles

P24-11 Anti-apoptotic Protein Networks in the Endoplasmic Reticulum
Lili Chen and John C. Reed
Burnham Institute

P24-12 CCR9-CCL25 Interaction Mediates Cell-signaling Cascades Involved in Prostate Cancer Cell Survival
Rajesh Singh[1], James W. Lillard Jr.[1], William E. Grizzle[2], Leland W.K. Chung[3], and Shailesh Singh[1]
[1]University of Louisville School of Medicine, [2]University of Alabama at Birmingham, [3]Emory University

P24-13 Fatty Acid Binding Proteins in Prostate Cancer Cells
Rina Das, Marta Desantis, Marti Jett, and Rasha Hammamieh
Walter Reed Army Institute of Research

Cancer Stem Cells

Sep 7 12:30 p.m.-2:30 p.m.
Odd-numbered: 12:30 p.m.-1:30 p.m.
Even-numbered: 1:30 p.m.-2:30 p.m.

P25-1 Prostate Cancer (PCa) Stem/Progenitor Cells: Regulation of Self-renewal and Involvement in Metastasis
Dean Tang
M.D. Anderson Cancer Center, University of Texas

P25-2 Formation of Human Prostate Tissues from Embryonic Stem Cells to Study the Initiation of Prostate Cancer
R. Taylor[1], G.R. Cunha[2], J. Pedersen[3], A. Trounson[1], S. Hayward[4], and G.P. Risbridger[1]
[1]Monash University, [2]University of California, San Francisco, [3]Tissupath Pty, Ltd., Australia, [4]Vanderbilt University

P25-3 Prostate Cancer Stem Cells in Resistance to Radiotherapy
Man-Tzu Wang and Dao-Tai Nie
Southern Illinois University

P25-4 Identification of Putative Stem Cell Markers, CD133 and CXCR4 in hTERT-immortalized Primary Non-malignant and Malignant Tumor-derived Human Prostate Epithelial Cell Lines and in Prostate Cancer Tissues
Jun Miki[1], Bungo Furusato[1], Hongzhen Li[1], David G. McLeod[1], Hirayaki Takahasi[1], Shin Egawa[2], Johng S. Rhim[1], Isabell A. Sesterhenn[3], and Shiv Srivastava[1]
[1]Center for Prostate Disease Research, [2]Jikei University School of Medicine, Tokyo, [3]Armed Forces Institute of Pathology

P25-5 ABCG2-mediated Efflux of Androgen in Putative Benign and Malignant Prostate Stem Cells
Wendy J. Huss and Gary J. Smith
Roswell Park Cancer Institute

P25-6 Telomerase-immortalized Nonmalignant Human Prostate Epithelial Cells Retain the

Properties of Multipotent Stem Cells
Hongzhen Li[1], Jun Miki[1], Bungo Furusato[1], Shiv Srivastava[1], David G. McLeod[1], Johng S. Rhim[1], JianJun Zhou[1], and Jonathan C. Vogel[2]
[1]Center for Prostate Disease Research, [2]National Cancer Institute

P25-7 The Role of Basal Epithelial Cells in Prostate Carcinogenesis
Nien-Tsu Chen and Su Hao Lo
University of California, Davis

P25-8 Isolation of a Clonal Prostate Cancer Cell with Self-renewal Capacity and Phenotypic Plasticity
Daniel Seiler[1], Gentao Liu[2], Mike Aldridge[1], Asa Oudes[3], Alvin Liu[3], Arie Belldegrun[1], and Gang Zeng[1]
[1]University of California, Los Angeles, [2]Cedars-Sinai Medical Center, [3]University of Washington

P25-9 p63 Is Essential for the Proliferative Potential of All Stratified Epithelia
Filipa Pinto, Makoto Senoo, Michael Byrnes, and Frank McKeon
Harvard Medical School

P25-10 Midbody Derivatives as a Novel Structural Stem Cell Marker
JeanMarie Houghton[1], Stephen Lyle[1], Alexey Terskikh[2], Stephen J. Doxsey[1], and Chun-Ting Chen[1]
[1]University of Massachusetts Medical School, [2]Burnham Institute

P25-11 In Pursuit of Prostate Cancer Stem Cells
Lubna Patrawala and Dean Tang
M.D. Anderson Cancer Center, University of Texas

P25-12 Cells with Unique Properties in Prostate Cancer-associated Stroma Are Mesenchymal Stem Cells
Donna M. Peehl and Hongjuan Zhao
Stanford University

Biomarkers II

Sep 7 12:30 p.m.-2:30 p.m.
Odd-numbered: 12:30 p.m.-1:30 p.m.
Even-numbered: 1:30 p.m.-2:30 p.m.

P26-1 Core per Gram Ratio Predicts Prostate Cancer Detection on Transrectal Ultrasound-guided Prostate Biopsy
Douglas S. Scherr, Michael J. Schwartz, Andrew Hung, David Hwang, Justin W. McClain, Jullet Han, M. Mendel Shemtov, R. Ernest Sosa, E. Darracott Vaughan, Jr., and Alexis E. Te
Columbia University

P26-2 Endogenous IgM Antibody against Ganglioside GD1a: A Promising Glycoimmunomic Biomarker for Organ-confined Prostate Cancer
Sakunthala Muthugounder[1], Donald L. Morton[1], Stanley Brosman[2], and Mepur H. Ravindranath[1]
[1]John Wayne Cancer Institute, [2]Pacific Clinical Research

P26-3 Using PSA Doubling Time (PSADT) to Predict Overall Survival for Prostate Cancer Patients Who Experience PSA Failure after Surgery
Yongmei Chen[1], Zhe Chang[1], Jinxiu Zhao[1], David G. McLeod[1], Stephen J. Freedland[2], Albert Dobi[1], and Jennifer Cullen[1]
[1]Center for Prostate Disease Research, [2]Duke University Medical Center

P26-4 The Testis-specific Protein Y-Encoded Gene Potentiates Cell Proliferation and Could Contribute to the Initiation of Prostatic Oncogenesis
Yun-Fai Chris Lau[1], Tatsuo Kido[1], Yunmin Li[1], Tin-Lap Lee[2], and Wai-Yee Chan[2]
[1]University of California, San Francisco, [2]National Institute of Child Health and Human Development

P26-5 Diffuse Optical Measurements of Prostate Blood Flow and Oxygenation during Interstitial Photodynamic Therapy
Turgut Durduran, Chao Zhou, Xiaoman Xing, Jarod C. Finlay, Theresa M. Busch, S. Bruce Malkowicz, Stephen M. Hahn, Arjun G. Yodh, Guoqiang Yu, and Timothy C. Zhu
University of Pennsylvania

P26-6 Search for Potential Molecular Signatures and Therapeutic Targets for Metastatic Prostate Cancer
Dong Lin[1], Akira Watahiki[1], Fang Zhang[1], Victor Ling[1], Alan So[2], Peter W. Gout[1], Marianne Sadar[1], YZ Wang[1], and Martin Gleave[2]
[1]British Columbia Cancer Agency, [2]University of British Columbia

P26-7 Portable Prostate Cancer Detection System
Bruce E. Bejcek, Krystal Anderson, and Massood Z. Atashbar
Western Michigan University

P26-8 Allelic Imbalance in Prostatectomy Tissues Correlates with Pathological Gleason Score and Predicts Clinical Outcome
Jeffrey K. Griffith, Christopher M. Heaphy, and Marco Bisoffi
University of New Mexico, Albuquerque

P26-9 Exploiting a Molecular Gleason Grade for Prostate Cancer Diagnosis, Prognosis, and Therapy
Ilsa Coleman[1], Hong-Gee Sim[1], Mengchu Wu[1], Sarah Hawley[1], Alan Huang[1], Roger Coleman[1], Milton Datta[2], Paul Lange[1], Daniel Lin[1], Leroy Hood[3], Lawrence True[1], Edward Gelmann[4], Beatrice Knudsen[1], Elahe Mostaghel[1], Peter S. Nelson[1], and Robert Vessella[1]
[1]University of Washington, [2]Emory University, [3]Institute for Systems Biology, [4]Georgetown University

P26-10 No Association with Risk of Prostate Cancer for LDOC1 and SPANX-C Candidate Genes within the HPC-X Locus in a U.S. Study Population
Bradford Elmore, Joan Breyer, Kevin Bradley, Kate McReynolds, Jeffrey R. Smith, and Brian Yaspan
Vanderbilt University

P26-11 Expression of the Novel Survival Peptide, Humanin Protein Is Associated with Prostate Cancer Recurrence
Bingrong Liu, David Hwang, Hong Yu, Sheila Tze, Jonathan Said, David Seligson, Laura Cob, and Pinchas Cohen
University of California, Los Angeles

P26-12 Identification and Characterization of Prostate Cancer Associated Protein Biomarkers Using High-throughput Mass Spectrometry
Lisa H. Cazares[1], Shamina G. Mitchell[1], Mary Ann Clements[1], Tarek Kandil[1], Brian Main[1], O. John Semmes[1], Jose I. Diaz[2], and Gunjan Malik[2]

[1]*Eastern Virginia Medical School,* [2]*University of Texas Health Science Center at San Antonio*

P26-13 Telomere Attrition of Isolated High-grade Prostatic Intraepithelial Neoplasia and Surrounding Stroma Is Predictive of Prostate Cancer
Anthony M. Joshua[1], Bisera Vukovic[1], Ilan Braude[1], Sundus Hussien[2], Maria Zielenska[3], John Srigley[2], Andrew Evans[1], and Jeremy A. Squire[1]
[1]*University Health Network, Toronto,* [2]*Credit Valley Hospital,* [3]*Hospital for Sick Children*

P26-14 Serum Glycan Profiling as a Prognostic Indicator for Prostate Cancer
Crystal Kirmiz, Ruth Vinall, David Rocke, Carlito Lebrilla, Ralph deVere White, and Suzanne Miyamoto
University of California, Davis

P26-15 Evaluation of Genomic Instability by Methylation Status in the Abnormal Prostate
Kimberly Butler, Jeffrey Griffith, Christina M. Haaland-Pullus, Christopher Heaphy, and Marco Bisoffi
University of New Mexico, Albuquerque

P26-16 Gain of Copy Number of an 18Q22.1 Region That Includes the Cadherin-7 Gene in Prostate Cancer
Veronica E. Contreras-Shannon[1], Sapna Vijayakumar[2], Robin J. Leach[1], Teresa L. Johnson-Pais[1], and Susan L. Naylor[1]
[1]*University of Texas Health Science Center at San Antonio,* [2]*Mount Sinai School of Medicine, New York*

P26-17 Investigation of a Putative Estrogen-imprinting Gene, Phosphodiesterase Type IV Variant 4 (PDE4D4) in Determining Prostate Cancer Risk
Wan-yee Tang
University of Massachusetts Medical School

P26-18 Analysis of Trace Metals in Paraffin-embedded Prostate Tissue Specimens Using Inductively Coupled Plasma Mass-spectrometry
Andrey Sarafanov[1], Todor I. Todorov[2], Andre Kajdacsy-Balla[3], Marion Gray[4], Virgilia Macias[3], and Jose A. Centeno[1]
[1]*Armed Forces Institute of Pathology,* [2]*U.S. Geological Survey,* [3]*University of Chicago,* [4]*James Cook University, Australia*

P26-19 Androgen Signaling and ER Stress Response Proteins in Prostate Cancer
Q. Wang[1], R. Mori[2], Danenberg P[2], K. Danenberg[2], and J. Pinski[1]
[1]*University of Southern California, Keck School of Medicine,* [2]*Response Genetics, Inc.*

P26-20 The Role of CaP-dependent Translation in Prostate Cancer Progression and Metastasis to Bone
Rebecca A. McGaha, Shannon Walls-Pylant, Susan J. Thornewell, Lisa K. Jones, Jennifer L. Carroll, and Briana Jill Williams
Louisiana State University Health Sciences Center

P26-21 PEDF Regulation of Adipogenesis and Leptin in Prostate Cancer
Mona Cornwell, Susan E. Crawford, and Jennifer A. Doll
Northwestern University Medical School

P26-22 Expression of Stress Response Protein Grp78 Is Associated with the Development of Castration-resistant Prostate Cancer
Llana Pootrakul, Ram H. Datar, Shan-Rong Shi, Jie Cai, Debra Hawes, Susan Groshen, Amy S. Lee, and Richard J. Cote
University of Southern California, Keck School of Medicine

P26-23 NECL-3a Expression in Human Prostate Cancer
Guimin Chang, Shuping Xu, N. Simone Harya, Federico A. Monzon, Rajiv Dhir, Jeffrey R. Gingrich, and Denise O'Keefe
University of Pittsburgh

Signaling II

Sep 7 12:30 p.m.-2:30 p.m.
Odd-numbered: 12:30 p.m.-1:30 p.m.
Even-numbered: 1:30 p.m.-2:30 p.m.

P27-1 Androgen Regulated Stromal Factors Mediate Fibroblast Growth Factor Receptor-2IIIb Activation during Prostatic Carcinogenesis
Emily A. Ricke, Alan Friedman, William A. Ricke, Yi-Fen Lee, and Karin Williams
University of Rochester

P27-2 Hypoxia and DNA Repair as Factors in Prostate Cancer Progression and Aggression
Tien Phan, Alice Meng, Trevor Do, Trudey Nicklee, Theo van der Kwast, Joan Sweet, Richard Hill, David Hedley, Robert G. Bristow, and Michael Milosevic
University Health Network, Toronto

P27-3 Potential Role of Growth Arrest and DNA Damage Inducible, Alpha in Treatment of Prostate Cancer
Kavitha Ramachandran, Gopal Gopisetty, Loida Navarro, Edna Gordian, and Rakesh Singal
University of Miami School of Medicine

P27-4 Nitric Oxide Signaling Inhibits Hypoxia-mediated Resistance of Prostate Cancer Cells to the Natural Cytotoxic Activity of Peripheral Blood Lymphocytes
Nianping Hu[1], Hugh Pross[1], Eugene Chung[1], A.K. Sheikhi[2], D. Robert Siemens[1], and Charles H. Graham[1]
[1]*Queen's University,* [2]*Zanjan University of Medical Sciences, Iran*

P27-5 The Role of Sex Hormone-binding Globulin in the Androgen Response of Human Prostate Cancer Cells
Atif M. Nahkla[1], Daniel J. Hryb[1], Yu-Hua Li[2], Jenny Xiang[3], Nicholas A. Romas[1], William Rosner[1], and Scott M. Kahn[1]
[1]*St. Luke's-Roosevelt Hospital Center,* [2]*Emory University,* [3]*Cornell University, Weill Medical College*

P27-6 EZH2 Regulates the Transcription of Estrogen-responsive Genes through Association with REA, an Estrogen Receptor Corepressor
Clara Hwang[1], Veda N. Giri[2], Casey W. Wright[1], Amanda S. Wilkinson[1], Kathleen A. Cooney[1], Colin S. Duckett[1], and John C. Wilkinson[1]
[1]*University of Michigan,* [2]*Fox Chase Cancer Center*

P27-7 p53 Gain-of-Function Cancer Mutants Commonly Identified in Prostate Cancers Induce Genetic Instability by Inactivating ATM
Monica Hollstein[1], Hoseok Song[2], and Yang Xu[2]

[1]*German Cancer Research Center (DKFZ),* [2]*University of California, San Diego*

P27-8 Synthesis and Function of Vascular Endothelial Growth Factor-C during Androgen Ablation in Prostate Cancer
Jinping Li, Francesca Rinaldo, Enfang Wang, Michael Muders, and Kaustubh Datta
Mayo Clinic and Foundation, Rochester

P27-9 Identification of ATM-Vitamin D/Vitamin D Receptor-DNA Repair Signal Axis to Extend Vitamin D Chemopreventive Effects against Prostate Carcinogenesis
Huei-Ju Ting, Bo-Ying Bao, and Yi-Fen Lee
University of Rochester

P27-10 Suppression of Prostate Cancer by Ink4C and PTEN
Yue Xiong
University of North Carolina School of Medicine

P27-11 Insulin-like Growth Factor Binding Protein-3 (IGFBP-3) Suppresses Prostate Tumor Growth in Both an IGF-dependent and IGF-independent Manner
Josef V. Silha, Patricia C. Sheppard, Suresh Mishra, Yaoting Gui, Jacquie S. Schwartz, Liam J. Murphy, and Janice G. Dodd
University of Manitoba

P27-12 Lipogenesis as a Regulator of Endoplasmic Reticulum Function and Tumor Cell Invasion
Joy L. Little[1], Frances Wheeler[1], Diane R. Fels[1], Darren F. Seals[1], Constantinos Koumenis[2], and Steven J. Kridel[1]
[1]*Wake Forest University Health Sciences,* [2]*University of Pennsylvania School of Medicine*

P27-13 Müllerian Inhibiting Substance (MIS) Is Up-regulated by Calcitriol in LNCaP Prostate Cancer Cells Via a Direct Interaction of the Vitamin D Receptor with a Vitamin D Response Element in the MIS Promoter
Peter J. Malloy, Lihong Peng, and David Feldman
Stanford University School of Medicine

P27-14 Interleukin-17 Receptor-like: A Novel Gene That Modulates Prostate Cancer Initiation and Progression
Ying Dong[1], Xiangtian Kong[1], Jonathan Melamed[2], Yi Zhang[1], Laurel A. Beckett[1], Regina Gandour-Edwards[1], Ralph W. de Vere White[1], A. Hari Reddi[1], Robert L. Vessella[3], and Zongbing You[1]
[1]*University of California, Davis,* [2]*New York University School of Medicine,* [3]*University of Washington Medical Center*

P27-15 Induction of CYP24 Epigenetic Silencing and Sensitivity to Calcitriol in Matrigel-derived Endothelial Cells (MDEC) by Tumor-conditioned Media
Adam R. Karpf, Norma Nowak, Wei-dong Yu, Rui-Xian Kong, Candace S. Johnson, Ivy Chung, and Donald L. Trump
Roswell Park Cancer Institute, Buffalo

P27-16 AP-2 Regulates the Transcription of Estrogen Receptor-beta by Acting through a Methylation Hotspot of the 0N Promoter in Prostate Cancer Cells
Yuet-Kin Leung, Shuk-mei Ho, and Xiang Zhang
University of Connecticut

P27-17 HOXC Gene Expression Modulates Androgen- and Vitamin D-mediated Actions in Human Prostate Cancer Cells
James R. Lambert, M. Scott Lucia, Sunshine N. Daddario, and Steven K. Nordeen
University of Colorado Denver, Health Sciences Center

P27-18 Regulation of CXCL14 Expression and Dendritic Cell Attraction in Prostate Cancer
Dmitry W. Gutkin, Galina V. Shurin, and Michael R. Shurin
University of Pittsburgh School of Medicine

P27-19 Consequences of Inhibition of CDK5 in Prostate Cancer Cells
Christopher J. Strock, Neha Pandey, Michael J. Ochs, and Barry D. Nelkin
Johns Hopkins University School of Medicine

P27-20 Paracrine Hedgehog Signaling Regulates Prostate Tumor Growth
Jerry Gipp, Wade Bushman, and Aubie Shaw
University of Wisconsin, Madison

P27-21 Loss of Corepressor Function in Prostate Cancer Alters Targeting of the Nucleosome Remodeling and Deacetylase Complex
Rajini Srinivasan, Rebecca Ward, and John Svaren
University of Wisconsin, Madison

P27-22 Characterization of a Novel 12(S)-HETE Receptor and Role in Prostate Cancer Progression
Yande Guo, Senlin Zhou, Keqin Tang, Yinlong Cai, and Kenneth V. Honn
Wayne State University

P27-23 Tumor Suppressor Activity of the EPHB2 Receptor in Prostate Cancer
Severine Roselli, Fatima Valencia, Nicole K. Noren, and Elena B. Pasquale
The Burnham Institute for Medical Research

P27-24 Canonical WNT Signaling in Prostate Organogenesis
Marianna Kruithof-de Julio, Cheng Gao, Nishita Desai, and Michael M. Shen
University of Medicine and Dentistry of New Jersey, Robert Wood Johnson Medical School

P27-25 TGFbeta Signaling in Prostate Cancer
Bala Balakumaran[1], Emily Pontzer[1], Raanan Berger[2], Phillip G. Febbo[1], William Hahn[3], and Mark Rubin[4]
[1]*Duke University Medical Center,* [2]*Chaim Sheba Medical Center,* [3]*Dana-Farber Cancer Institute,* [4]*Brigham and Women's Hospital*

P27-26 Role of the Protein Kinase BMX in Prostate Cancer
Xinnong Jiang, Christopher L. Carpenter, Steve P. Balk, and Abdelhafidh Saci
Beth Israel Deaconess Medical Center, Boston

P27-27 Mechanism of BMP Inhibition of Prostate Cancer
Jin-Taek Hwang[1], Jerry Zhu[1], and Michael Freeman[2]
[1]*Brigham and Women's Hospital,* [2]*Children's Hospital, Boston*

P27-28 A Kinomic Approach for Identifying Kinase Substrates in Prostate Cancer
Bin Guan, Xiang Li, and Charles J. Bieberich
University of Maryland, Baltimore County

P27-29 Effects of Lysophosphatidic Acid, Sphingosine-1-Phosphate, Epidermal Growth Factor, and Lycopene on Signal Transduction in Human Prostate Cancer Cells
Maria V. Rubio, Zhihong Zhang, Terra C. Gibbs, Yuhuan Xie, Daniel Brauner, Kevin Kipp, and Kathryn E. Meier
Washington State University, Pullman

P27-30 Alpha-catenin and a Small Molecule Inhibitor Suppress Beta-catenin Oncogenic Signaling: Implications for Prostate Cancer Treatment
Landon J. Inge and Ayyappan K. Rajasekaran
Nemours Center for Childhood Cancer Research, A.I. DuPont Hospital for Children

Androgen Receptor

Sep 7 12:30 p.m.-2:30 p.m.
Odd-numbered: 12:30 p.m.-1:30 p.m.
Even-numbered: 1:30 p.m.-2:30 p.m.

P28-1 Essential Role for Integrins in Androgen Receptor Activation Mediated by Androgen and Insulin-like Growth Factor-I in Prostate Cancer Cells
Naved Alam, Hira Lal Goel, and Lucia R. Languino
University of Massachusetts Medical School

P28-2 Roles of Androgen Receptor and Its Cofactors in Prostate Tumorigenesis and Prostate Cancer Progression
Liran Zhou, Wei Qi, Shen Gao, and Zhengxin Wang
M.D. Anderson Cancer Center, University of Texas

P28-3 Suppression of Androgen Receptor Activity by Histone Deacetylase 4 through Receptor Sumoylation
Yonghua Yang[1], Qiuping Ma[1], Wei Fu[1], Pengfei Li[1], Umesh Jinwal[1], Santo V. Nicosia[1], Xiaohong Zhang[2], and Wenlong Bai[1]
[1]University of South Florida College of Medicine, [2]H. Lee Moffitt Cancer Center

P28-4 Bone Morphogenetic Proteins 2 Induced Androgen Receptor Expression Via Extracellular Signal-regulated Protein Kinases and Heterogeneous Nuclear Ribonucleoprotein K Signal Pathways in Human Prostate Cancer Progression
Yimin Wu[1], Hui-Hsiu Chuang[1], Mary MacDougall[2], and Shuo Chen[1]
[1]University of Texas Health Science Center at San Antonio, [2]University of Alabama at Birmingham

P28-5 Structure-based Studies of Androgen Receptor-coactivator Interface as a Putative Drug Design Target for Treatment of Prostate Cancer
X. Edward Zhou, Kelly Suino-Powell, and H. Eric Xu
Van Andel Research Institute

P28-6 Broad Chromatin Markings Induce Super-efficient Transcription in Castrate-resistant Prostate Cancer
Li Jia and Gerhard A. Coetzee
University of Southern California

P28-7 Function of an Androgen Receptor Coactivator Regulated in Prostate Development and Prostate Cancer
Jerome C. Nwachukwu[1], Rachel Ruoff[1], Susan Ha[1], Miles Brown[2], Susan K. Logan[1], Michael J. Garabedian[1], and Samir S. Taneja[1]
[1]New York University School of Medicine, [2]Harvard Medical School

P28-8 Identification of Novel Androgen Receptor Target Genes in Prostate Cancer
Unnati Jariwala[1], Jennifer Prescott[1], Li Jia[1], Artem Barski[1], Steve Pregizer[1], Jon P. Cogan[1], Armin Arasheben[1], Wayne D. Tilley[2], William L. Gerald[3], Grant Buchanan[1], Gerhard A. Coetzee[1], Baruch Frenkel[1], and Howard I. Scher[3]
[1]University of Southern California, [2]University of Adelaide, [3]Memorial Sloan-Kettering Cancer Center

P28-9 Prostate Specific shRNA Library, Construction and Selection of Genes Involved in Progression toward Androgen Independence
Patrice Ohouo, Natalie Warholic, Mirza Baig, Elina Levina, Igor Roninson, Ralph Buttyan, and Michael Shtutman
Ordway Research Institute, Inc.

P28-10 11Beta-Alkyl-Delta9-19-Nortestosterone Derivatives: High-affinity Ligands and Potent Partial Agonists of the Androgen Receptor
Smita S. Muddana, Aimee M. Price, Megan M. MacBride, and Blake R. Peterson
Pennsylvania State University

P28-11 NF-kappaB Regulates Androgen Receptor Expression and Prostate Cancer Growth
Saleh Altuwaijri[1], Fangming Deng[2], Lishi Chen[3], Priti Lal[4], Ruslan Korets[3], Sven Wenske[3], William L. Gerald[3], Chawnshang Chang[5], Hans G. Lilja[3], Howard I. Scher[3], and Liying Zhang[3]
[1]University of Michigan, [2]State University of New York, Upstate Medical University, [3]Memorial Sloan-Kettering Cancer Center, *[4]University of Pennsylvania, [5]University of Rochester*

P28-12 Distinct Expression and Function of Androgen Receptor Coactivator ARA70alpha and ARA70beta and Novel Treatment for Prostate Cancer Overexpressing ARA70beta
Peng Lee[1], Yi Peng[1], Caihong X. Li[1], Fei Chen[1], Jianjun Wei[1], Jonathan Melamed[1], William Gerald[2], Michele Pagano[1], Martin Ligr[1], Michael Garabedian[1], and Zhengxin Wang[3]
[1]New York University School of Medicine, [2]Memorial Sloan-Kettering Cancer Center, [3]M.D. Anderson Cancer Center, University of Texas

P28-13 Histone Deacetylase 6 Regulates the Stability of Androgen Receptor and Epidermal Growth Factor Receptor
Yasheng Gao and Tso-Pang Yao
Duke University Medical Center

P28-14 Prx1 Interacts with Androgen Receptor and Enhances Its Trans-activation by Hypoxia/Reoxygenation
Soo-Yeon Park, Xiaofei Yu, Clement Ip, Paul N. Bogner, James L. Mohler, and Young-Mee Park
Roswell Park Cancer Institute, Buffalo

P28-15 Translational Repression of Androgen Receptor by Regulatory Noncoding RNAs
Girish C. Shukla
Cleveland State University

P28-16 Molecular Determinants of Corepressor Actions in the Functions of Selective Androgen Receptor Modulators
T. Zhou, P. Zhu, S.H. Baek, C.K. Bourk, E.M. Glass, and M.G. Rosenfeld
University of California, San Diego

P28-17 Androgen Receptor Downregulation by the Antiestrogen Fulvestrant (ICI 182,780) in LNCaP Human Prostate Cancer Cells
Aruna V. Krishnan, Srilatha Swami, Rumi S. Bhattacharyya, and David Feldman
Stanford University

P28-18 Targeting the Androgen Receptor for Ubiquitination and Degradation: A New Strategy for Therapy in Prostate Cancer
Kedra Cyrus[1], Michael Salcius[2], Craig Crews[2], Kyung Kim[1], Raymond J. Deshaies[3], Kathleen M. Sakamoto[4], and Agustin Rodriguez[4]
[1]University of Kentucky, [2]Yale University, [3]California Institute of Technology, [4]University of California, Los Angeles

P28-19 The PPAR Gamma Ligand Ciglitazone Reduces Androgen Receptor Activity in Androgen-dependent, but Not Androgen-independent, Human Prostate Cancer Cells
Patrice Moss, Besstina Lyles, and LaMonica V. Stewart
Meharry Medical College, Nashville

P28-20 Prostate Cell-specific Regulation of Androgen Receptor Phosphorylation in Vivo
Rachel Ruoff, Susan Ha, Hong Ying Huang, Ellen Shapiro, Jonathan Melamed, Susan K. Logan, Michael J. Garabedian, and Samir S. Taneja
New York University School of Medicine

P28-21 Regulation of Androgen Receptor Activity by Atypical Ubiquitylation Induced by a Ring-domain Containing Protein RNF6 in Prostate Cancer Cells
Yingqiu Xie, Kexin Xu, and Yun Qiu
University of Maryland, Baltimore

P28-22 Disruption of the Androgen Receptor Activation Pathway through Identification of the Androgen Receptor-BAF57 Interaction Site: A Potential Point of Intervention for Prostate Cancer
Clay Comstock[1], Nathan Powers[1], Tamzin Tanner[2], Frank Claessens[2], Karen E. Knudsen[1], and Kevin A. Link[1]
[1]University of Cincinnati, [2]University of Leuven

P28-23 Evidence for Calpain-mediated Androgen Receptor Cleavage as a Mechanism for Androgen Independence and Potential Therapeutic Target in Prostate Tumors
Stephen J. Libertini, Clifford G. Tepper, David M. Asmuth, Hsing-Jien Kung, and Maria Mudryj
University of California, Davis

P28-24 Expression of CHIP, a Co-chaperone Which Interacts with the Androgen Receptor, Results in Loss of AR Expression and Growth Inhibition of Prostate Cancer Cells
Xiaoyoung Zheng, Antonio Otero, Erwin Wang, Yuancheng Wang, Sherwin Zargaroff, Jian Pu, Mary Kunjappu, Avrom Caplan, Simon Hall, and Waleed Hassen
Mount Sinai School of Medicine, New York

P28-25 An Androgen Receptor-Skp2 Pathway Promotes Proliferation of Androgen-dependent Prostate Cancer Cells
Hongbo Wang[1], Daqian Sun[1], Fred Bauzon[1], Peng Ji[1], James L. Mohler[2], and Liang Zhu[1]
[1]Albert Einstein College of Medicine of Yeshiva University, [2]Roswell Park Cancer Institute, Buffalo

P28-26 Identifying Molecular Factors in Androgen Receptor Nuclear Export
Minh M. Nguyen, Yujuan Wang, and Zhou Wang
University of Pittsburgh

Hormone Refractory Prostate Cancer

Sep 7 12:30 p.m.-2:30 p.m.
Odd-numbered: 12:30 p.m.-1:30 p.m.
Even-numbered: 1:30 p.m.-2:30 p.m.

P29-1 Conditional AKT Activation Promotes Androgen-independent Progression of Prostate Cancer
Paul F. Terranova, J. Brantley Thrasher, and Benyi Li
University of Kansas Medical Center, Kansas City

P29-2 Role of ANXA7 in Metastatic and Hormone Refractory Prostate Cancer
Mirta Glasman, Ximena Leighton, Katerina Mezhevaya, Shanmugam Naga, Harvey B. Pollard, and Meera Srivastava
Uniformed Services University of the Health Sciences

P29-3 Baseline Pain Predicts Overall Survival in Men with Metastatic Hormone-resistant Prostate Cancer (HRPC)
Susan Halabi[1], San-San Ou[1], Alice B. Kornblith[2], Philip W. Kantoff[2], Nancy A. Dawson[3], Nicholas J. Vogelzang[4], and Eric J. Small[5]
[1]Duke University Medical Center, [2]Dana-Farber Cancer Institute, [3]University of Maryland, [4]Nevada Cancer Institute, [5]University of California

P29-4 Oxidative Stress Induction of L1 Cell Adhesion Molecule Expression Promoted Androgen-independent Prostate Cancer Cell Survival: A Potential Therapeutic Application in Radiation Therapy
Shian-Ying Sung, Ira Rajbhandari, Nicole A. Johnson, Rebecca S. Arnold, Peter A. S. Johnstone, Chia-Ling Hsieh, and John A. Petros
Emory University

P29-5 Genomic Analysis of Circulating Hormone Refractory Prostate Cancer Cells
Pamela L. Paris, Shivaranjani Sridharan, and Jonathan E. Rosenberg
University of California, San Francisco

P29-6 Role of ER Stress in Bortezomib-mediated TRAIL Sensitization
Keyi Zhu, Anne Kwan, Nancy Nibilsi, and David McConkey
M.D. Anderson Cancer Center, University of Texas

P29-7 The 44 kDa PIM-1 Kinase Phosphorylates BCRP/ABCG2 and Promotes Its Drug Resistant Activity in Human Prostate Cancer Cells

Yingqiu Xie[1], Douglas Linn[1], Takeo Nakanishi[1], Douglas Ross[1], Hegang Chen[1], Ladan Fazli[1], Zhiyong Guo[1], Kexin Xu[1], Yun Qiu[1], and Martin E. Gleave[2]
[1]University of Maryland School of Medicine, [2]Vancouver General Hospital

Imaging

Sep 7 12:30 p.m.-2:30 p.m.
Odd-numbered: 12:30 p.m.-1:30 p.m.
Even-numbered: 1:30 p.m.-2:30 p.m.

P30-1 Whole-body Optical Imaging of Prostate Cancer Metastasis and Gene Expression
Ping Jiang, Meng Yang, and Robert M. Hoffman
Anticancer Incorporated

P30-2 Noninvasive Imaging of Firefly Luciferase Reporter Gene Expression Using Bioluminescence Imaging in Human Prostate Cancer Models
Hongwei Li, Jin Zhong Li, Gregory A. Helm, and Dongfeng Pan
University of Virginia

P30-3 In Vivo Molecular Imaging for Photodynamic Therapy of Prostate Cancer
Jeffrey Duerk, Nancy Oleinick, and Baowei Fei
Case Western Reserve University

P30-4 Excitation Enhanced Imaging for Prostate Cancer Detection: In Vitro and in Vivo Results
Raymond J. Ro[1], William T. Shi[1], Michael K. Knauer[2], Kausik Sarkar[3], Anne L. Hall[4], Chris Vecchio[2], Richard Bernardi[2], and Flemming Forsberg[1]
[1]Thomas Jefferson University, [2]Spectrasonics, Inc., [3]University of Delaware, [4]GE Healthcare

P30-5 Adenoviral Vector Enabled Non-invasive Imaging of Sentinel Lymph Node Metastases of Prostate Cancer
Jeremy Burton, Mai Johnson, Shuwen Koh, Makoto Sato, and Lily Wu
University of California, Los Angeles

P30-6 A Novel Approach to Monitoring Prostate Tumor Oxygenation: Proton MRI of the Reporter Molecule, Hexamethyldisiloxane
Vikram Kodibagkar, Xianghui Wang, Weina Cui, and Ralph Mason
University of Texas Southwestern Medical Center at Dallas

P30-7 Application of Metabolomic Imaging in Prostate Cancer Detection
Kate W. Jordan[1], Eva Ratai[1], Jinhua Sheng[2], Christopher J. Wiggins[1], Graham Wiggins[1], George Dai[1], Bruce G. Jenkins[1], Leslie Ying[2], Chin-Lee Wu[1], and Leo L. Cheng[1]
[1]Massachusetts General Hospital, [2]University of Wisconsin, Milwaukee

P30-8 Correlation of in Vivo MR Imaging Findings with Whole Mount Histological Sections from Radical Prostatectomy Patients
Bao Zhang, Khan A. Siddiqui, Steven Roys, John Papadimitriou, Harry Yfantis, Danielle Hollanda, James Borin, Michael Naslund, and Rao Gullapalli
University of Maryland, Baltimore

P30-9 Using 2-Fluoro-4-nitrophenyl Beta-D-galactopyranoside to Detect Beta-galactosidase in PC3 Prostate Xenograft by 19F NMR
Li Liu[1], Jian-Xin Yu[1], Vikram D. Kodibagkar[1], Stephen L. Brown[2], and Ralph P. Mason[1]
[1]University of Texas Southwestern Medical Center at Dallas, [2]Henry Ford Hospital

P30-10 IL-13Ralpha2, a Novel Marker for Imaging of Epithelial-Mesenchymal Transition (EMT) in Human Prostate Cancer Cells Using Semiconductor Quantum Dots
Ying Zhu, Weiping Qian, Haiyen E. Zhau, Leland W.K. Chung, Ruoxiang Wang, and Chunmeng Shi
Emory University

P30-11 The Consequences of Fatty Acid Synthase in Prostate Tumors: PET Imaging of FAS Expression in Vivo
Amy L. Vāvere and Jason S. Lewis
Washington University

P30-12 Multi-dimensional MR Spectroscopic Imaging of Human Prostate Cancer in Vivo
Rajakumar Nagarajan, Steven S. Raman, Mittul Gulati, Nader Binesh, Daniel Margolis, Allan Pantuck, David Lu, Robert E. Reiter, and Michael Albert Thomas
University of California, Los Angeles

P30-13 Nuclear Magnetic Resonance Spectroscopy of Expressed Prostatic Secretions: Metabolite Citrate and Derivatives Are Potential Markers of Prostate Cancer
Eduard J. Gamito, Richard H. Jones, Colin O'Donnell, E. David Crawford, Tammy Hedlund, and Natalie Serkova
University of Colorado Denver, Health Sciences Center

P30-14 Fluorescence Imaging of Verteporfin-mediated Photodynamic Therapy Targeting Prostate Tumor Vasculature
Bin Chen, Chong He, Curtis Crane, and Brian Pogue
Dartmouth University

P30-15 Integration of Diagnostic and Interventional MRI for the Study of Persistent Prostate Cancer after External Beam Radiotherapy
Gregory Bootsma, Mathew Filleti, Cathy Rocca, Anna Kirilova, Masoom Haider, David Jaffray, and Cynthia Menard
University Health Network, Toronto

P30-16 Noninvasive Localization of Prostate Cancer via Diffusion-sensitive MRI
Peter A. Humphrey, Adam S. Kibel, Abraham Z. Snyder, Vamsidhar R. Narra, Joseph J.H. Ackerman, Sheng-Kwei Song, and Junqian Xu
Washington University

P30-17 PSMA-targeted Polygadolinium Clusters: A Novel Agent for Imaging Prostate Cancer
Chang-Tong Yang, Donald D. Nolting, John Thurston, David Rotsch, Yibo Zhou, and Louis Messerle
University of Iowa

P30-18 Targeting Prostate Cancer through the Androgen Receptor with Organometallic Tc-99M Complexes
Elsa Silva-Lopez, Brienne Bottenus, and Paul Benny
Washington State University, Pullman

P30-19 Design and Synthesis of Novel LacZ Responsive Enhanced MRI Agent
Vikram D. Kodibagkar, Ralph P. Mason, and Jian-Xin Yu
University of Texas Southwestern Medical Center at Dallas

P30-20 Development of a Mouse Model for Prostate Cancer Imaging and the Study of Disease Progression

Ying Cai, Scott Hahm, Isla Garraway, and Robert Reiter
University of California, Los Angeles

P30-21 Synthesis, Radiolabeling, and Biodistribution of Novel PSMA Ligands in Mouse Models of Prostate Cancer
Sangeeta B. Ray, Ronnie C. Mease, James J. Fox, Catherine A. Foss, and Martin G. Pomper
Johns Hopkins University School of Medicine

P30-22 Inhibitor-directed Imaging of Prostate Cancer
Lisa Wu, Tiancheng Liu, Marat Kazak, and Clifford E. Berkman
San Francisco State University

P30-23 New Strategies for Interpreting in Vivo Prostate Magnetic Resonance Imaging/Magnetic Resonance Spectroscopy: Manipulating Expression of Genes of Choline Metabolism to Enhance Cancer Specificity
Andrew Guerra, Dana Goldner, Robert E. Lenkinski, Jin-Rong Zhou, and Sandra M. Gaston
Beth Israel Deaconess Medical Center, Boston

Immunotherapy

Sep 7 12:30 p.m.-2:30 p.m.
Odd-numbered: 12:30 p.m.-1:30 p.m.
Even-numbered: 1:30 p.m.-2:30 p.m.

P31-1 Combined Gene Therapy with Cytosine Deaminase Plus Uracil Phosphoribosyl Transferase and Immunostimulatory IL12 and IL18 Cytokines for Treating Prostate Cancer in C57BL/6 Mice
Yasmin Husaini, Kim Ow, Jane Chapman, Lara Perryman, Aparajita Khatri, and Pamela J. Russell
University of New South Wales

P31-2 Adoptive Transfer of Tumor-specific TGF-Beta Insensitive CD8+ T Cells for Prostate Cancer: Introduction of the Anti-tumor Immune Response Cycle
Qiang Zhang and Chung Lee
Northwestern University

P31-3 Icon-mediated Immunotherapy for Prostate Cancer
Zhiwei Hu and Alan Garen
Yale University

P31-4 A Roadmap for the Development of a Prostate Cancer Vaccine
William A. Rose II[1], Thomas B. Albrecht[1], Eugene P. Knutson[1], Rolf Konig[1], Joana R. Perdigao[2], Alexandra P.A. Nguyen[2], David A. Ansari[2], Angela J. Jorgensen[2], Theresa K. Umhoefer[2], Tzu G. Wu[2], and W. Robert Fleischmann, Jr.[2]
[1]*University of Texas Medical Branch, Galveston,* [2]*University of Minnesota Medical School*

P31-5 A New Target for Immunotherapy of Prostate Cancer
Fang Guo, Ivelina Gueorguieva, Sang Ryu, and Boris R. Minev
University of California, San Diego

P31-6 Exploiting the Innate Antitumor Activity of Adoptively Transferred GammaDelta-T Cells for the Treatment of Prostate Cancer in a Mouse Model
Zhiyong Liu, Isam-Eldin Eltoum, Ben L. Guo, Gretchen A. Cloud, Benjamin H. Beck, and Richard D. Lopez
University of Alabama at Birmingham

P31-7 Anti-B7-1/B7-2 mAb Treatment Enhanced Anti-tumor Immunity in TRAMP Mice
Penghui Zhou, Xincheng Zheng, Huiming Zhang, Yang Liu, and Pan Zheng
University of Michigan

P31-8 Targeting the Intratumoral Dendritic Cells by the Oncolytic Adenoviral Vaccine Expressing RANTES Elicits Potent Anti-tumor Immunity
Natalia Lapteva, Melissa Aldrich, David Weksberg, Tatiana Goltsova, Lisa Rollins, Si-Yi Chen, and Xue Huang
Baylor College of Medicine

P31-9 Optimization of rsPSMA Protein Vaccine for Immunizing Prostate Cancer Patients with Minimal Disease
Fusataka Koide, Susan F. Slovin, Philip O. Livingston, and Govind Ragupathi
Memorial Sloan-Kettering Cancer Center

P31-10 Artificial Antigen Presenting Cells, AAPC, a New Tool for Adoptive Immunotherapy for Prostate Cancer
Ophelia Rogers, Mathias Oelke, and Jonathan P. Schneck
Johns Hopkins University School of Medicine

P31-11 Modification of Endothelin Axis Affects Dendritic Cells Antitumor Activity
Renee Kancelarich, Sean Taheri, Mark L. Jordan, and Georgi Guruli
University of Medicine and Dentistry of New Jersey, Robert Wood Johnson Medical School

P31-12 An Oligonucleotide Agonist of TLR9 for Prostate Cancer Therapy
Wei Wang, Mao Li, Ruiwen Zhang, Hui Wang, Elizabeth Rayburn, and Zhuo Zhang
University of Alabama at Birmingham

P31-13 Shedding of MIC Promotes Tumor Establishment: Overexpression of a Mutant Non-sheddable Form of MIC Prevents Prostate Tumor Arising in Vivo
Katie L. Atteridge, Jun Wang, Stephen R. Plymate, and Jennifer D. Wu
University of Washington

P31-14 Polarized DC1-based Vaccine against Prostate Cancer
Robbie Mailliard[1], Gurkamal Chatta[2], and Pawel Kalinski[1]
[1]*University of Pittsburgh,* [2]*University of Pittsburgh Cancer Institute*

P31-15 Prostate Stem Cell Antigen Vaccination Induces a Lifelong Protective Immune Response against Prostate Cancer in the Absence of Autoimmunity
W. Martin Kast[1], Andrew Gray[1], Bolyn Hubby[2], Otto J. Klinger[1], Maria de la Luz Garcia-Hernandez[1]
[1]*University of Southern California, Keck School of Medicine,* [2]*AlphaVax*

P31-16 Prostate Cancer Peptide Vaccines: Immune System Reconstitution to Generate Tumor Rejection Molecules
Ashok Badithe, Robert Suriano, Devyani Chaudhuri, Abraham Mittelman, and Raj K. Tiwari
New York Medical College

P31-17 Low Doses of Lentivirus-transduced DCs Overcome Self-tolerance and Protect against ERBB2-expressing Prostate Tumors

Miriam E. Mossoba[1], Jagdeep S. Walia[1], Vanessa I. Rasaiah[1], Jason E. Foley[2], Nicole Buxhoeveden[2], Daniel H. Fowler[2], and Jeffrey A. Medin[1]
[1]University Health Network, Toronto, [2]National Institutes of Health

Targeted Therapy

Sep 7 12:30 p.m.-2:30 p.m.
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Even-numbered: 1:30 p.m.-2:30 p.m.

P32-1 Targeting Defective Signal Pathways in Androgen-independent Prostate Cancer Using a Novel Delivery Vehicle
Jian Zhou, Jinhai Fan, Jennifer Stanfield, and Jer-Tsong Hsieh
University of Texas Southwestern Medical Center at Dallas

P32-2 A Novel Strategy for Prostate Cancer Therapy by Targeting STAT3 with G-Quartet Oligonucleotides
Priya Weerasinghe, Yifei Li, Qiqing Zhun, Yongli Guan, and Naijie Jing
Baylor College of Medicine

P32-3 Antisense MDM2 and Adenoviral E2F1 Sensitize Prostate Cancer Cells to Androgen Deprivation and Radiation
Thirupandiyur S. Udayakumar[1], Paul Hachem[1], Sudhir Agrawal[2], and Alan Pollack[1]
[1]Fox Chase Cancer Center, [2]Idera Pharmaceuticals, Inc.

P32-4 The "Combi-Targeting" Concept: A Novel Cell Signaling-based Approach for the Therapy of Advanced Prostate Cancer
Juozas Domarkas, Quyu Qiu, Ranjita Banerjee, and Bertrand J. Jean-Claude
McGill University

P32-5 Hormone Conjugates of Membrane-disrupting Peptides Target and Destroy Prostate Cancers and Their Metastases
William Hansel[1], Fred Enright[2], and Carola Leuschner[1]
[1]Pennington Biomedical Research Center, [2]Louisiana State University School of Veterinary Science

P32-6 Control of Micrometastatic Prostate Cancer Using Bi-213-labeled Multiple Targeted Alpha Therapy
Yong Li, Syed M. Abbas Rizvi, Emma Song, Paul J. Cozzi, Carl A. Power, Barry J. Allen, and Pamela J. Russell
St. George Hospital, University of New South Wales, Australia

P32-7 Differential Efficacy of Combined Therapy with Radiation and AEE788 in High and Low EGFR-expressing Androgen-independent Prostate Tumor Models
Kenneth J. Niermann, Christopher Willey, Michelle Reyzer, Dinesh Thotala, Arthur Fleishcher, Richard Caprioli, Dennis E. Hallahan, Dong Wook, Nathan Kim, and Jessica Huamani
Vanderbilt University Medical Center

P32-8 Antivascular Effects of VEGFR Inhibition Combined with Radiotherapy in Human DU145 Prostate Xenografts
Scott F. Paoni and Bruce M. Fenton
University of Rochester Medical Center

P32-9 Androgen Receptor-targeted Taxane Analogs for Androgen-independent Prostate Cancer
William L. Farrar[1], Jun Qi[2], David G.I. Kingston[2], and Nima Sharifi[1]
[1]National Cancer Institute, [2]Virginia Polytechnic Institute and State University

P32-10 Vascular Targeting Antibody Improves Chemotherapy of Prostate Cancer
Yi Yin[1], Xianming Huang[1], Connie Chang[2], Steven W. King[2], and Philip E. Thorpe[1]
[1]University of Texas Southwestern Medical Center at Dallas, [2]Peregrine Pharmaceuticals, Inc.

P32-11 Treatment of Prostate Cancer by Targeting Vascular Endothelial Growth Factor Receptors and Micrometastases with Bismuth-213 Labeled Vectors
Emma Y. Song[1], Julia Beretov[1], Chand Raja[1], Alfred Morgenstern[2], Christos Apostolidis[2], Barry J. Allen[3], Syed M. Abbas Rizvi[3], and Pamela J. Russell[3]
[1]St. George Hospital, [2]European Commission Joint Research Center, [3]University of New South Wales

P32-12 Vatuximab: Optimizing Therapeutic Strategies for Prostate Cancer Based on Dynamic MR Tumor Oximetry
Ralph P. Mason[1], Weina Cui[1], and Dawen Zhao[1], Albert J. van der Kogel[2], Johan Bussink[2], Jesús Pacheco Torres[3], Jennifer McAnally[1], Linda Watkins[1], Peter Peschke[4], and Philip Thorpe[1]
[1]University of Texas Southwestern Medical Center at Dallas, [2]University Medical Center Nijmegen, Netherlands, [3]Instituto de Investigaciones Biomédicas "Alberto Sols," Madrid, Spain, [4]German Cancer Center, Heidelberg, Germany

P32-13 Monotherapy with a Tumor-targeting Mutant of *S. typhimurium* Cures Orthotopic Metastatic Mouse Models of Human Prostate Cancer
Jack Geller, Huaiyu Ma, Meng Yang, Robert M. Hoffman, and Ming Zhao
Anticancer Incorporated

P32-14 Prostate-specific Membrane Antigen (PSMA): An Ideal Target for Developing Radiolabeled Monoclonal Antibodies for Diagnosis and Therapy
Stanley J. Goldsmith, Peter M. Smith-Jones, Neil H. Bander, and Shankar Vallabhajosula
New York-Presbyterian Hospital and Weill Medical College of Cornell University

Clinical Trials

Sep 7 12:30 p.m.-2:30 p.m.
Odd-numbered: 12:30 p.m.-1:30 p.m.
Even-numbered: 1:30 p.m.-2:30 p.m.

P33-1 Induction of PSA-specific CD8+T Lymphocytes in HLA-a2+ Patients with Prostate Cancer by Peptide Vaccination
Sigrun Hallmeyer, Hui Xie, Samarth Reddy, Mahmud Nadim, Linda Bressler, Supriya Perambakam, and David Peace
University of Illinois, Chicago

P33-2 ^{90}Yttirum-Dota-J591, a Radiolabeled Monoclonal Antibody Specific to the Extracellular Domain of Prostate Specific Membrane Antigen (PSMA): Radioimmunotherapy (RIT) Phase I Dose Escalation Studies in Patients with Prostate Cancer

Stanley J. Goldsmith, Neil H. Bander, Mathew I. Milowsky, David M. Nanus, and Shankar Vallabhajosula
New York-Presbyterian Hospital and Weill Medical College of Cornell University

P33-3 A Randomized Phase II Study of ^{153}Sm-EDTMP (Quadramet®) with or without a PSA/Tricom Vaccine in Men with Androgen-independent Prostate Cancer Metastatic to the Bone

Philip M. Arlen, Ravi Madan, James Hodge, William L. Dahut, Jeffrey Schlom, and James L. Gulley
National Institutes of Health

P33-4 Neoadjuvant Anti-angiogenesis Therapy in Men with High-grade and Locally Advanced Prostate Cancer Undergoing Prostatectomy Including a Subgroup Analysis of Men of African American Descent

Christopher Starks[1], Charles Brendler[2], and Mitchell H. Sokoloff[3]
[1]University of Chicago, [2]Northwestern University, [3]Oregon Health & Science University

P33-5 Phase I Trial of Anti-PSMA Designer T Cells in Advanced Prostate Cancer

Richard P. Junghans
Roger Williams General Hospital

P33-6 Intra-operative Dosimetry in Prostate Brachytherapy

Ameet Jain, Anton Deguet, Iulian Iordachita, Gouthami Chintalapani, Jack Blevins, Yi Le, Elwood Armour, Clif Burdette, Danny Song, and Gabor Fichtinger
Johns Hopkins University, Acoustic MedSystems, Inc., Johns Hopkins University School of Medicine

P33-7 Locally Advanced Prostate Cancer—Results from a Prospective Phase 2 Trial of Intermittent Androgen Suppression for Men with Evidence of PSA Relapse after Radiotherapy

Juanita Crook[1], S. Larry Goldenberg[2], Nicholas Bruchovsky[3], and Laurence Klotz[4]
[1]Princess Margaret Hospital, Toronto, [2]University of British Columbia, [3]Vancouver General Hospital, [4]Sunnybrook and Women's College Health Science Centre

POSTER/AUTHOR INDEX

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FACULTY DISCLOSURE STATEMENT

Last Name	First Name	Grant/Research Support	Consultant	Stock Shareholder (directly purchased excluding diversified mutual funds)	Honorarium	Other Financial or Material Support	MD Nurse's Disclosure
Allison	James	None	Medarex; Cell Genesys; Bristol Myers Squibb	None	None	None	Consultant with Medarex; Cell Genesys; Bristol Myers Squibb
Bertinuson	Anne	None	None	None	None	None	None
Blank	Thomas	None	None	None	None	None	None
Bolden	Greg	None	None	None	None	None	None
Bright	William	None	None	None	None	None	None
Carey	Robert	None	None	None	None	None	None
Carpten	John	None	None	None	None	None	None
Catalona	William	Beckman-Coulter, Incorporated	Beckman-Coulter, Incorporated	None	Beckman-Coulter, Incorporated	None	Beckman-Coulter, Incorporated (grants, consultant, honorarium)
Chinnaiyan	Arul	Gen-Probe, Inc.	Gen-Probe, Inc. and Compendia Biosciences	Compendia Biosciences	None	None	Gen-Probe, Inc. (grant, consultant) and Compendia Biosciences (consultant, stock shareholder)
Demark-Wahnefried	Wendy	None	None	None	None	None	None
DeMarzo	Angelo	None	None	None	None	None	None
deVere White	Ralph W.	National Cancer Institute and AminoUp Chemical Co., Japan	None	Pfizer	None	None	None
Drake	Charles	Cell Genesys, Inc.	Dendreon Inc, Bristol Myers Squibb	None	Cell Genesys Inc, Medarex Inc., Cerus Corporation	Under a licensing agreement between Cell Genesys Inc. and the Johns Hopkins University; the university is entitled to milestone payments and royalties on the sale of immunotherapy products. The terms of this arrangement are being managed by the Johns Hopkins University in accordance with its conflict of interest policies.	Cell Genesys, Inc (grants, honorarium); Dendreon Inc, Bristol Meyers Squibb (consultant); Medarex Inc., Cerus Corporation (honorarium)
Freedland	Stephen	Atkins Foundation	AstraZeneca	None	AstraZeneca	None	None
Freeman	Harold	Walt Disney Foundation; Breast Cancer Research Foundation	None	None	None	None	None

Last Name	First Name	Grant/Research Support	Consultant	Stock Shareholder (directly purchased excluding diversified mutual funds)	Honorarium	Other Financial or Material Support	MD Nurse's Disclosure
Gelmann	Edward	None	Novacea	Genentech, GlaxoSmithKline	Pfizer	None	Genentech, GlaxoSmithKline (stock); Pfizer (honorarium); Novacea (consultant)
George	Daniel	Novartis	Novartis	None	None	None	Novartis (grants, consultant)
Giambarresi	Leo	None	None	None	None	None	None
Gillespie	Richard	None	None	None	None	None	None
Giovannucci	Edward	None	None	None	None	None	None
Gleave	Martin	National Cancer Institute, National Institutes of Health, AstraZeneca, OncoGenex	AstraZeneca; Sanofi Aventis; OncoGenex	OncoGenex	None	None	None
Grönberg	Henrik	None	None	None	None	None	None
Harisinghani	Mukesh	None	None	None	None	None	None
Ho	Shuk-mei	National Institutes of Health, Department of Defense	None	None	None	None	None
Howe	Richard	Tex Us TOO	American Medical Systems Incorporated	None	None	None	American Medical Systems Incorporated (consultant)
Howe	Desiree	Tex Us TOO	American Medical Systems Incorporated	None	None	None	American Medical Systems Incorporated (consultant)
Hricak	Hedvig	None	None	None	None	None	None
Isaacs	John	None	None	None	None	None	None
Jones	Lovell	None	None	None	None	None	None
Kaime	Melissa	None	None	None	None	None	None
Kiefert	James	None	None	None	Cell Genesys Inc., Medarex Inc., Cerus Corporation	None	None
Klein	Eric	None	None	None	None	None	None
Klotz	Laurence	None	None	None	AtraZeneca, Merck, Abbott, and Sanofi Aventis	None	None
Knight	Sara	Department of Veterans Affairs	Pacific Business Group on Health; Foundation for Informed Medical Decision Making	None	National Cancer Institute; Physicians Academy for Clinical and Management Excellence; Department of Defense	None	None
Kyprianou	Natasha	None	None	None	None	None	None
Latini	David	American Cancer Society	None	None	None	None	None
Liebert	Monica	None	None	None	None	None	None

Last Name	First Name	Grant/Research Support	Consultant	Stock Shareholder (directly purchased excluding diversified mutual funds)	Honorarium	Other Financial or Material Support	MD Nurse's Disclosure
Logothetis	Christopher	National Cancer Institute; Department of Defense; Prostate Cancer Foundation	ImClonel; Lilly Research Laboratories; Pfizer; Bristol Myers Squibb; Dendreon; ISIS Pharmaceuticals; Novartis Pharmaceuticals	None	Pfizer; Bristol Myers Squibb; Dendreon; ISIS Pharmaceuticals; Novartis Pharmaceuticals	The University of Texas MD Anderson Cancer Center	Listed as unknown; none by reviewer
McGuinness	James	None	None	None	None	None	None
McGuire	Marielena	None	None	None	None	None	None
Miller	Donald	National Institutes of Health	None	Antisoma	None	None	None
Miller	Theresa	None	None	None	None	None	None
Mohler	James	None	None	None	None	None	None
Mural	Jane	None	None	None	None	None	None
Nelson	Peter S.	None	None	None	None	None	None
Nelson	William	National Cancer Institute; Prostate Cancer Foundation	Merck; GlaxoSmithKline; Cell Genesys; Abbott Pharmaceuticals	Cell Genesys; ProQuest	None	Intellectual property licensed to Oncomethylome Sciences	National Cancer Institute; Prostate Cancer Foundation (grants); Merck; GlaxoSmithKline; Cell Genesys; Abbott Pharmaceuticals (consultant); Cell Genesys; ProQuest (stock); Intellectual property licensed to Oncomethylome Sciences (Other)
Nie	Shuming	None	None	None	None	None	None
Nissenberg	Merel	None	None	None	None	None	None
Penson	David	Dendreon, Amgen	Dendreon, Sanofi Aventis	None	None	Pfizer, Boehringer Ingleheim	None
Pienta	Kenneth J.	None	None	None	None	None	None
Pollak	Michael	None	None	None	None	None	None
Price	Marva	None	None	None	None	None	None
Prins	Gail S.	National Institutes of Health	None	None	None	None	None
Ratliff	Timothy	None	None	None	None	None	None
Reed	Eddie	Centers for Disease Control and Prevention	None	IBM	None	None	None
Roach	Mack	None	None	None	None	None	None
Robins	Diane	None	None	None	None	None	None
Rubin	Charles	None	None	None	None	None	None
Scher	Howard	None	None	Biogen; Innovive	MGI Pharma	None	Biogen, Innovive (stockholder)
Schröder	Fritz	Ell, Dutch Cancer Society	Terring Corporation	None	Yes (unclear which)	None	None
Scott	Lisa	None	None	None	None	None	None
Sheinfeld Gorin	Sherri	DOD, CDC	None	None	None	None	DOD, CDC
Sholes	Westley	None	None	None	None	None	None

Last Name	First Name	Grant/Research Support	Consultant	Stock Shareholder (directly purchased excluding diversified mutual funds)	Honorarium	Other Financial or Material Support	MD Nurse's Disclosure
Simons	Virgil	Sanofi-Aventis; GPC Biotech; VF Corporation; Healthcare Foundation of New Jersey	None	None	None	None	None
Simons	Jonathan	None	None	None	None	None	None
Slovin	Susan	None	SynAce	None	Novartis, Sanofi Aventis, AstraZeneca	None	None
Soule	Howard	None	OncoGenex Technologies; Ascenta Therapeutics; Attenuon; ProQuest Investments1; Epilepsy Therapy Development Project2; Semafore Pharmaceuticals; Novacea; Eaturna LLC; Knowledge Universe Health and Wellness Group3	OncoGenex Technologies; Ascenta Therapeutics; Semafore Pharmaceuticals; Novacea	None	CanFite Biopharmaceuticals	OncoGenex Technologies; Ascenta Therapeutics; Attenuon; ProQuest Investments1; Epilepsy Therapy Development Project2; Semafore Pharmaceuticals; Novacea ; Eaturna LLC; Knowledge Universe Health and Wellness Group3(consultant); OncoGenex Technologies; Ascenta Therapeutics; Semafore Pharmaceuticals; Novacea (stock); CanFite Biopharmaceuticals (other)
Sproat	William	None	Constella Health Sciences	None	None	None	None
Stampfer	Meir	None	None	None	None	None	None
Theisen	Patrick	None	None	None	None	None	None
Tindall	Donald	None	None	None	None	None	None
Vasquez	Manuel	Texas Us TOO!	None	None	None	None	None
Warlick	Christopher	None	None	None	None	None	None
Willey	John	None	None	None	None	None	None
Williams, Jr.	James E.	None	None	None	None	None	None
Williams, Jr.	James E.	None	None	None	None	None	None
Xu	Jianfeng	None	None	None	None	None	None
Young	Roland	None	None	None	None	None	None

CONTINUING MEDICAL EDUCATION (CME) FOR PHYSICIANS AND CONTINUING EDUCATION UNITS FOR NURSES

This educational activity is sponsored by the Occupational Health and Emergency Medical Training and Preparedness Office of Science Applications International Corporation (SAIC).

Educational Needs

The Department of Defense (DOD) Prostate Cancer Research Program (PCRP) Innovative Minds in Prostate Cancer Today (IMPaCT) meeting provides PCRP-funded investigators with a public forum to share the results of their DOD-funded investigations with others in the scientific community, policymakers, and the lay public. This meeting also allows investigators from different fields to share knowledge and ideas that could lead to novel approaches to difficult research problems.

The intent of this activity is to improve physicians', nurses', and researchers' competence in the area of prostate cancer.

Purpose Statement

An important need exists to provide physicians and nurses involved in prostate cancer prevention, diagnosis, treatment, or quality of life care with important updates to stay informed of new research and the most current technologies and treatments for prostate cancer. This activity is being given because prostate cancer is the most commonly diagnosed cancer in men, accounting for 30 percent of all cancers in men. The ability to cure prostate cancer decreases with disease progression; therefore, new detection, diagnostic, therapeutic, and quality of life research is needed to find prevention strategies and new cures or improve survival and life after prostate cancer. The IMPaCT meeting serves an identified educational need to provide PCRP-funded investigators with a public forum to share the results of their DOD-funded investigations with others in the scientific community, policymakers, and the lay public. The meeting allows investigators from different fields to share knowledge and ideas that could lead to novel approaches toward solving difficult research problems in prostate cancer.

Target Audience

This activity is designed for civilian and military physicians; nurses; and researchers, especially oncologists, radiologists, pathologists, general/family practitioners, and osteopaths who might be involved in patient diagnosis, treatment, prevention, or post-cancer care.

Learning Objectives

At the conclusion of this activity, participants should be able to:

- Describe and discuss recent research results and the innovative approaches that are now being used to study the basic biology, prevention, detection, diagnosis, and treatment of prostate cancer, and ways to improve patient quality of life.
- Discuss the latest advances in the genetics and biology of prostate cancer.
- Discuss the latest advances in the field of prostate cancer prevention.
- Interact and collaborate with prostate cancer researchers who work in different scientific and clinical disciplines and network with consumer advocacy organizations.

Faculty Disclosure

It is the policy of the Occupational Health and Emergency Medical Training and Preparedness Office of SAIC to require the disclosure of the existence of any relevant financial interest or any other relationship a faculty member including spouse/partner has with the manufacturer(s) of any commercial product(s) discussed in an educational presentation. The presenting faculty reported the disclosure of no relevant conflict of interest.

The lecture(s) may contain images of types of medical equipment available. These items are referred to as examples only. These materials make no endorsement or certification of any item or manufacturer of the equipment.

All participants who plan to apply for credit must sign in at the Continuing Education Booth EACH day of the meeting.

Commercial Support Disclosure

No commercial support is given to this activity.

Accreditation Statements

Physicians

"The Occupational Health and Emergency Medical Training and Preparedness Office of SAIC is accredited by the Accreditation Council for Continuing Medical Education (CME) to provide continuing medical education for physicians."

"The Occupational Health and Emergency Medical Training and Preparedness Office of SAIC designates this educational activity for a maximum of 14.5 *AMA PRA Category 1 credit(s)*^{TM}. Physicians should only claim credit commensurate with the extent of their participation in the activity."

Nurses

"This offering has been approved for a maximum of 14.5 contact hour(s) by the Maryland Nurses Association which is accredited as an approver of continuing education in nursing by the American Nurses' Credentialing Center's Commission on Accreditation."

Instructions for Credit

To receive continuing education credit for participation in this meeting, participants must validate participation in each session for which they request credit. Participants will receive an Exchange Voucher from the Registrar in each session room at the beginning of the session. At the completion of the session, participants must return the completed Exchange Voucher to the Registrar to receive credit. This information will be used to calculate credits, and certificates will be mailed within 6–8 weeks after the meeting.

Making the Most of IMPaCT: A Guide for Consumers

This guide is provided to maximize your benefit from attending the IMPaCT meeting. Scheduling is important because the first rule of attending a large scientific meeting is: **You cannot do it all.**

If you take a look at the draft program, you will see that the day begins at 7:00 a.m. and runs until the evening with only brief breaks. You will have to decide how important it is to attend every session. Within this schedule, there are symposia sessions where six sessions are running concurrently. You can only be in one place at a time.

Rather than making last-minute decisions on which session you will attend, plan ahead. When you register at the meeting, you will receive program and proceedings books with abstracts for every presentation. Reading the abstracts for your planned sessions will help you to refine your plan. Hint: after reading the abstracts, leave the large, heavy proceedings book in your hotel room rather than carrying it around to the sessions.

Give yourself a break.

Program some down time by skipping a session if necessary. If you are having a hard time following a session or you are most interested in only one speaker in the session, it is okay to leave quietly during the session. If you have a friend or colleague at the meeting, you can each attend different sessions and report back to each other later on what you learned. The proceedings book is a resource that you can take home to read later, and abstracts also will be available on the Congressionally Directed Medical Research Programs (CDMRP) website http://cdmrp.army.mil after the meeting.

What you can expect in different session types.

Plenary Sessions by definition are those where all meeting attendees are present. This does not mean that you are required to attend, but these sessions are held in a large room that can accommodate all attendees, and distinguished speakers who will attract a large audience are recruited for these sessions. The speakers may present an overview of a topic or their own new research results. There will be some time for questions and answers after the speakers' presentations, but because the audience is large, only a small percentage of the audience will have an opportunity to ask questions.

You will get the most out of these sessions if you are familiar with the topic areas being presented because some of the speakers will be presenting complex scientific ideas. This is particularly true of the Basic Research and Translational Research sessions, which

cover a wide range of topics. The Clinical Research session focuses on prostate-specific antigen (PSA) screening and monitoring for "expectant management" or "active surveillance" of prostate cancer, a hot topic in prostate cancer today. The Health Disparities session addresses the role of racial/ethnic, biological, and socioeconomic differences in prostate cancer incidence, treatment, and mortality. Finally, the Quality of Life session addresses decision making and other areas where quality of life for prostate cancer survivors can be improved.

Spotlight Sessions are a type of plenary session developed for the IMPaCT meeting to highlight collaborative projects of which the PCRP is especially proud. The North Carolina–Louisiana Prostate Cancer Project led by Dr. James Mohler is a large, multidisciplinary study designed to uncover reasons why prostate cancer mortality is higher in African Americans than Caucasian Americans. The PCRP Clinical Consortium led by Dr. Howard Scher provides the infrastructure to streamline clinical trial approval and patient recruitment to expedite testing therapeutic agents or approaches for the management or treatment of prostate cancer. The research consortium led by Drs. Jonathon Simons and Leland Chung has been called the "Manhattan Project" because it uses real-time interaction among collaborators all over the country to develop a plan of attack on metastatic, hormone-refractory prostate cancer, the "lethal phenotype."

Early Morning Educational Sessions offer an excellent opportunity for hearing in-depth presentations on scientific topics presented by experts in the field. These sessions are structured to allow about 20 minutes for questions and answers. Consumers attending other CDMRP-sponsored meetings have reported that these sessions offered the best chance to interact with the speakers.

Sessions 6, "Prostate Cancer 101," 26, "Prostate Cancer Screening," and 40, "Treatment and Management of Prostate Cancer" were developed specifically for the consumer audience.

Symposium Sessions offer 10-minute presentations by several PCRP-funded investigators on their recent research results, followed by questions and answers. You will get the most out of these sessions if you are familiar with the topic areas being presented, as they tend to be rapid presentations of complex scientific topics. The question-and-answer sessions (whether your own question or someone else's) are often very helpful in understanding the concepts presented. If you are finding it hard to choose between concurrent sessions, you might attend half of two different sessions.

Poster Sessions allow you to study at your own pace the investigators' research and results presented in a graphic format. Posters will be displayed for the entire meeting in the Grand Hall, and you can visit the hall anytime during the meeting. At the scheduled poster sessions at lunchtime on Thursday and Friday, the poster presenters will be standing near their posters to answer questions. The odd-numbered poster presenters will be available during the first hour, and the even-numbered poster presenters will be available during the second hour. Poster session P1, "Prostate Cancer Advocacy," presents advocacy and education activities of prostate cancer advocacy organizations. You may want to take advantage of the **Poster Tours for Consumers**, in which scientists will give groups of consumers a guided tour of selected posters. You can sign up for these tours at the Registration Desk.

Speaker Additions

Thursday, September 6, 2007

Symposium 13: Stem Cells. The consumer speaker will be Robert Carey of the Georgia Prostate Cancer Coalition and Men Coming Together.

Symposium 18: Epidemiology and Biomarkers. *Is Disparity in Prostate Cancer Rates among Different Ethnic Groups Associated with Well-done Meat Consumption and Specific Acetylator Genotypes.* Sangita Sharma will not be presenting.

Symposium 19: Preclinical Drug Discovery. The consumer speaker will be Norwood Sloan, American Cancer Society Outreach Specialist.

Symposium 21: Signal Transduction I. *Thioredoxin Reductase 1 Expression Coincides with the Onset of Androgen-independent Growth of Prostate Cancer*. James Mohler of Roswell Park Cancer Institute will replace Swaroop S. Singh.

Symposium 22: Training the Next Generation. Additional presentations will include:

Toxicity of Endothelin Receptor Antagonists on Prostate Cancer Cell Lines. Presented by Nikesha J. Haynes.

Imatinib Blocks the Prostate Cancer-induced Osteoblast Proliferation and New Bone Formation in Vitro. Presented by Lauren Wiggins.

Prostate Cancer: How Diet Affects Tumor Growth. Presented by Cymara Tolbert-Warren.

A Comparative Study of Genetic Susceptibility and Risk Factors for Men with and without Prostate Cancer. Presented by Nitrecus Simmons.

Adhesion-mediated Chemoresistance of PC3 Cells to Docetaxel. Presented by Osemeke Edobor.

Friday, September 7, 2007

Symposium 33: Tumor Suppressors. The consumer speaker will be Richard Gillespie of the Us TOO Chapter Cosponsored by the Westminster at Lake Ridge and Potomac Hospital.

Symposium 33: Tumor Suppressors. *Inflammatory Cytokines Induce Ubiquitination and Loss of Prostate Suppressor Protein NKX3.1*. Edward Gelmann of Columbia University will replace Mark Markowski.

Symposium 34: Targeting Apoptosis. The consumer speaker will be Westley Sholes of the California State Prostate Cancer Coalition.

Symposium 36: MultiCenter Collaborations for Clinical Trials. The consumer speaker will be Virgil Simons of the Prostate Net.

Saturday, September 8, 2007

Symposium 44: Signal Transduction II. The consumer speaker will be James Williams, Jr. of the Intercultural Cancer Council, Pennsylvania Prostate Cancer Coalition, and Alliance for Prostate Cancer Prevention.

Symposium 46: Collaborative Partnership Panel. The consumer speaker will be Quince Fleming, Jr., of Rex Healthcare. Panel particpants will be Omar Bagasra, Nagi Kumar, Robert Sikes, and Flora Ukoli.

Additional Poster Session

Summer Training Program
Sep 7 12:30 p.m.-2:30 p.m.
Odd-numbered 12:30 p.m.-1:30 p.m.
Even-numbered: 1:30 p.m.-2:30 p.m.

P34-1 **It Takes Two: Beauty and the BeHOLDen**
Lori A. Gordon
Florida A&M University, Tallahassee

P34-2 **Geographical and Racial Differences in the Quality of Life (QOL) of Men after Treatment for Localized Prostate Cancer**
Olivia R. Marks
Florida A&M University, Tallahassee

P34-3 **Determination of Endothelin Expression Levels in Prostate Cancer**
Bisola C. Awoyemi[2], Christine J. Weydert[1], Alison Esser[1], Ruth Mejia[1], Justin Drake[1], and Michael D. Henry[1]
[1]University of Iowa, [2]Lincoln University

P34-4 **Generation of CD8+ Memory T-Cells Following Tumor Vaccine Immunization**
Shaynah Browne[2], Jennifer Paisley[1], and David M. Lubaroff[1]
[1]University of Iowa, [2]Lincoln University

P34-5 **Socioeconomics Correlates of Prostate Cancer Incidence, Stage at Diagnosis and Survival**
Caroline Oliveira Dias[2], Margaret Voelker[1], Michele West[1], Gerard Rushton[1], and Elizabeth Chrischilles[1]
[1]University of Iowa, [2]Lincoln University

P34-6 **A Comprehensive and Global Genomic Assessment of P53-regulated Transcriptional Targets**
Steve Manduku[2], Nicole L. Pinaire[1], Kevin B. Spurgers[1], Raymond Meyn[1], and Timothy J. McDonnell[1]
[1]M.D. Anderson Cancer Center, University of Texas, [2]Texas Southern University

P34-7 **ProstaScint – The New Wave of Prostate Cancer Detection**
Michael Bannister[1], Marva M. Price[2], Thomas J. Polascik[3], Vladimir Mouraview[3], and Janice Mayes[4]
[1]North Carolina Central University, [2]Duke University School of Nursing, [3]Duke University Medical Center [4]Bennett College for Women

P34-8 **Do RECIST or WHO Criteria Give a More Accurate Assessment of Treatment Response in Solid Tumors?**
Sharhonda Harvey[2], Marva M. Price[1], Daniel J. George[3], and Patrica Creel[3]
[1]Duke University School of Nursing, [2]North Carolina Central University, [3]Duke University Medical Center

P34-9 **The Roles of RhoG, Rac1, and Rac3 GTPase in PC-3 Human Prostate Cancer Tumor Cells**
Diapedesis Mashariki Kabaila[2], Moumita Chatterjee[2], and Kenneth van Golen[1]
[1]University of Delaware, [2]Lincoln University

P34-10 **Establishing Dose-Response Curves for Chemotherapeutics in Prostate Cancer: Baseline Data for Synergistic Drug Interactions**
Brenda Mogere[2] and Robert A. Sikes[1]
[1]University of Delaware, [2]Lincoln University

P34-11 **Investigating the Effects of Disease and Stress on Cytokine and Hormone Expression in the Brain**
Bryan Mayfield[2], Katron Bloomfield[3], and Harlan P. Jones[1]
[1]University of North Texas Health Science Center, Fort Worth, [2]University of Houston, [3]University of Louisiana, Monroe

P34-12 **Isolation of Apo A-I, a Component of the Rhdl Drug Delivery System, a Novel Approach for Cancer Chemotherapy**
Gima Mudoh[2], Andrass Lack[1], and Maya Nair[1]
[1]University of North Texas Health Science Center, Fort Worth, [2]Tuskegee University

P34-13 **Effect of PKCη Phosphorylation on Its Stability**
Je'Kel Smith[2], Shalini Persaud[1], and Alakananda Basu[1]
[1]University of North Texas Health Science Center, Fort Worth, [2]Texas Southern University

P34-14 **Role of Shp-1 in Enhancing Dendritic Cell-based Anti-Tumor Vaccines for Prostate Cancer**
Alem Tewoldeberhan[2] and Jonathan M. Levitt[1]
[1]Baylor College of Medicine, [2]Prairie View A&M University

P34-15 **Enhancing 1,25 Dihydroxyvitamin D3 Action in Prostate Cancer Cells**
Jerecia E. Watson[2], Michele N. Washington[1], and Nancy L. Weigel[1]
[1]Baylor College of Medicine, [2]Prairie View A&M University

P34-16 **Recruitment of Reactive Stroma in Prostate Cancer Progression**
Mark Anthony Williams II[2] and David Rowley[1]
[1]Baylor College of Medicine, [2]Prairie View A&M University

Changes to Posters

P10-2 *The Function of Rex1 in Human Prostate Epithelial Cells*. Authors are Mi-Young Lee, Lorraine Gudas, and Chunyang Zheng, Cornell University, Weill Medical College.

P11-11 *Complete Restoration of Cell Surface Activity of Transmembrane-truncated Membrane-type Matrix Metalloproteinase-1 by a Glycosylphosphatidylinositol Anchor: Implications for MT1-MMP Activity in Cell Invasion in Three-dimensional Matrix.* Authors are Jianbo Yang[1], Jing Nie[2], Jing Pei[1], Malcolm Blumenthal[3], and Duanqing Pei[1], [1]University of Minnesota, Twin Cities, [2]Burnham Institute, [3]University of Minnesota Medical School.

P22-10 *Prostate Cancer Risk Associated with Ambient Pesticide Exposure in California's Central Valley* will be attended by author Myles Cockburn during the Thursday poster session.

Posters Withdrawn from Poster Sessions

P2-1 *Decisions and Outcomes in Early Prostate Cancer: Stories Men Tell of Meeting the Challenge of Choosing Their Treatment*. J. Clark et al.

P4-12 *Interrogating Chromosome 12 for Prostate Cancer Susceptibility Genes in African Americans Using an Admixture Mapping Approach*. C. Bonilla et al.

P6-25 *Cub and Sushi Multiple Domains1 in Prostate Cancer*. A. Dibner et al.

P11-7 *Characterization of a Novel Protein, Lyric, and Its Potential Role as a Mediator of Prostate Tumor Cell Migration and Invasion*. S. Ash et al.

P16-1 *Peroxisome Proliferator-activated Receptor-Delta Antagonism as a Therapeutic Strategy for Prostate Cancer*. N. Zaveri et al.

P18-4 *Androgen Signaling Axis as Targets of Selenium Anticancer Action*. S. Liu et al.

Abstracts

S22-6: Toxicity of Endothelin Receptor Antagonists on Prostate Cancer Cell Lines

Nikesha J. Haynes[1], Joshua R. Danke[1], and Michael D. Henry[2]
[1]Lincoln University
[2]University of Iowa

Endothelins (ETs) and their receptors – the ET axis – play vital roles in the health and function of normal tissue. Tumor progression seems to be promoted by ET receptor activation. The mechanisms it adopts are: inhibition of apoptosis in cancer cells, matrix remodeling, and bone deposition in skeletal metastasis through the activation of osteoblasts. ET also contributes to angiogenesis. The interaction between the ETs (ET-1, ET-2, and ET-3) and their receptors (ETA and ETB) plays a role in the metastasis of cancer cells. Previous experiments in our lab showed that antagonists of ET receptors were able to suppress the metastasis of prostate cancer in SCID mice. However, it was not certain if this observed effect was because of the effects of these drugs on the tumor microenvironment or direct action on the cancer cells. We first determined the growth curves of prostate cancer cell lines 22rv1 and PC-3 in 96 well plates in the presence and absence of serum, and we measured the percentage of viable cells using WST assays. This involved the incubation of cells with WST-1 followed by the spectrophotometric assay of the colored product. Next, in order to ascertain if there was a direct action of the drug on the cells, we did dose treatments in the presence and absence of serum ranging from 0.01µM to 0.01x10-6µM using ETA receptor antagonist (Atrasentan), ETA&B receptor antagonist (A-182 086), ETB receptor antagonist (A-192 621), and Paclitaxel as a positive control. There was no effect seen with the in vitro treatment of these cell lines with the drugs. Hence, there is no conclusive evidence to prove that the previous results were because of direct blockade of ET receptors on prostate cancer cells. Thus, the prevention of metastasis may have been as a result of the action of the drug on the tumor microenvironment. With prostate cancer being the second leading cause of death among men in the United States, more research is necessary in order to bring about a significant change in this area. Death from prostate cancer is not a result of primary tumor growth, but because of its ability to metastasize to secondary sites, namely bone, liver, lung, and other sites. Further discovery of ET's role in the metastasis of prostate cancer and the stage at which its effect is greatest will provide insight into the development of drugs to decrease and/or prevent the metastasis of prostate cancer.

The U.S. Army Medical Research and Materiel Command under W81XWH-06-1-0266 supported this work.

S22-7: Imatinib Blocks the Prostate Cancer-induced Osteoblast Proliferation and New Bone Formation in Vitro

Lauren Wiggins[1] and Nora Navone[2]
[1]Texas Southern University
[2]University of Texas M.D. Anderson Cancer Center

Background: Understanding the mechanisms underlying the osteoblastic bone metastases of prostate cancer may serve as a rational basis for therapy design. Preclinical and clinical studies suggested that Imatinib mesylate modulates the prostate cancer-bone interaction and prostate cancer sensitivity to chemotherapy through inhibition of platelet-derived growth factor receptor (PDGFr).

Objectives: To assess the role of prostate cancer cells-osteoblast interaction in the sensitization effect of Imatinib to chemotherapy.

Methods: We assessed the effects of Imatinib on prostate cancer-induced osteoblast proliferation, AP activity, bone formation, and sensitization of prostate cancer cells to Docetaxel.

Results: Imatinib had a growth inhibitory effect in osteoblast proliferation and new bone formation; marked inhibition of the prostate cancer-induced osteoblast proliferation but with only a marginal effect in total alkaline phosphatase activity in osteoblasts. Prostate cancer cells produced PDGF-AB, and PDGF induced osteoblast proliferation but not alkaline phosphatase activity. Imatinib had no effect on the growth of prostate cancer cells alone or in coculture with osteoblasts. Finally, Imatinib did not increase the cytotoxic effect of Docetaxel on prostate cancer cells growing alone or in coculture.

Conclusions: Imatinib regulates the prostate cancer-induced osteoblast proliferation and new bone formation but not differentiation. Imatinib regulation of prostate cancer-induced osteoblast proliferation is not sufficient to control prostate cancer growth or to sensitize prostate cancer cells to Docetaxel. Decreases in alkaline phosphatase secondary to Imatinib are likely to result from a reduction in osteoblast numbers and thus identify a subpopulation of patients with effective response to Imatinib.

The U.S. Army Medical Research and Materiel Command under W81XWH-05-1-0172 supported this work.

S22-8: Prostate Cancer: How Diet Affects Tumor Growth

Cymara Tolbert-Warren[1], Marva M. Price[2], and Stephen J. Freedland[3]
[1]Bennett College for Women
[2]Duke University School of Nursing
[3]Duke University School of Medicine

Background: Diet and nutrition are critical components of health maintenance. One-third of cancer deaths in the United States may have been prevented in part by healthier diets. Previous research has shown that a ketogenic diet slows tumor growth and keno bodies inhibit cancer. If the brain lacks glucose, ketone bodies are produced to provide the brain with glucose. Ketosis may be induced by two methods: depriving the body of food or consuming a diet low in glucose.

Purpose: The purpose of this study is to induce ketosis through a specific diet in mice. Once the proper diet is identified, the diet will be replicated and tested in humans.

Methods: One hundred and five Severe Combined Immune Deficiency (SCID) mice were injected with LAPC-4 xenografts. The mice were randomized into 7 groups of 15, and each group was placed on different variations of a Western diet at different caloric levels. Out of the seven groups, two were given an ad-lib amount of food but had different feeding schedules. Three of the groups were fasted different times during the week, and three were fed a restricted diet. All food was administered to the mice on different feeding schedules. Food measurements were recorded daily. Tumor sizes and weights for each mouse were measured biweekly. Mice were euthanized when the volume of the tumor averaged out to 1,500 mm or more. The mice were also euthanized if ulcerations of the tumor occurred. From each mouse euthanized, blood samples along with the liver, the tumor, and the prostate were collected for further examination and research.

Results: The mice that were fasted the most during the week and placed on a restricted diet had the highest survival rate. Mice from the other groups died sooner because they failed to achieve the same level of ketosis despite their low caloric intake.

Discussion: The group of mice that were fasted and placed on a restricted diet lived the longest because they became ketotic. Ketone bodies slow the growth of their tumors. Therefore, a diet lower in calories or even a fasting diet increases the rate of survival and slows tumor growth.

IMPaCT: This research has the potential of impacting the prevention not only for prostate cancer, but all cancers. If people understand the importance of a healthy diet and are equipped with the knowledge and tools to make the proper lifestyle changes, it could initially mean higher survival rates and a slowing and reduction in tumor growth. The long-term ramifications of this research would be understanding cancer and developing successful methods for prevention until a cure is found.

The U.S. Army Medical Research and Materiel Command under W81XWH-05-1-0209 supported this work.

S22-9: A Comparative Study of Genetic Susceptibility and Risk Factors for Men with and without Prostate Cancer

Nitrecus Simmons[1], Marva M. Price[2], and Stephen J. Freedland[3]
[1]Bennett College for Women
[2]Duke University School of Nursing
[3]Duke University School of Medicine

Background: Previous studies have shown that genetic variations of the IGF1 gene and differences in serum occur in men both with and without prostate cancer; however, they have shown limited data among minorities. In addition, researchers have found that the repeated occurrence of the homozygous IGF1 gene was much lower in African American men than Caucasian men, which may explain the increased prostate cancer incidence in black men versus white men.

Purpose: The aim of this study is to compare predisposing genetic factors for men with prostate cancer versus men without prostate cancer to determine those risk factors that will predict a greater likelihood of a positive biopsy among minorities undergoing a prostate needle biopsy.

Methods: The 32 patients in this study consisted of minorities ranging from of 40 to 70 years of age and were chosen from the Durham VA Medical Center. The patients were chosen based on PSA (prostate specific antigen) and DRE (digital rectal exam) exams and placed in groups depending on the results of their prostate needle biopsy. Once patients had consented, questionnaires and blood samples were collected for analysis. DNA was extracted to identify and compare potential predisposing genetic factors. Information on other factors such as physical activity, dietary eating habits, and serum was also collected, and they were examined as contributors to prostate cancer. In conducting this study, we looked for subtle differences in the DNA to show what genes are susceptible in men with and without prostate cancer.

Results: It is expected that serum IGF1 (insulin growth factor) will be higher in men with prostate cancer and lower in men without prostate cancer. IGF1 is a factor that is responsible for cellular growth, multiplication, and replacement in adults. The best predictor for determining the likelihood of prostate cancer was the presence of different forms of the IGF1 gene in DNA.

Conclusion: In conclusion, men with prostate cancer are more likely to have a high IGF1 due to the growth and metastases of prostate cancer cells. Therefore, genes (IGF1) are correlated to prostate cancer in that their presence in DNA causes cancer cells to grow at a rapid pace.

IMPaCT: This research will provide knowledge of the increased vulnerability to prostate cancer in minorities due to genes, other contributing risk factors, and ways of preventing prostate cancer in advance. Understanding how genes may make some men more susceptible to prostate cancer helps researchers develop tests to determine the necessary forms of screening and treatments for potential prostate cancer patients.

The U.S. Army Medical Research and Materiel Command under W81XWH-05-1-0209 supported this work.

S22-10: Adhesion-mediated Chemoresistance of PC3 Cells to Docetaxel

Osemeke Edobor[1], Freddie Pruitt[1], Robert A. Sikes[2], Kenneth van Golen[2], Mary C. Farach-Carson[2], and Carlton Cooper[2]
[1]Lincoln University
[2]University of Delaware

Men who develop metastatic prostate cancer (PCa) and fail androgen ablation therapy rely on docetaxel (taxotere) as the next therapy of choice. Unfortunately, patients frequently relapse after developing docetaxel chemoresistance. Understanding the cellular mechanism that underlies chemoresistance could improve treatment response for bone metastatic PCa. To determine if type I collagen, which comprises 90% of the bone extracellular matrix (ECM), contributes to chemoresistance, we used an androgen-independent bone metastatic PCa cell line, PC3. PC3 cells preferentially activate survival pathways during adhesion to type I collagen (Kiefer et al, 2001). We hypothesize that adhesion of PC3 cells to type I collagen mediates the chemoresistance to docetaxel. Our data show that PC3 cells, when adhered to type I collagen, show an increase in p-Akt as compared to PC3 cells on fibronectin and plastic. MTT analysis shows that PC3 cells on type I collagen are more viable in response to increasing concentrations of docetaxel. Western blot analysis shows that type I collagen inhibits the activation of the apoptosis effector Caspase 7. We believe that type I collagen protection is mediated by signaling between P13-kinase and Akt, and treatment of PC3 with P13-kinase inhibitor LY-294002 was able to negate the protective effects of type I collagen. In essence, we conclude that adhesion of PCa cells to components of the bone microenvironment may be a critical component of the acquired docetaxel chemoresistance seen in men with bone metastatic PCa.

The U.S. Army Medical Research and Materiel Command under W81XWH-06-1-0244 supported this work.

P34-1: It Takes Two: Beauty and the BeHOLDen

Lori A. Gordon
Florida A&M University

Innovative interventions, specifically aimed at, and conducive with, the African American community, are key to address the high incidence and mortality rate of prostate cancer in African American men. The beauty salon provides an ideal community setting for the dissemination of health information: the stylist is presented as a key conduit of information. Moreover, African American women serve as "gatekeepers" of the health of the family, commanding a strong influence over the receipt and implementation of health information for men. The relationship between the stylist and client and the environment of the salon encourage oral dialogue, a preferred method of communication among African Americans. To the researcher's knowledge, there have been no prostate cancer interventions or other men's health topics situated within beauty salons. Thus, the overall goal of this exploratory pilot study is to assess the feasibility of the beauty salon as a novel setting to communicate prostate cancer information. Specifically, the aims are to (1) assess the current level of prostate cancer awareness among African American women and (2) explore the feasibility of utilizing beauty salons as an innovative community educational setting to communicate prostate cancer information (by assessing interest, preferred communication methods, and the potential for transfer). The use of live, theatrical performances will also be examined. The study methods are face-to-face interviews among 45 self-identified African American women (15 stylists, 30 clients) and observations of the salon atmosphere. Each survey has an awareness and a feasibility component. The results from this study will be used as a basis for further studies and possible communication interventions.

The U.S. Army Medical Research and Materiel Command under W81XWH-06-1-0295 supported this work.

P34-2: Geographical and Racial Differences in the Quality of Life (QOL) of Men after Treatment for Localized Prostate Cancer

Olivia R. Marks
Florida A&M University

Background/Purpose: Prostate cancer and associated treatments cause a significant burden of suffering in American men. Due to advances in early detection and treatment options for early-stage, localized prostate cancer, quality of life (QOL) outcomes have become a major topic of research. However, to date there has been little prospective research on racial differences in QOL between African Americans and Caucasians before and after treatment for localized prostate cancer. Furthermore, there has been no research exploring differences in QOL based on geographical regions of the United States. Thus, this study may be the first to examine racial and geographical differences in QOL in men with localized prostate cancer using a longitudinal design.

Methods: Baseline (pretreatment) and post-treatment data from CaPSURE, a longitudinal observational database, will be analyzed. African American and Caucasian patients who were treated from 1995 to 2006 for localized prostate cancer, regardless of treatment type, will be included in the study. Patients who have locally advanced or metastatic prostate cancer and are not African American or Caucasian will be excluded from this study. Patients

will have completed QOL assessments pretreatment and at 6 and 12 months after treatment using the RAND SF-36 and the UCLA-Prostate Cancer Index (PCI) questionnaires. Descriptive statistics (frequencies, means, standard deviations) will be used to summarize demographic, disease, and treatment information of the study population. In addition, inferential statistical analyses (including correlational and multiple regression) will be conducted as appropriate to address the objectives of this study. Specifically, these analyses will assess racial differences, explore geographic differences, and document magnitude of changes in QOL over time (pre/post-treatment). Results will be reported to address the void in the published literature.

The U.S. Army Medical Research and Materiel Command under W81XWH-06-1-0295 supported this work.

P34-3: Determination of Endothelin Expression Levels in Prostate Cancer

Bisola C. Awoyemi[1], Christine J. Weydert[2], Alison Esser[2], Ruth Mejia[2], Justin Drake[2], and Michael D. Henry[2]
[1]Lincoln University
[2]University of Iowa

Endothelin-1, a potent vasoconstrictive peptide, has been found to be associated with cancer and tumor growth. ET-1 is secreted by prostate cancer cells and may act in an autocrine fashion by binding to ET receptors on prostate cancer cells or in a paracrine fashion by binding to receptors on other cell types in the tumor. The endothelin axis is thought to play a role in proliferation, angiogenesis, apoptosis, and metastases. In prostate cancer, ET-1 and the ET(A)R have been particularly implicated in the proliferation and metastases of cancer cells in bone. Previous studies from our lab have shown that forced overexpression of ET-1 stimulated prostate cancer cell proliferation in vitro, but paradoxically reduced prostate tumor growth in mice. To determine if there is a correlation between endothelin-1 expression level and prostate cancer growth, we sought to find prostate cancer cell clones secreting endothelin at various levels via endothelin enzyme-linked immunosorbent assay, analyze the growth changes using WST-1 assay, and then inject nude mice with these clones to measure tumor growth over time. We found that ET-1 levels were not correlated with growth rates. However, unlike cells in which ET-1 was overexpressed, the levels of ET-1 secreted into conditioned medium by the clones were below the threshold for activation of the ET(A)R receptors. Thus, in vitro, autocrine growth stimulation in these clones via ET-1 is not likely.

The U.S. Army Medical Research and Materiel Command under W81XWH-06-1-0266 supported this work.

P34-4: Generation of CD8+ Memory T Cells following Tumor Vaccine Immunization

Shaynah Browne[1], Jennifer Paisley[2], and David M. Lubaroff[2]
[1]Lincoln University
[2]University of Iowa

Prostate cancer is the most commonly diagnosed cancer in the United States, with an estimated 234,460 new cases each year. Once a patient develops androgen-independent prostate cancer, the disease is fatal. Several immunotherapeutic approaches, including BCG (Bacillus Calmette-Guerin) injection, virus therapy, dendritic cell therapy, and Interleukin (IL-2), are being investigated as novel approaches to treating these patients. Of particular interest to our lab is the use of a recombinant adenovirus to activate the immune system to develop an anti-PSA (prostate specific antigen) response. Previous work in our lab has demonstrated the same effect with the tumor model antigen ovalbumin (OVA). In both cases, tumor destruction was facilitated by CD8+ Cytotoxic T-Lymphocytes (CTLs). Also under investigation is the use of adjuvant, such as non-methylated CpG ODN (oligodeoxynucleotide). The data show that when used with adenovirus immunization, CpG ODNs result in an increased anti-tumor response when compared to adenovirus alone, despite a reduction in in vitro CTL activity. One hypothesis for the enhanced tumor protection despite reduced CTL activity is that CpG may effect the development of memory and effector T cells. In recent studies of the effect of CpG ODNs on the development of memory T cells, it was demonstrated that memory T cells were reduced when immunized with Ad5-OVA + CpG compared to Ad5-OVA. This project focused on the generational differences of memory CD8+ T cells after immunization with Ad5-OVA or Ad5OVA + CpG. It is hypothesized that a reduction in CD8+ memory T cells will be seen when immunized with Ad5-OVA+ CpG ODN. To test this hypothesis, mice were immunized with Ad5-OVA and Ad5-OVA+CpG. The percentage of OVA-specific CD8+ cells was determined by flow cytometry analysis of tetramer CD8, CD127, CD44, CCR7 staining. Our data show a reduction in OVA-specific memory CD8+ T cells when immunized with AdOVA+ CpG ODN. Future studies will focus on the changes of OVA-specific CD8+ T cells in lymph node lymphocytes compared to spleen-derived lymphocytes.

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P34-5: Socioeconomics Correlates of Prostate Cancer Incidence, Stage at Diagnosis and Survival

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Background: Socioeconomic factors have been associated with incidence and mortality of prostate cancer. Incidence of prostate carcinoma in Iowa is slightly lower than the U.S. average. However, the mortality is slightly greater than the national average.

Objectives: To examine the incidence, stage of diagnosis, and mortality of prostate cancer in the Iowa population; to theorize possible reasons for observed patterns; and to suggest possible cancer control activities.

Methods: We used the following: SEER*Stat, statistical software by SEER; cancer data from the Iowa Cancer Registry; county border data from the Natural Resources GIS library; and American Cancer Society.

Results: Early stage of diagnosis has been detected at earlier ages, which suggests that PSA (prostate specific antigen) screening may have an important role in controlling this disease. Men diagnosed with prostate cancer at earlier stage and ages have relatively higher survival compared to men diagnosed at late stage and age. Men with less education and lower health insurance have less PSA screening. The southwest lower quadrant of Iowa seems to be an area of a particular concern due to the low incidence and high proportion of cases diagnosed at a late stage, suggesting low screening rates. This area includes rural and urban counties, and rural counties did not seem more likely than urban to exhibit late stage of diagnosis.

Conclusion: Earlier prostate cancer detection is needed to prevent late-stage diagnosis and improve men's survival. People with less education and health insurance have much lower rates of PSA screening. This health disparity should be better investigated in terms of conduct adopted by the physician and patient beliefs. In Iowa, the southwest quadrant would be a place to start such an investigation.

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P34-6: A Comprehensive and Global Genomic Assessment of p53-regulated Transcriptional Targets

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p53 is a transcription factor and tumor suppressor that mediates responses, including apoptosis and cell cycle arrest, to various cellular stresses. These cellular responses are, in general, attributable to the differential expression of a relatively small number of p53-responsive target genes. However, based on estimated p53 binding site frequency, the number of potential p53-regulated genes numbers several thousand. Our study was designed to characterize the transcriptional response to p53 and to what extent it may be modulated by the presence of BCL-2 in androgen-insensitive prostate cancer cells. Affymetrix U1333A olignucleotide arrays were used to identify genes responsive to adenoviral-mediated p53 gene transfer (Ad-p53) in vector control and BCL-2-overexpressing, p53-null PC3 prostate cancer cells.

Genes transactivated or repressed more than twofold ($p <= 0.05$) in both cell lines were observed. A total of 224 genes exhibited significant upregulation (1.54% of total genes on chip) in response to Ad-p53, and 111 genes were significantly downregulated. p53-responsive, upregulated targets were validated using RT-PCR in PC3 cells. Additionally, targets were validated in p53 wild-type LNCaP prostate cancer cells following DNA damage with etoposide. A validation rate of >95.5% was established for the complete dataset. siRNA knockdown studies confirm that these responses are p53-dependent. The majority of these genes are not currently characterized as p53-responsive. Several genes exhibited differential responses to Ad-p53 BCL-2-expressing cells compared to vector control cells. These differences, however, could not be confirmed in subsequent validation studies. In addition to well-established p53 functions, gene ontology and functional overrepresentation analysis support a role for p53-transactivated target genes in cell adhesion and motility. These findings identify a validated cohort of potentially p53-responsive target genes and support additional p53-regulated cellular functions that may be important in p53-mediated tumor suppression and stress response. Additionally, the ability of BCL-2 to inhibit p53-mediated cell death appears to be largely, if not exclusively, downstream of p53-regulated transcriptional responses.

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P34-7: ProstaScint – the New Wave of Prostate Cancer Detection

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Background: The ProstaScint program has recently emerged as a prostate cancer detection and imaging system. Combining the qualities of a CT (computed tomography) and PET (positron emission tomography) scan, ProstaScint locates tumors and metastases in the prostate and surrounding tissue (seminal vesicles). Marked efficiency of this scanning system can reduce the need for prostate and seminal vesicle biopsies. To date, the efficiency of ProstaScint to these other techniques has not been empirically tested.

Purpose: The purpose of this study is to compare the accuracy of the ProstaScint program to the actual seminal vesicle and prostate biopsies performed by urologic surgeons. It is hypothesized that ProstaScint will more accurately identify the location of cancer tumors and spreading of cancer in comparison to prostate and seminal vesicle biopsies.

Methods: A sample of 69 patients was selected according to the following criteria: (1) they failed radiation treatment and (2) they received a ProstaScint scan before a prostate/seminal vesicle biopsy. After these critical inclusion criteria were met, the population was reduced to 22 patients. Additional observed patient characteristics included PSA (prostate specific antigen) values before biopsies, Gleason scores, MRI (magnetic resonance imaging) results, and biopsy results of the prostate and seminal vesicles. Data were analyzed using crosstabs in SPSS comparing the results (positive or negative) of ProstaScint scans to seminal vesicle biopsies.

Results: The ProstaScint program proves to be an extremely accurate scan. The Negative Predictive Value (N.P.V.) was 92%, indicating that if a patient received a ProstaScint scan and the results were negative, the cancer was not present within the seminal vesicles or prostate 92% of the time. The Positive Predictive Value (P.P.V.) was 50%, indicating that when patients received a positive ProstaScint scan, cancer was present within the seminal vesicles or prostate up to 50% of the time.

Conclusion: In this study, ProstaScint demonstrates high accuracy in detecting the absence of prostate cancer. These findings are particularly beneficial for prostate cancer patients as negative scan results are accurate 92% of the time, indicating that the cancer is gone. However, since the P.P.V. is only 50%, patients who receive negative scan results should have another diagnostic scan, such as an MRI, to check the validity of the ProstaScint scan. Since ProstaScint is a recent technique for tumor imaging, more studies need to be performed to confirm the accuracy of this program. Studies with larger populations need to be conducted to assess the N.P.V. and P.P.V. of the scan.

IMPaCT: This study could potentially have a large impact on prostate cancer mortality through the treatment of metastases. If the ProstaScint program could be perfected, then physicians would be better able to assess the aggressiveness and the location of the cancer, and the patients would spend less time in the hospital and other health care facilities due to the accuracy of the ProstaScint program.

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P34-8: Do RECIST or WHO Criteria Give a More Accurate Assessment of Treatment Response in Solid Tumors?

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Background: Tumor response is often the main objective in clinical trials when evaluating the effects of anti-cancer treatment, including prostate cancer treatment. The two methods that have been used to measure tumor response in clinical trials are World Health Organization (WHO) and Response Evaluation Criteria in Solid Tumors (RECIST).

Purpose: The purpose of this review is to show the advantages and disadvantages of each method and compare objective tumor response to determine which method is more accurate.

Methods: First, a literature review was conducted using PubMed and MEDLINE databases to identify studies that compared WHO and RECIST objective tumor response. Second, interviews were conducted with two physicians at Duke University Medical Center who have experience using RECIST criteria. Third, a clinical trial comparing WHO and RECIST was reviewed to compare the efficacy of the response criteria across various types of cancer.

Results: The literature review revealed that the new RECIST criteria are a simplified version of the WHO criteria; however, RECIST demonstrates concordance with modern technological advances such as magnetic resonance imaging (MRI) and computed tomography (CT) scans. The interviews that were conducted suggest that in reference to prostate cancer, neither WHO nor RECIST is beneficial because neither criteria can measure bone metastases or prostate specific antigen (PSA) values. WHO and RECIST are radiographic measures, while PSA is a biochemical measurement. These two components are very important in determining the progression of prostate cancer. Finally, the clinical trial suggests that both WHO and RECIST criteria are comparable.

Conclusion: After reviewing the evidence, it was found that WHO and RECIST criteria are comparable in assessing tumor response in solid tumors. However, RECIST criteria are not as useful in assessing treatment response in prostate cancer because other factors such as bone metastasis and PSA need to be considered, and perhaps a new method of measuring tumor response for bone metastases should be explored.

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P34-9: The Roles of RhoG, Rac1, and Rac3 GTPase in PC-3 Human Prostate Cancer Tumor Cells

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Based on previous research, the downregulation of the RhoC GTPase in PC-3 human prostate cancer cells derived from bone metastasis leads to increased and sustained levels of Rac GTPase activity. It has been shown that the Rac GTPases are involved in prostate cancer cell migration and invasion, particularly through bone marrow endothelial cells. In the current study, we

examine the levels of expression, activation, and phenotypic effects of Rac1, Rac3, and RhoG GTPases. The relative and quantitative levels of Rac1, Rac3, and RhoG were compared in PC-3 cells and C3 exotransferase-treated PC-3 cells. In the future, it will be compared to siRNA-treated cells. A tumor cell diapedesis assay will be done across a monolayer of bone marrow endothelial cells after siRNA treatment of Rac1, Rac3, or RhoG to determine the individual contributions of each GTPase to a cell's invasive capability. We will determine the phenotypic and physiological effects of Rac1, Rac3, and RhoG more closely. We plan to calculate changes in morphology, cell deformation, and binding strength using atomic force microscopy.

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P34-10: Establishing Dose-Response Curves for Chemotherapeutics in Prostate Cancer: Baseline Data for Synergistic Drug Interactions

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Cancer cells have the extraordinary ability to alter their phenotypes and mutate their genotype to attain selective advantage and produce one cell that will survive and colonize at the metastatic site. This is a major cause of treatment failure creating a tremendous hurdle to overcome in designing novel cancer therapeutics. One alternate approach is to examine the interactions between drugs to determine if a previously undiscovered synergy exists. This would enhance tumor cell kill and increase the tolerance of the therapy in the patient by reducing the therapeutic dosage of both drugs. These drugs are chemosensitizers or cooperative chemotherapeutics. Adding drugs at the IC20s helps determine whether synergy with traditional chemotherapy occurs.

This research involved obtaining inhibitory concentration curves for different drugs to determine the IC50, IC20, and IC10. Five drugs commonly used to treat prostate cancer (Docetaxel, Vinblastine, Cisplatine, Verapamil, and Etoposide) were used in this research and two different assays (MTT and crystal violet) were applied to determine cell viability. LNCaP cells were plated at 360,000 cells per 48 well plate followed by treatment with respective drugs for 5 or 7 days, and the results were analyzed. To date, we have obtained good dose-response curves for Docetaxel that will allow us to calculate the IC20 and begin synergy experiments with sodium channel blockers.

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P34-11: Investigating the Effects of Disease and Stress on Cytokine and Hormone Expression in the Brain

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Short Description: A common problem in the African American (AA) community is disease: cancer, diabetes, asthma, etc. Corresponding with higher rates of disease, statistics on poverty, low-income housing, and unemployment demonstrates that AAs are exposed to more stressful conditions than any other racial group in the United States. There is increased evidence linking neuroendocrine and immune systems through the regulation of stress response factors and cytokines, respectively. Thus, we believe that investigation of the role that stress has on the biological mechanisms involved in mediating disease states will provide innovative approaches in the treatment and prevention of chronic disease and improve health disparities among AAs.

Purpose: Using an experimental model of stress and antigenic activation of immune responses along the respiratory tract, we examined the influence that stress had on brain-associated cytokine gene expression.

Methods: Mice were subjected to controllable, uncontrollable, and non-stressed environments and exposed to an experimental antigen (Ovalbumin in aluminum hydroxide) by intranasal administration. Total RNA was extracted from the whole brain tissue, and cytokine gene mRNA levels (IL-1β, TNF-α, IL-6, IL-10, TFG-β, CRH, CRHR1, and CRHR2) were determined using quantitative real-time RT-PCR (qRT-PCR) techniques. Similarly, total RNA was extracted from hypothalamic and cortex regions of the brain using laser capture microdissection (LCM) followed by qRT-PCR.

Results: Our results demonstrate differences in the quality and localization of cytokine and CRH/CRH receptor expression under conditions of stress.

Conclusions: The results from these studies describe the effect of controllability of stress on cytokine gene expression in the brain and provide a tool to investigate the use of LCM to demonstrate localization of cytokine and CRH/receptor expression.

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P34-12: Isolation of Apo A-I, a Component of the rHDL Drug Delivery System, a Novel Approach for Cancer Chemotherapy

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Short Description: Chemotherapy continues to be a major treatment option for most cancerous tumors. Despite recent successes in single or combination therapy, solubility and toxic side effects remain a serious concern during the intravenous delivery of anti-cancer drugs.

Purpose: This project aims to develop a novel, targeted delivery vehicle with increased efficiency aiming to reduce the toxic side effects of anti-cancer drugs by using reconstituted high-density lipoproteins (HDLs). Cancer cells are likely to have an enhanced expression of lipoprotein receptors due to their high proliferative rate and thus the need for excess cholesterol.

Methods: Column chromatography was used to extract the HDL from human plasma instead of the traditional Rudel procedure, thus permitting a greater plasma load and consequently a better percentage recovery of HDL from the plasma. Large scales such as 1000 mL of human plasma were loaded onto a butyl sepharose column, and approximately 80% of pure plasma HDL was recovered. Further analyses were performed in order to compute its characteristics. The HDL was then delipidated and ApoA1 was isolated by gel chromatography.

Results: Our results indicated that this new method of extracting HDL from human plasma was rapid, inexpensive, and industrially scalable. It allowed us to have a high percentage of recovery of HDL from the plasma used.

Conclusion: The results from the project allowed us to prepare reconstituted HDL particles from pure HDL extracted from human plasma. The results also helped show that the general characteristics of native HDL were the same as those of our reconstituted HDL. In addition, using the ApoA1 isolated from the new procedure, rHDL/drug nanoparticles can be prepared.

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P34-13: Effect of PKCη Phosphorylation on Its Stability

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Purpose: Protein kinase C (PKC), comprised of a large family of serine/threonine kinases, is activated by extracellular signals. PKCs are categorized into three subclasses: conventional, novel, and atypical. PKCη is a member of novel PKCs. It is often elevated in several cancers. In addition, overexpression of PKCη was associated with resistance to anticancer drugs. Thus, an understanding of how PKCη is regulated is important to target this kinase for cancer therapy. PKCs are activated by phorbol esters, but prolonged treatment with phorbol esters causes their degradation or downregulation. The regulation of PKCη is unique. It is upregulated by PKC activators and downregulated by PKC inhibitors. We hypothesize that phosphorylation of PKCη regulates its stability. Our objective was to mutate potential phosphorylation sites in PKCη and determine its effect on PKCη stability.

Methods: Since PKCη is phosphorylated at Threonine 513, Threonine 655, and Serine 674 sites, we mutated these phosphorylation sites to non-phosphorylatable Alanine (AAA) or phospho-mimicking glutamate (EEE). Wild-type (WT) and mutant PKCη were transfected into MCF-7 cells and treated with an activator, PDBu (phorbol 12-13 dibutyrate) or an inhibitor, Gö 6983. First, we compared the RNA levels using RT-PCR. Then we looked at the protein by using SDS-PAGE. Since phosphorylation of PKC may affect its antibody recognition, we tagged the proteins with GFP (green-fluorescent protein).

Results: Mutation of PKCη did not change mRNA levels. WT-PKCη protein was upregulated when treated with the activator and downregulated when treated with the inhibitor. WT-PKCη-GFP and phospho-mimicking mutant (EEE-PKCη-GFP) were expressed in cells, but the phospho-defective mutant (AAA-PKCη-GFP) was not expressed.

Conclusion: The stability of PKCη is regulated by phosphorylation. (Supported by the grants W81XWH-06-1-0284 from the Department of Defense Prostate Cancer Research Program and CA71727-07S1 from the National Cancer Institute.)

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P34-14: Role of Shp-1 in Enhancing Dendritic Cell-based Anti-Tumor Vaccines for Prostate Cancer

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One of the primary objectives in Dr. Levitt's lab is the development of dendritic cell-based, anti-tumor vaccines, specifically against tumors of the prostate. Much of my work this summer involved investigating the roles in dendritic cells (DCs) of SHP-1 (src homology region 2 domain-containing phosphatase-1), a specific inhibitory molecule active in many hematopoietic cells.

To investigate the roles of SHP-1 in DCs, I used RNA interference to knock down its expression in these cells. This was achieved by treating bone marrow-derived DCs with adenovirus containing anti-SHP-1 shRNA. Using western blot in SHP-1 knockdown DCs, I observed that Akt, a serine/threonine kinase involved in promoting cell survival and inhibiting apoptosis, activation was enhanced. Using staining with Annexin V/PI (both of which measure relative states of apoptosis) in flow cytometry, increased survival in SHP-1-inhibited DCs was observed.

A substantive portion of the summer was spent in a DC-based anti-tumor vaccine study in which both the role of SHP-1 inhibition and the immunogenicity of different peptides against TRAMP tumors (a prostate cancer tumor cell line) had on the efficacy of our vaccine. To make vaccines, we extracted bone marrow from C57BL/6 mice, and then we cultured DCs from the bone marrow. We subsequently treated these DCs with adenovirus containing anti-SHP-1 shRNA to knock down SHP-1 expression. We had five groups of vaccines, each of which was loaded with a different peptide against prostate specific antigen, to determine which would be most effective against TRAMP tumors. Another population of C57BL/6 mice was then inoculated with TRAMP tumor cells. Three days after tumor inoculation, the mice were vaccinated, and observations on tumor growth and mice survival were made every 3 days.

While we have shown that inhibition of SHP-1 both increases Akt activation and DC survival, its role in increasing the efficacy of DC-based vaccines against TRAMP tumors is still ongoing.

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P34-15: Enhancing 1,25 Dihydroxyvitamin D3 Action in Prostate Cancer Cells

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1,25 dihydroxyvitamin D3 (1,25D), the active hormone of vitamin D, inhibits growth of prostate cancer cells including the LNCaP prostate cancer cell line. However, although PC346C prostate cancer cells have functional vitamin D receptor (VDR), our preliminary results suggested that they were not growth inhibited by 1,25D. Our first hypothesis as to why PC346C cells are not responsive to 1,25D is that the strong induction of 24OHase limits the activity of 1,25D. 24OHase is a vitamin D receptor target gene that hydroxylates and inactivates the vitamin D receptor (VDR) ligand, 1,25D. Because of the intracellular metabolism of 1,25D, prostate cancer cell growth may not be inhibited in that cell line. This study focused on whether the inhibition of 24OHase expression or activity would cause PC346C cells to be more responsive to 1,25D. There is one report that dihydrotestosterone (DHT) inhibits 1,25D-induced expression of 24OHase in LNCaP cells. There is also evidence that genistein inhibits 24OHase enzymatic activity. Therefore, we treated LNCaP and PC346C cells with EtOH, as a control, 1,25D, DHT (dihydrotestosterone), or a combination of 1,25D and DHT and measured cell number using a Coulter counter. The data show that the combination of 1,25D and DHT did not cause growth inhibition in the PC346C cell line. However, 24OHase levels were not reduced with the combination treatment in the PC346C cells. Although genistein inhibited cell growth, the combination of 1,25D and genistein did not perform any better than genistein alone in the PC346C cell line. Genistein is also a tyrosine kinase inhibitor, so the effects of genistein may be through inhibition of kinase. In the LNCaP cell line, the combination treatment of DHT and 1,25D was more effective in inhibiting growth than either alone, but it did not reduce 24OHase levels. The second hypothesis tested was that 1,25D action was limited because Erk-dependent phosphorylation of RXR alpha prevents dimerization with VDR, reducing VDR activity. To test this, we treated cells with 1,25D, U0126, or a combination of the two. UO126 inhibits MEK, which is part of the kinase cascade of Raf, MEK, and Erk, and inhibition of MEK prevents activation of Erk. U0126 was successful in reducing cell growth in both cell lines, with a greater effect on the PC346C cells, but reduction was not enhanced with 1,25D. In summary, neither DHT, genistein, nor U0126 appeared to enhance the PC346C response to 1,25D. Thus, another factor other than 24OHase expression or activated Mek could cause PC346C cells to be more resistant to 1,25D.

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P34-16: Recruitment of Reactive Stroma in Prostate Cancer Progression

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The lab of Dr. David Rowley studies human prostate cancer reactive stroma, whereby the stromal compartment of prostate tissues undergoes changes in cell architecture and gene expression as a repair response. The reactive stroma microenvironment is important to prostate cancer; however, specific mechanisms of its role in regulating prostate cancer progression are poorly understood. Among the most important of these reactive cells is the myofibroblast, the appearance of which is directly correlated with prostate cancer severity. This highly synthetic cell with a contractile phenotype expresses vimentin and smooth-muscle alpha actin, two key biomarkers that help distinguish reactive stroma from normal tissues. This unique cell type is not observed in normal prostate and is believed to co-evolve with the tumor microenvironment in an effort to re-establish tissue homeostasis. Recent studies suggest that myofibroblasts at sites of reactive stroma might originate from circulating "fibrocytes" linked to the hematopoietic lineage. Mouse lungs treated with allergen resulted in recruitment of circulating CD34+ progenitors to bronchial tissue where they subsequently differentiated to myofibroblasts. This study also showed allergic asthma patients had fibrocytes in bronchial mucosa that were positive for CD34, collagen I, and smooth muscle alpha actin, suggesting that progenitors were from circulating bone marrow derived cells.

It is our hypothesis that human prostate cancer reactive stroma is composed of myofibroblasts recruited from circulating hematopoetic progenitors. Many years ago, our lab developed a differential reactive stroma (DRS) xenograft model system as a novel way to study human prostate cancer in a mouse. Interestingly, there seemed to be a recruitment of mouse host cells to

sites of tumor whenever human LNCaP prostate cancers cells were employed in the xenograft. As a modification of the DRS approach, we developed a "matrix trapping" procedure whereby we combined conditioned media from LNCaP cells with Matrigel to more effectively study host cell recruitment to the xenograft implants. We used immunohistochemistry to more effectively assay for the expression of tenascin, collagen I, smooth muscle alpha actin, and CD34. Preliminary data from the work this summer showed that the matrix traps stained negative for smooth muscle alpha actin and positive for tenascin, pro collagen type 1, and CD34. These results suggest that host cells are synthesizing collagen type 1 and tenascin, and they are not yet committed to a myofibroblast lineage as evidenced by their lack of smooth muscle alpha actin expression. Furthermore, positive CD34 staining suggests that a subpopulation of reactive stroma originates from circulating bone marrow progenitor cells.

This particular area of prostate cancer research is understudied, yet it may be an important focus area for novel therapeutic intervention early on in prostate cancer. Future directions include bone marrow transplantation studies, validation of our CD34 findings in the matrix traps, and in vitro analysis of mechanisms of host cell recruitment. If our hypothesis is correct, then a circulating progenitor cell could be a putative target for novel therapeutics.

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