SBUR 2017 FALL SYMPOSIUM

November 9-12, 2017 • Tampa, FL

The Westshore Grand

MEETING PROGRAM

SBUR

SOCIETY FOR BASIC UROLOGIC RESEARCH, INC.

THE SOCIETY FOR BASIC UROLOGIC RESEARCH THANKS THE FOLLOWING COMPANY FOR BEING A SILVER SUPPORTER FOR THE 2017 FALL SYMPOSIUM.

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*Thank you to the National Institutes of Health for the grant award in support of this meeting. Research reported at this meeting was supported by the NIDDK of the NIH under award number 1R3DK112664-01. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

As of 10/31/17

Welcome Colleagues and Friends!

Welcome to the Society for Basic Urologic Research Fall 2017 meeting. It's great to see all of you here in beautiful Tampa.

The inheritance and transmission of information required to maintain a defined biological state is fundamental to defining how cells and tissues behave and how they misbehave in disease. Therefore, the focus of our annual meeting this year is on the genetic and epigenetic mechanisms that underlie both normal and urologic disease states. We will investigate both classical genetic and epigenetic mechanisms, explore some new ideas about inheritance, and figure out some strategies for overcoming pathological effects of 'bad' inheritance.

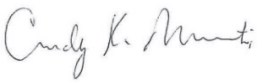
The meeting will commence with the "Trainee Affairs Career Symposium" led by Dr. Shawn Lupold followed by five Plenary Sessions covering the following areas: 1) Epigenetic Modifiers 2) Genetic Modifiers 3) Alternative Regulatory Mechanisms 4) Model Systems in Urology 5) Epigenetic and Genetic Reprogramming.

New technologies are critical for advancing research and medicine. However, it is also important to critically evaluate their positive and negative attributes. Several of our speakers will discuss the application of new technologies to Urology, including quantitative imaging, proteomic profiling, and the development of new models. There will also be a discussion panel on the pros and cons of isolating and using CTCs and secretory vesicles in research and medicine entitled "Cellular Debris: Is It Really Useful?" Thus, these will be great opportunities for dynamic discussion and reflection.

I would like to thank the SBUR 2017 Fall Meeting Program Committee members for their invaluable assistance in putting this meeting together and to the NIH NCI for supporting our trainees with travel awards. A special thanks to Dr. Zongbing You for chairing this year's Abstracts Review committee.

Finally, on behalf of the Program Committee, we thank you for joining us in Tampa and hope that the presentations and discussions at this dynamic meeting will translate into exciting research opportunities and collaborations back home.

Hari K. Koul, MSC, PhD, FASN
President



Cindy Miranti, PhD
2017 Scientific Program Chair

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General Information

REGISTRATION HOURS

Grand Foyer, Lobby Level

Thursday, November 9 . . . . . 2:00– 8:30 p.m.

Friday, November 10 . . . . . 7:00 a.m. – 6:00 p.m.

Saturday, November 11 . . . . . 7:00 a.m. – 6:00 p.m.

Sunday, November 12 . . . . . 7:00 a.m. – 11:00 a.m.

GENERAL SESSION

Bayshore Ballroom

Thursday, November 9 . . . . . 7:00 – 8:30 p.m.

Friday, November 10 . . . . . 8:00 a.m. – 5:00 p.m.

Saturday, November 11 . . . . . 8:00 a.m. – 5:00 p.m.

Sunday, November 12 . . . . . 8:00– 11:30 a.m.

TRAINEE AFFAIRS CAREER SYMPOSIUM

Bayshore Ballroom

Thursday, November 9 . . . . . 3:00– 5:00 p.m.

LUNCH

The Atrium

Friday, November 10 . . . . . 12:00 – 1:30 p.m.

Saturday, November 11 . . . . . 12:15 – 1:15 p.m.

Evening Events

Join old and new friends and colleagues at these evening events.

Grand Foyer
Welcome Reception

Thursday, November 9
8:30 – 10:00 p.m.
Business Casual

Grand Foyer
Poster Session I

Friday, November 10
5:00– 7:00 p.m.
Business Casual

Meet at Registration
Trainee Affairs Group Dinner

Friday, November 10
7:00 p.m.
Business Casual

Grand Foyer
Poster Session II

Saturday, November 11,
5:10– 7:00 p.m.,
Business Casual

*At attendee's expense. See registration desk to RSVP.

Educational Needs and Objectives

STATEMENT OF NEED

The development of epigenetic screens in combination with genetics represents a new frontier in personalized medicine that is required for effective decision making with respect to when and how to screen, treat, and counsel patients. However, many challenges remain before this concept can be fully implemented. Understanding the myriad of mechanisms associated with epigenetics, connecting the underlying genetic landscapes with epigenetic states, defining the context in which they occur in both normal and disease tissue, and determining whether epigenetics can provide opportunities for therapeutic intervention all still need to be resolved. A further requirement is the integration of multiple fields including medicine, basic and clinical medical research, genetics and genomics, biotechnology, data analysis and disease modeling. Decision making in personalized medicine is continually changing and advancing, and a concerted educational effort must be made to enable researchers and urologists to keep up with these changes. To keep up, researchers need (1) education on the components underlying genomic medicine and how these could be integrated to develop more effective, evidence-based approaches to patient care, (2) education regarding the different levels of complexity in a patient that would need to be considered in guiding precision medicine, including proteins, cell types, organs, and even organisms that live within the urologic tract and could influence treatment outcomes, and (3) education regarding the requirement of an multidisciplinary team approach across a wide range of disciplines to advance personalized medicine.

LEARNING OBJECTIVES

At the conclusion of this conference, the attendees should be able to:

a. Highlight recent advances in genetic and epigenetic data in normal and urologic disease states.

b. Discuss the pros and cons of new technology to measure genetic and epigenetic changes.

c. Describe how the combined use of genetic and epigenetic data can affect patient care.

d. Identify collaborators across multiple disciplines to apply genetic and epigenetic data in patient care.

CONTINUING MEDICAL EDUCATION

Accreditation: The American Urological Association (AUA) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

Credit Designation: The American Urological Association designates this activity for a maximum of 20.5 *AMA PRA Category 1 Credits™*. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Other Learners: The AUA is not accredited to offer credit to participants who are not MDs or DOs. However, the AUA will issue documentation of participation that states that the activity was certified for *AMA PRA Category 1 Credit™*.

Evidence Based Content: It is the policy of the AUA to ensure that the content contained in this CME activity is valid, fair, balanced, scientifically rigorous, and free of commercial bias.

AUA Disclosure Policy: All persons in a position to control the content of an educational activity (i.e., activity planners, presenters, authors) are required to disclose to the provider any relevant financial

relationships with any commercial interest. The AUA must determine if the individual's relationships may influence the educational content and resolve any conflicts of interest prior to the commencement of the educational activity. The intent of this disclosure is not to prevent individuals with relevant financial relationships from participating, but rather to provide learners information with which they can make their own judgments.

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- Peer review for valid, evidence-based content of all materials associated with an educational activity by the course/program director, editor, and/or Education Content Review Committee or its subgroup.
- Limit content to evidence with no recommendations
- Introduction of a debate format with an unbiased moderator (point-counterpoint)
- Inclusion of moderated panel discussion
- Publication of a parallel or rebuttal article for an article that is felt to be biased
- Limit equipment representatives to providing logistics and operation support only in procedural demonstrations
- Divestiture of the relationship by faculty

Off-label or Unapproved Use of Drugs or Devices: Please consult the prescribing information for full disclosure of approved uses.

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Special Assistance/Dietary Needs: The AUA complies with the Americans with Disabilities Act §12112(a). If any participant is in need of special assistance or has any dietary restrictions, please see the registration desk.

Disclosures

Disclosures of individuals who are in control of content for the SBUR 2017 Fall Symposium (faculty, planners, authors, etc.) can be viewed at www.sbur.org.

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Congratulations to the 2017 SBUR Award Winners

DISTINGUISHED SERVICE AWARD

Presented annually at the Spring Meeting, this award recognizes an SBUR member who has helped the SBUR with his/her services and/or influences.

Zhou Wang, PhD

MERITORIOUS ACHIEVEMENT AWARD

Presented annually at the Fall Meeting, this award recognizes a researcher (can be a clinician researcher) who has made excellent contributions in the field of urological research.

Ralph W. de Vere White, MD

SWIU/SBUR AWARD FOR EXCELLENCE IN UROLOGIC RESEARCH

SWIU and SBUR have a common interest in recognizing female scientists with an accomplished background of basic science urological research. The award (presented at the Spring Meeting) represents the collaborative efforts of these two societies toward their common goals.

Susan Kasper, PhD

YOUNG INVESTIGATOR AWARD RECIPIENTS

The SBUR Young Investigator Awards are presented annually at the Fall Meeting. These awards are given to SBUR members under the age of 45, within 5 years of their first faculty position, who have made significant contributions to urologic research.

Justin M. Drake, PhD
Rutgers Cancer Institute of New Jersey

Mehdi Mollapour, PhD
Upstate Medical University

Mohummad Minhaj Siddiqui, MD
University of Maryland School of Medicine

YOUNG INVESTIGATOR AWARD NOMINEES

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Michael C. Haffner, MD, PhD
The Johns Hopkins University School of Medicine

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Memorial Sloan Kettering Cancer Center

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Wake Forest School of Medicine

Takashi Kobayashi, MD, PhD
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Hung-Ming Lam, PhD
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Harvard Medical School

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Yale School of Medicine

Douglas W. Strand, PhD
UT Southwestern Medical Center

Huimin Sun, PhD
Xijing Hospital, China

Yi Yin, MD, PhD
University of Texas Southwestern Medical Center

TRAVEL AWARDS

A primary goal of SBUR is to provide travel grants/stipends to researcher trainees. These grants support travel to/from the Fall Symposium. Award recipients must be SBUR members and a recipient is not allowed to receive the award in two consecutive years.

Aymen Alqazzaz, BS
University of Maryland School of Medicine

Jason Van Batavia, MD
The Children's Hospital of Philadelphia, University of Pennsylvania

Bethany Baumann, PhD
University of Illinois

Zachary Connelly, BS
LSU Health Sciences Center, Shreveport

Metsiel Donate, MA
School of Pharmacy of University of Puerto Rico and Comprehensive Cancer Center UPR

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The Hormel Institute University of Minnesota

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Van Andel Research Institute

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Moffitt Cancer Center

Jeremy McGuire, BS
Moffitt Cancer Center

Krizia Rohena Rivera, PhD
Cedars-Sinai Medical Center

Kenny Roman, PhD
Northwestern University

Mirja Rotinen, PhD
Cedars-Sinai Medical Center, Michael Freeman Lab

Catherine Seager, MD
Boston Children's Hospital

Gauri Shishodia, PhD
LSU Health Sciences Center

Samuel Thomas, BS
University of Wisconsin, Madison

Zongwei Wang, PhD
Massachusetts General Hospital, Harvard Medical School

Yadong Xu, BS
UPMC Shadyside

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Named Lecturer Bios

AUA LECTURER

DAVID JARRARD, MD

David Frazier Jarrard, MD, is a tenured Professor of Urology and Molecular and Environmental Toxicology at the University of Wisconsin. He obtained his BA and MD from the University of Virginia and completed a Urologic Surgery residency at the University of Chicago Hospitals. He was a fellow in Urologic Oncology at the Brady Urologic Institute of the Johns Hopkins Hospital. He is currently Vice Chair for the Department of Urology, Associate Director at the UW Carbone Cancer Center, leads the Section of Urologic Oncology, and holds the John Livesey Chair in Urologic Oncology. Dr. Jarrard has served on numerous American Urological Association committees and is President-Elect for the North Central Section. He is Past President for the Society for Basic Urologic Research (SBUR) and serves on the boards of the Society for Urologic Oncology (Member at large) and Society for Academic Urology (Treasurer).

He has active basic science and translational research interests supported through the National Institutes of Health and the Department of Defense Prostate Cancer Research Programs examining epigenetic factors underlying prostate cancer development and novel approaches to treating advanced disease. His clinical work concentrates on advanced Urologic Oncology and improving cancer detection and outcomes. Awards include the SBUR Young Investigator and Distinguished Service Awards, Madison Magazine's 'Best Physicians in Wisconsin', Castle Connolly 'Top Doctors', 'Best Doctor in America™' and the Society for Urologic Oncology Distinguished Service Award. Dr. Jarrard has published numerous chapters, articles and abstracts in the field of prostate cancer.

LELAND W.K. CHUNG LECTURE

DARIO C. ALTIERI, MD

Dario C. Altieri, M.D., is an academic leader, scientist, and advocate who has made impactful contributions to biomedical research, filing nine patents and authoring more than 220 scientific articles. The Altieri Lab studies how cancer cells reprogram mechanisms of mitochondrial metabolism, cell motility, and apoptosis to acquire more aggressive traits of disease progression and metastatic competence. Most of his current work focuses on advanced, castration-resistant and metastatic prostate cancer and aims at identifying actionable therapeutic targets in these patients.

Born in Milan, Italy, and educated at the University of Milan School of Medicine, Dr. Altieri became a practicing clinician at the same University, where he would later earn a post-graduate specialty degree in clinical and experimental hematology. In 1987, he joined the Scripps Clinic and Research Foundation in La Jolla, California, first as a research fellow and later as a member of the faculty. In 1994, Dr. Altieri became an associate professor at the Yale University School of Medicine, where he was named professor in 1999, and served in that role until 2002 when he was recruited as the founding chair of the Department of Cancer Biology at the University of Massachusetts Medical School. Dr. Altieri became the eighth president and CEO of The Wistar Institute in March of 2015, and he is also Wistar's Cancer Center Director, a position he has served since 2010.

Dr. Altieri's dedication to turn biomedical discoveries into future cancer therapies fuels his strong belief in advocacy and mentorship. A champion for cancer patients, he cofounded

the Pancreatic Cancer Alliance, a patient advocacy group devoted to helping people and families cope with pancreatic cancer. He's also a steadfast supporter of the next generation of young scientists and was the cofounder of the Cancer Biology Training Consortium, a national organization that promotes scientific excellence in young cancer researchers.

Scientific Program

Travel Award

Multiple Genetic and Epigenetic Mechanisms of Urologic Disease

2017 PROGRAM COMMITTEE

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THURSDAY, NOVEMBER 9, 2017

2:00 - 8:30 p.m. **Registration**

Grand Foyer, Lobby Level

3:00 - 5:00 p.m. **Trainee Affairs Career Symosium**

Shawn Lupold, PhD
Trainee Affairs Committee Chair

7:00 - 7:15 p.m. **Welcome & Introductory Remarks**

Hari K. Koul, MSC, PhD, FASN
SBUR President
LSU Health Sciences Center
Shreveport, LA

7:15 - 7:20 p.m. **Opening Session**

Introduction:

Hari K. Koul, MSC, PhD, FASN
SBUR President
LSU Health Sciences Center
Shreveport, LA

Discussion Leaders:

Cindy Miranti, PhD
University of Arizona Cancer Center
Tucson, AZ

Marc Cox, PhD
University of Texas at El Paxo
El Paso, TX

7:20 – 8:20 p.m. **AUA Lecture**

Epigenetics and Therapy of Urologic Cancers

David Jarrard, MD
University of Wisconsin
Madison, WI

An important question in prostate cancer biology is what underlies the remarkable incidence prostate cancer susceptibility with aging. The epigenome is susceptible to modulation by many factors associated with aging, including dietary and oxidative stress. We have found that the peripheral zone of the prostate from men with prostate cancer, where prostate tends to arise, commonly contains biallelic Insulin-like growth factor (Igf2) expression (Clin Can Res 1996) a potent growth factor, in contrast to other organs where the gene is only expressed from one allele and thus demonstrates what is termed genomic imprinting. Importantly, an age-related degradation of imprinting occurs in the murine and human prostate and underlies the development of prostate cancer (JBC 2007: Can Res 2009, Can Res 2017). Loss of CTCF binding protein during aging mechanistically causes the relaxation in Igf2 imprinting seen during aging (Prostate 2011; Plos One 2014) and furthermore targets specific regions of the genome for hypermethylation and inactivation. Thus, a degradation of the epigenome leads to a field of cancer susceptibility in the prostate serving as a marker for the disease, as well as a potential avenue for preventative therapy and treatment.

8:20 – 8:30 p.m. **Discussion**

8:30 – 10:00 p.m. **Welcome Reception and Networking**

FRIDAY, NOVEMBER 10, 2017

Breakfast on Own

6:30 - 8:00 a.m. **Publications Committee Meeting**

8:00 a.m. - 12:00 p.m. **Plenary Session I: Epigenetic Modifiers**

Discussion Leaders:

Scott Dehm, PhD
University of Minnesota
Minneapolis, MN

Christina Jamieson
University of California, San Diego
San Diego, CA

8:00 - 8:25 a.m. **Epigenetics of BPH**

Aria Olumi, MD
Harvard Medical School
Boston, MA

We have found that 30% of human adult prostate glands do not express 5-alpha reductase 2, the gene responsible for development and growth of the prostate, and the target enzyme for anti-androgen treatment of BPH. 5-AR2 is silenced through epigenetic mechanisms involving inflammatory mediators. Coupled with absence of 5-AR2, we have found an "androgenic to estrogenic switch" in the prostate gland, suggesting that alternate pathways, beside anti-androgenic targets, may be utilized for management of prostatic diseases.

8:25 - 8:35 a.m. **Discussion**

8:35 - 9:00 a.m. **Chromatin Regulation in Prostate Cancer**

Haojie Huang, PhD
Mayo Clinic
Rochester, MN

The Polycomb group (PcG) protein EZH2 is an only enzymatic subunit of the Polycomb Repressive complex 2 (PRC2) that is primarily known for its function as a methyltransferase responsible for histone H3 lysine 27 trimethylation and gene silencing, a Polycomb-dependent function. Increasing evidence indicates there is a Polycom-independent and yet methyltransferase activity-dependent function of EZH2 that promotes oncogenesis via gene activation. In the current presentation a previously uncharacterized non-methyltransferase function of EZH2 will be discussed.

9:00 - 9:10 a.m. **Discussion**

9:10 - 9:35 a.m. **Chromatin Modifiers in Non-Invasive Bladder Cancer**

Margaret Knowles, PhD
Leeds Institute
Leeds, UK

Recent studies have identified frequent mutations in chromatin modifier genes in bladder cancer and this is well documented for muscle-invasive tumors. As the number of non-invasive samples studied increases, it is becoming clear that the frequency of mutations in some of these genes is higher still. Ultimately this may suggest novel approaches to localized therapy for this large group of patients.

9:35 – 9:45 a.m. **Discussion**

9:45- 10:00 a.m. **Break**

10:00 - 10:25 a.m. **Networks That Control BPH Development**

Magdalena Grabowska, PhD
Case Western Reserve University

Cleveland, OH

Benign prostatic hyperplasia (BPH) is a benign overgrowth of the epithelium and stroma of the prostate. Using cell lines, transgenic mice, and human patient samples, we have correlated loss of nuclear factor I/B (NFIB) with development of prostatic hyperplasia through AR-dependent and AR-independent mechanisms. We are now exploring how these NFIB-regulated networks contribute to therapy resistance in BPH and prostate cancer.

10:25 – 10:35 a.m. Discussion

10:35 – 10:50 a.m. Travel Award Winner Presentation #1

Androgenic to Estrogenic Switch in Prostate Gland as a Result of Epigenetic Silencing of Steroid 5-α Reductase 2

Zongwei Wang, PhD
Massachusetts General Hospital, Harvard Medical School
Boston, MA

10:50 – 10:55 a.m. Discussion

10:55 - 11:20 a.m. New Proteomic Approach for Profiling Urine

William Ricke, PhD
University of Wisconsin
Madison, WI

Biomarkers for disease states in addition to treatment success and/or response are important clinical tools that ultimately improve patient outcomes. Development of new mass spectrometry (MS) methods and new uses for current MS technologies will assist in identifying patients with particular diseases as well as the stratification of patients for use of specific therapeutic regiments. We will discuss current and advanced MS technologies and their application to urologic specimens with the goal of identifying new biomarkers.

11:20 – 11:30 a.m. Discussion

11:30 – 11:45 a.m. Travel Award Winner Presentation #2

Loss of Epigenetic Regulation Results in Congenital Malformations of the Urogenital System

Catherine Seager, MD
Boston Children's Hospital
Boston, MA

11:45 – 11:55 a.m. Discussion

12:00 - 1:30 p.m. Lunch

1:30 – 5:00 p.m. Plenary Session II: Genetic Modifiers

Discussion Leaders:

Beatrice Knudsen, MD, PhD
Cedars-Sinai Medical Center
Los Angeles, CA

Xue Sean Li, PhD
Boston Children's Hospital, Harvard Medical School
Boston, MA

1:30 - 1:55 p.m. **Prostate Cancer Genetics and Disparities**

Michael Ittmann, MD, PhD
Baylor College of Medicine
Houston, TX

African American men have a higher incidence and significantly higher mortality rate from prostate cancer than European American men. We have carried out the largest existing combined study of gene expression and copy number alterations in African American prostate cancer to elucidate novel mechanisms of prostate carcinogenesis in African American men. We have identified RGS12 as a tumor suppressor whose loss results in activation of multiple important pathways linked to oncogenic transformation and therapy resistance. Of note, MNX1, an oncogene that we have identified as being markedly increased in African American prostate cancer, is increased by RGS12 loss and this results in activation of multiple oncogenic pathways. The proteins and associated downstream pathways we have identified may be important biomarkers of disease aggressiveness in African American prostate cancer as well as novel therapeutic targets.

1:55 - 2:05 p.m. **Discussion**

2:05 - 2:30 p.m. **Bladder Cancer Genetics**

Dan Theodorescu, MD, PhD
University of Colorado, Denver
Denver, CO

Innovative molecular tools and approaches used in discovery and validation of driver genes in cancer and therapeutics targeted at these for use in patient treatment.

2:30 - 2:40 p.m. **Discussion**

2:40 – 2:50 p.m. **Awards Presentations**

2017 Distinguished Service Award Presentation

Zhou Wang, PhD
University of Pittsburgh Medical Center
Pittsburgh, PA

2017 Meritorious Achievement Award Presentation

Ralph W. de Vere White, MD
University of California, Davis School of Medicine
Sacramento, CA

2017 Young Investigator Awards Presentation

Justin M. Drake, PhD
Rutgers Cancer Institute of New Jersey
New Brunswick, NJ

Mehdi Mollapour, PhD
Upstate Medical University
Syracuse, NY

Mohummad Minhaj Siddiqui, MD
University of Maryland School of Medicine
Baltimore, MD

2:50 – 3:05 p.m. **Break**

3:05 – 3:30 p.m. **SBUR Annual Business Meeting – MEMBERS ONLY**

Hari K. Koul, MSC, PhD, FASN
President

3:30 - 3:55 p.m. **Therapy-induced Drug Reprogramming of Prostate Cancer**

Ralph Buttyan
University of British Columbia
Vancouver, Canada

Survival of metastatic prostate cancer is limited by the development of resistance to any/all therapeutics used to treat the disease. While mutations play a significant role in androgen-receptor-dependent resistance, less is known about resistance driven by changes in cancer cell state as is exemplified in neuroendocrine prostate cancer. We explored the idea that this form of disease is associated with a “developmental reprogramming” involving a neural stem cell-like intermediate. Our cell culture models now show that we can readily convert androgen-responsive prostate cancer cell lines into metastable, plastic stem-like cells with neural stem cell features and maintain them as such through many transfers. Further studies show that this neural stem state is recapitulated by hormone or taxane treatments. These models may be useful for assessing new treatments to prevent resistance in prostate cancer patients.

3:55 – 4:05 p.m. **Discussion**

4:05 – 4:30 p.m. **Myc-linked Epigenetic Regulators in Prostate Cancer**

Sarki A. Abdulkadir, MD, PhD
Northwestern University
Chicago, IL

The MYC oncogene is involved at various stages of prostate cancer development and progression, including therapy resistance. While MYC has generally been recognized as a potential target in prostate and other cancers, it has been difficult to target therapeutically. I will discuss studies validating the MYC pathway as a target in prostate cancer and new strategies to target it therapeutically.

4:30 – 4:40 p.m. **Discussion**

4:40 – 4:55 p.m. **Travel Award Presentation #3**

ONECUT2 is a Targetable Master Regulator of Lethal Prostate Cancer Variants

Mirja Rotinen, PhD
Cedars-Sinai Medical Center
Los Angeles, CA

4:55 – 5:00 p.m. **Discussion**

5:00 – 7:00 p.m. **Poster Session I**

P1 – P38

6:45 – 7:00 p.m. **Presentation of 2017 Travel Awards**

Zongbing You, MD, PhD
Abstract and Travel Awards Committee Chair

7:00 p.m. **Evening on Own**

7:00 p.m. **Trainee Affairs Group Dinner**

*At attendee's expense. See Registration Desk to RSVP.

SATURDAY, NOVEMBER 11, 2017

Breakfast on Own

6:30 - 8:00 a.m. **Mission UroSciences Committee Meeting**

8:00 – 9:15 a.m. **Keynote Session: Perspectives on Epigenetic Modulation**

Introduction:

Hari Koul, MSC, PhD, FASN
SBUR President
LSU Health Sciences Center
Shreveport, LA

Discussion Leaders:

Cindy Miranti, PhD
University of Arizona Cancer Center
Tucson, AZ

Marc Cox, PhD
University of Texas at El Paxo
El Paso, TX

8:00 – 9:00 a.m. **Leland W.K. Chung Lecture**

Mitochondrial Reprogramming in Cancer

Dario Altieri, PhD
President & CEO

Wistar Institute Cancer Center
Philadelphia, PA

With a median survival of 15 to 36 months, multiple mechanisms of drug resistance, and constitutive insensitivity to leading therapies for other tumors, advanced, castration-resistant and metastatic prostate cancer remains an acutely unmet medical need that requires new therapeutic approaches. Recent experimental evidence has highlighted a novel role of mitochondria as pivotal disease drivers in these patients, fueling the machinery of tumor cell motility and invasion, while countering apoptosis of disseminated tumor cells. Although mechanisms of mitochondrial reprogramming affect a broad array of tumor responses in bioenergetics, redox balance and gene expression, these pathways are also druggable, offering fresh therapeutic opportunities in the management of patients with advanced prostate cancer.

9:00 – 9:15 a.m. **Discussion**

9:15 a.m. – 12:15 p.m. **Plenary Session III: Alternative Regulatory Mechanisms**

Discussion Leaders:

Kerstin Junker, MD, PhD
Saarland University Medical Center
Homburg/Saar, Deutschland

Todd Morgan, MD
University of Michigan
Ann Arbor, MI

9:15 – 9:40 a.m. **AR Splicing in Prostate Cancer**

Scott Dehm, PhD
University of Minnesota
Minneapolis, MN

Truncated variants of the androgen receptor (AR) are drivers of prostate cancer resistance to AR-targeted therapies and arise via alternative splicing mechanisms or genetic rearrangements that re-wire AR gene architecture. AR variants function as constitutively active transcription factors, but there are currently no therapies available to inhibit their expression or activity. We have found that alternative polyadenylation is a master regulator of AR splicing that coordinately controls expression of AR splicing variants including AR-V7 and AR-V9. Inhibiting alternative polyadenylation of AR variants blocks expression of these AR splicing variants and restores sensitivity of prostate cancer cells to AR-targeted therapies.

9:40 – 9:50 a.m. **Discussion**

9:50 – 10:05 a.m. **Break**

10:05 – 11:30 a.m. **miRNAs and Exosomes in Renal Cancer**

Kerstin Junker, MD, PhD

Saarland University Medical Center
Homburg/Saar, Deutschland

MicroRNAs (miRNA) are playing an essential role in posttranscriptional regulation of gene expression. They regulate several tumor associated processes like proliferation, migration, invasion and angiogenesis. The understanding of their role in specific tumor diseases can lead to the development of new therapeutic strategies. Because of the high stability of miRNAs they can be used as diagnostic, prognostic and predictive biomarkers. In addition, miRNAs are an important component of extracellular vesicles (EV). EVs are involved in intercellular communication between tumor cells and the tumor microenvironment. This talk will give an overview on the role of miRNAs and EVs in renal cell carcinoma and the development of miRNA biomarkers.

11:30 – 10:40 a.m. **Discussion**

10:40 a.m. – 12:00 p.m. **Discussion: Cellular Debris: Is It Really Useful?**

Discussion Leaders:

Susan Kasper, PhD
University of Cincinnati
Cincinnati, OH

Zongbing You, MD, PhD
Tulane University
New Orleans, LA

Panel:

Todd Morgan, MD
University of Michigan
Ann Arbor, MI

Edwin Posadas, MD
Cedars-Sinai Medical Center
Los Angeles, CA

Larisa Nonn, PhD
University of Illinois at Chicago
Chicago, IL

Michael Freeman, PhD
Cedars-Sinai Medical Center
Los Angeles, CA

12:00 – 1:30 p.m. **Lunch**

1:30 - 5:00 p.m. **Plenary Session IV: Model Systems in Urology**

Discussion Leaders:

Xin Li, PhD
Baylor College of Medicine
Houston, TX

Darius Jehan Bägli, MDCM FRCSC FAAP FACS
The Hospital for Sick Children and Research Institute
Toronto, ON

1:30 - 1:55 p.m. **SPOP Role in Prostate Cancer Oncogenesis**

Christopher Barbieri, MD
Weill Cornell Medical College
New York, NY

About 10% of prostate cancers have mutations in the SPOP gene – roughly 20,000 men will be diagnosed with SPOP mutant prostate cancer in the US each year. These cancers show distinct molecular features, and activation of specific signaling pathways. Our research provides insights into the biology underlying this subclass of prostate cancer, and will define the ability to specifically target these cancers, and identify novel diagnostic and therapeutic approaches across prostate cancer classes.

1:55 - 2:05 p.m. **Discussion**

2:05 - 2:30 p.m. **New BPH Mouse Model**

Jose Teixeira, PhD
Michigan State University
Grand Rapids, MI

Benign prostatic hypertrophy (BPH) affects older men and is a significant clinical burden because of lower urinary tract symptoms resulting from enlargement of the prostate in the peri-urethral region, but the precise mechanisms driving the disease have remained elusive. We have developed a mouse model of BPH by conditional deletion tumor suppressor genes that results in prostatic stromal growth, specifically in the caudal stromal remnant of the Mullerian duct. The affected signaling pathways and downstream expression targets in this model can be studied for clues to help us understand the development and progression in the human disease.

2:30 – 2:40 p.m. **Discussion**

2:40 – 3:05 p.m. **KLF5 Role in Prostate Cancer Epigenetics**

Jin-Tang Dong, PhD
Emory University School of Medicine
Atlanta, GA

The basic transcription factor KLF5 is both suppressive and promoting in prostatic carcinogenesis; and acetylation appears to be one mechanism for such a context-dependent function. Once acetylated, KLF5 becomes an effector of TGF-β signaling to suppress tumor growth, while its deacetylation leads to more rapid tumor growth. Understanding how KLF5 regulates the development and progression of prostate cancer could result in therapeutic opportunities.

3:05 – 3:15 p.m. **Discussion**

3:15 – 3:25 p.m. **AUA Research Update**

Carolyn Best, PhD
AUA Research
Linthicum, MD

To address the diversity of the needs for basic science in urologic research, the American Urological Association (AUA) Office of Research, through advocacy, education, and funding, supports existing and new opportunities for researchers to enhance their urologic research and careers. Through its collaborations with the SBUR, every year the AUA brings new prospects for federal and private funding, educational courses and conferences, publication and other communication platforms, resources that catalyze research, and other avenues for investigator support. New developments in these programs arise every year, and the Office of Research seeks to maximize the impact of support for research by increasing awareness for all SBUR members.

3:25 – 3:40 p.m. **Break**

3:40 - 4:05 p.m. **Mouse Model of LUTS**

Laura Lamb, PhD
Beaumont Hospital
Royal Oak, MI

Lower urinary tract symptoms (LUTS) are storage, voiding, and postmicturition symptoms affecting the lower urinary tract. LUTS prevalence rises markedly with age. This talk will focus on the utility of animal models for understanding the underlying mechanism and possible treatment strategies for two diseases with LUTS: radiation cystitis and underactive bladder.

4:05 - 4:15 p.m. **Discussion**

4:15 – 4:30 p.m. **Travel Award Presentation #4**

TRPV1 Modulates Pelvic Pain in a Murine Model of Chronic Prostatitis

Kenny Roman, PhD
Northwestern University
Evanston, IL

4:30 – 4:35 p.m. **Discussion**

4:35 – 5:00 p.m. **Prostate Epithelial Differentiation**

Cindy Miranti, PhD
University of Arizona Cancer Center
Tucson, AZ

A lack of understanding of the molecular mechanisms by which

oncogenes transform human prostate epithelial cells is limiting our ability to distinquish indolent from lethal disease. This talk will report on a newly identified transcriptional program that is driven by Pten loss in a human differentiation and tumorigenesis model.

5:00 – 5:10 p.m. **Discussion**

5:10 – 7:00 p.m. **Poster Session II**

P39 – P79

7:00 p.m. **Evening on Own**

SUNDAY, NOVEMBER 12, 2017

Breakfast on Own

8:00 a.m. - 12:00 p.m. **Plenary Session V: Epigenetic and Genetic Reprogramming**

Discussion Leaders:

Laura Lamb, PhD
Beaumont Health
Royal Oaks, MI

Conor Lynch, PhD
Moffit Cancer Center
Tampa, FL

8:00 - 8:25 a.m. **Digital Image Analysis and Computational Pathology**

Beatrice Knudsen, MD, PhD
Cedars-Sinai Medical Center
Los Angeles, CA

8:25 - 8:35 a.m. **Discussion**

8:35 - 9:00 a.m. **Nanoparticle Delivery of miRNA in Bladder Disease**

Darryl Martin, PhD
Yale University School of Medicine
New Haven, CT

One of the main challenges in treating urological cancers is overcoming tumor progression. To tackle this, we first utilized surface-modified nanoparticles that enhanced the targeting of specific cells. Then we loaded nanoparticles with cargo that focused on cancer signaling pathways involved in inhibiting tumor progression with the ultimate goal of reducing tumor burden in animal models.

9:00 - 9:10 a.m. **Discussion**

9:10 - 9:35 a.m. **Regenerative Pharmacology in Urologic Disease**

Koudy Williams, PhD
Wake Forest University

Winston-Salem, NC

Cell therapy for urinary incontinence has had mixed results with approximately 50% improvement in symptoms in 50% of the women. We have taken the approach that the best treatment for women with this chronic disease may be the use of small molecules that cells produce to stimulate cell mobilization to the urinary sphincter. When compared to local cell injections into the urinary sphincter of nonhuman primates with chronic urinary incontinence, injection of the chemokine CXCL12 (ready produced by cell at the sites of injury) did a much superior job in restoring urinary sphincter structure and function than the injection of skeletal muscle precursor cells (a common cell type used in cell therapies. We propose that the use of small molecules to stimulate tissue regeneration (Regenerative Pharmacology) is a safer, cheaper and more effective approach to stimulate tissue regeneration

9:35 – 9:45 a.m. **Discussion**

9:45 - 10:00 a.m. **Break**

10:00 - 10:15 a.m. **Travel Award Award Presentation #5**

A Novel Approach to Treat Neuroendocrine and Metastatic Prostate Cancer: Targeting S100A4 Protein by Small Molecule Inhibitor

Arsheed Ganaie, PhD
The Hormel Institute, University of Minnesota
Austin, MN

10:15 - 10:20 a.m. **Discussion**

10:20 – 10:45 a.m. **Targetin NGF in Overactive Bladder**

Pradeep Tyagi, PhD
University of Pittsburgh
Pittsburgh, PA

The pathophysiology of OAB remains unclear, but the aberrant sensation of urine volume in bladder is considered critical in the manifestation of urgency, an quintessential symptom of OAB. Nerve growth factor (NGF) represent a key molecular target in the neural pathways driving the aberrant bladder sensations. Studies targeting the expression and function of NGF have advanced the clinical care of OAB and uncovered new epigenetic targets in OAB pathophysiology.

10:45 – 10:55 a.m. **Discussion**

10:55- 11:20 a.m. **Neuroendocrine Differentiation in Castration-Resistant Prostate Cancer**

Amina Zoubeidi, PhD
University of Vancouver
Vancouver, British Columbia

Resistance to newly developed androgen receptor pathway inhibitors (ARPIs), such as abiraterone and enzalutamide, rapidly emerges and patients generally die within two years. In particular, a subset of patients who relapse following ARPI therapy exhibit lineage switching whereby tumours shed their dependence on AR signaling and emerge with neuroendocrine features. These tumours, termed treatment induced neuroendocrine prostate cancer (t-NEPC), carry an extremely poor prognosis and, to date, treatment remains decades old cytotoxic chemotherapy which carries a short-lived response at the cost of significant toxicity. Thus, the need to develop targeted treatments for this devastating disease is of paramount importance. Dr Zoubeidi will discuss how cell plasticity including cancer stem cells and neuroendocrine are mechanisms of ENZ resistance and why the transcription factor BRN2 is a major regulator/driver and a promising target for t-NEPC.

11:20 – 11:30 a.m. **Discussion**

11:30 a.m. **Farewell**

Ganesh Raj, MD, PhD
SBUR President

Travel Awardees

Podium Presentations

Full Abstracts

FRIDAY, NOVEMBER 10, 2017 10:35 – 10:50 A.M.

Androgenic to Estrogenic Switch in Prostate Gland as a Result of Epigenetic Silencing of Steroid 5-α Reductase 2

Zongwei Wang[1], Libing Hu, 021141, Keyan Salari, 021141, Seth Bechis, 021141, Rongbin Ge, 021142, Shulin Wu, 021141, Shahin Tabetabaei, 021141, Chin-Lee Wu, 021143, Douglas Strand4, Aria Olumi, 021141.

[1]Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA, [2]University of Massachusetts Medical Center, Worcester, MA, USA, 3Massachusetts General Hospital, Harvard Medical School, Belmont, MA, USA, 4UT Southwestern Medical Center, Dallas, TX, USA.

Background: The steroid 5-α reductase type 2 (SRD5A2) is critical for prostatic development and growth. Strategies to block SRD5A2 using 5-alpha reductase inhibitors (5ARI) remain a mainstay in the treatment of benign prostatic hyperplasia (BPH). However, one-third of men are resistant to 5ARI therapies. We previously showed that expression of SRD5A2 is not static, since epigenetic modulations by DNA methyltransferase and pro-inflammatory cytokines somatically silence SRD5A2 during adulthood. Here we wished to identify whether absence of prostatic SRD5A2, when androgenic pathways are blocked, leads to modification of alternate hormonal pathways.

Methods: Prostatic samples were obtained from patients with symptomatic BPH undergoing transurethral resection of prostate (TURP) surgery. Methylation of SRD5A2 promoter was assessed using Methylated CpG Island Recovery Assay (MIRA). RNA was extracted for whole-transcriptome profiling analysis by Illumina Human BeadChip Arrays. Prostatic protein expression of SRD5A2, androgen receptor (AR), estrogen receptor (ER) subunits, and aromatase were determined in a panel of six BPH patients by Western blot, immunohistochemistry (IHC), and ELISA assays. Prostatic levels of testosterone (T), dihydrotestosterone (DHT), estradiol (E) were measured by HPLC-MS. In in vitro study, primary prostatic stroma cells and epithelial cell line, BPH-1, were cultured and treated with TNF-α and IL-6, and mRNA levels were determined by qPCR.

Results: In prostate specimens that were methylated at the SRD5A2 promoter locus, estrogen response genes were identified as one of the most significantly upregulated gene family members as determined by gene expression analysis. The levels of T, E and aromatase were significantly upregulated, while DHT was significantly decreased. Absence of SRD5A2 significantly upregulated the phosphorylation of ERα (pERα), but did not significantly affect the levels of total ERα, total ERβ or pERβ. In primary prostatic stromal cells, administration of TNF-α, but not IL-6, suppressed the level of SRD5A2 and upregulated aromatase activity and ERα expression. However, treatment of prostatic epithelial cells with TNF-α or IL-6 did not change the androgenic or estrogenic signalling, suggesting that stromal cells regulate the androgenic to estrogenic switch when SRD5A2 is absent.

Conclusions: Our study demonstrates for the first time that there is an androgenic to estrogenic switch when SRD5A2 is absent in the prostate gland. Somatic epigenetic silencing of SRD5A2 changes the prostatic hormonal milieu, and may modulate prostatic homeostasis and growth. Targeting the aromatase-estrogen-ER axis may serve as an effective treatment strategy in BPH patients who lack SRD5A2 expression.

FRIDAY, NOVEMBER 10, 2017 11:30 – 11:45 A.M.

Loss of Epigenetic Regulation Results in Congenital Malformations of the Urogenital System

Catherine Seager[1], Mary Taglienti[1], Feng Shen[2], Jordan Kreidberg[1].

[1]Boston Children's Hospital, Boston, MA, USA, [2]Washington University School of Medicine, St. Louis, MO, USA.

Background: We are investigating the role of epigenetics in the development of the urogenital system with the goal of understanding how impaired epigenetic regulation may lead to congenital anomalies such as ureteropelvic junction obstruction and ureterovesical junction obstruction. Reciprocal epithelial mesenchymal inductive interactions are critical to regulating gene expression during development of the kidney and the lower urogenital tract. The mature pelvis and ureter consist of an inner epithelial compartment and an outer mesenchymal compartment. The ureteric bud gives rise to the epithelium of the collecting system while the smooth muscle and fibroblasts of the ureter are derived from a TBX-18-expressing mesenchyme. We are using gene targeting in mice to inactivate the Eed gene, an essential compoment of Polycomb Regulatory Complex 2, in either the epithelial or mesenchymal compartments during development of the urogenital system.

Methods: Eed was inactivated in the epithelial and mesenchymal compartments of the collecting system and ureter using HoxB7-Cre and TBX18-Cre respectively. Kidneys and lower

urogenital tract phenotypes were characterized through histological and gene expression analyses. Functional assessment of drainage was performed by ink injection study.

Results: Conditional inactivation of Eed in the epithelium (Eed flox/flox;HoxB7-Cre) resulted in animals with severe bilateral hydroureteronephrosis by postnatal day 21 (P21). Kidneys from Eed flox/flox;HoxB7-Cre mice on P1 were decreased in size as compared to controls indicating that correct epigenetic regulation in the ureteric bud lineage is necessary for the maintenance of nephrogenesis. Eed flox/flox;HoxB7-Cre mice showed decreased expression of uroplakin at the ureteropelvic junction (UPJ) while ink injection studies suggested impaired drainage at the level of the bladder.

Conditional inactivation of Eed in the ureteral mesenchymal progenitors (Eed flox/flox-;TBX18-Cre) resulted in bilateral hydroureteronephrosis by P21. There was increased expression of smooth muscle actin (SMA) and SM22-alpha at the ureteropelvic junction and ink injection studies showed impaired drainage at the level of the UPJ. Vesicoureteral reflux was noted on injection of dye into the bladder.

Conclusions: Inactivation of the Eed gene in epithelial and mesenchymal cell-type progenitors of the ureter and collecting system results in abnormalities of gene expression during ureteral and renal development. These studies are providing insights about how urogenital anomalies in humans may be caused by abnormal epigenetic regulation during fetal and early postnatal development.

FRIDAY, NOVEMBER 10, 2017 4:40 – 4:55 P.M.

ONECUT2 is a Targetable Master Regulator of Lethal Prostate Cancer Variants

Mirja Rotinen1, Sungyong You1, Julie Yang1, Simon Coetzee1, Wen-Chin Huang1, Fangjin Huang1, Xinlei Pan1, Alberto Yáñez1, Dennis Hazelett1, Chia-Yi Chu1, Leland Chung1, Stephen Freedland1, Dolores Di Vizio1, Isla Garraway2, Ramachandran Murali1, Beatrice Knudsen1, Michael Freeman1.

1Cedars-Sinai Medical Center, Los Angeles, CA, USA, 2UCLA, Los Angeles, CA, USA.

Background: Androgen deprivation therapy is associated with the emergence of aggressive variants of metastatic castration-resistant prostate cancer (mCRPC) that exhibit little or no dependence on the androgen receptor (AR). Here we identify the developmental transcription factor ONECUT2 as a negative regulator of the AR axis, that emerge in aggressive prostate cancer (PC) variants to control transcriptional networks linked to CRPC and neuroendocrine (NE) differentiation. We further show that ONECUT2 can be targeted with a small molecule that inhibits CRPC metastasis in mice.

Methods: ONECUT2 was confirmed as a mCRPC-relevant protein and to be targetable by: computational modeling and bioinformatics, enforced expression, silencing, microarray, ChIP-Seq, immunohistochemistry, immunofluorescence, quantitative imaging, functional assays, in vivo experiments and surface plasmon resonance.

Results: We have performed a master regulator analysis using transcriptome datasets from 260 samples of mCRPC and developed a model transcription factor network that associates OC2 with metastatic progression. Gene expression profiling of ONECUT2-engineered PC cell lines, has allowed us to generate a ONECUT2 activity signature that reveals high positive correlation with pro-neural and NE prostate cancer (NEPC) signatures, and a negative correlation with AR activation pathways. We find that ONECUT2 is a negative

regulator of AR expression and a repressor of its transcriptional program through direct binding to AR target genes. We also find that ONECUT2 is significantly increased in human NEPC and that confers NE properties to CRPC through direct up-regulation of the NEPC driver PEG10 and direct down-regulation of the NEPC inhibitor FOXA1. Finally, we show that ONECUT2 is required for cell growth and survival and that it can be targeted with a small molecule that, by binding to its C-terminal DNA binding domain, inhibits CRPC growth and metastasis in mice.

Conclusions: OC2 is a master regulator of aggressive mCRPC variants that drives AR-dependent adenocarcinoma toward NEPC differentiation by blocking AR/FOXA1-activity and inducing PEG10. OC2 can be targeted with a small molecule that inhibits growth and metastasis in mice. Thus, patients with OC2 active tumors could benefit from OC2 inhibitor therapy.

SATURDAY, NOVEMBER 11, 2017 4:15 – 4:30 P.M.

TRPV1 Modulates Pelvic Pain in a Murine Model of Chronic Prostatitis

Kenny Roman, PhD, Anthony J. Schaeffer, MD, Praveen Thumbikat, DVM, PhD.

Northwestern University, Chicago, IL, USA.

Background: Patients with chronic prostatitis/chronic pelvic pain syndrome (CP/CPPS) develop multiple symptoms that include chronic pain (from various pelvic regions) and the cause is poorly understood. Therefore, reliable treatment options are lacking. The goal of the proposed project is to elucidate the pathogenesis of CP/CPPS by identifying TRPV1 receptor as a mediator of chronic pelvic pain in an autoimmune mouse model of CP/CPPS, called experimental autoimmune prostatitis (EAP). Also, this project will determine the role of TRPV1 in peripheral inflammation by measuring the levels of activated mast cells and inflammation scoring of the prostate tissue.

Methods: C57BL/6 (B6) and TRPV1 knockout (KO) male mice (2-3 months old), were used for this study. Mice were injected with prostate antigen (1 mg/ml) subcutaneous to elicit EAP. To determine whether mice exhibited prostate-specific pelvic pain we measured for suprapubic tactile allodynia on days 0, 7, 10, 14, and 20. The prostate, dorsal root ganglia (DRG), and spinal cord were excised from mice with EAP and respective control cohorts at day 20. The prostate, DRG, and spinal cord were processed for immunoblotting or immunohistochemistry.

Results: Our behavioral measurements verified that EAP mice develop pelvic pain at day 7 thru day 20. However, TRPV1-KO mice do not develop increased tactile pelvic allodynia compared to control cohorts at day 20. Our immunoblots showed that p-ERK1/2 increased in the DRG and spinal cord (lumbosacral) of mice with EAP at day 20, compared to control. However, the DRG and spinal cord excised from TRPV1-KO with EAP at day 20 showed no changed in p-ERK1/2 expression compared to respective controls. In addition, the excised prostate lobes were stained with Toluidine blue and we observed increased mast cell activation in mice with EAP at day 20, compared to control. In contrast, mast cell activation was reduced in TRPV1-KO mice with EAP, compared to controls. Interestingly, inflammation scores showed that the prostates excised from TRPV1-KO mice with EAP have reduced inflammation compared to mice with EAP at day 20.

Conclusions: Our project determined that 1) TRPV1-K0 mice with EAP do not develop pelvic pain, 2) the lumbosacral DRG and spinal cord from TRPV1-KO mice with EAP do not have increased ERK1/2 phosphorylation, 3) TRPV1-KO mice with EAP showed diminished mast cell activation, and 4) overall prostate inflammation might be reduced in TRPV1-KO mice with EAP. We propose that targeted TRPV1 inhibition may alleviate chronic pain symptoms associated with CP/CPPS.

SUNDAY, NOVEMBER 12, 2017 10:00 – 10:15 A.M.

A Novel Approach to Treat Neuroendocrine and Metastatic Prostate Cancer: Targeting S100A4 Protein by Small Molecule Inhibitor

Tabish Hussain[1], **Arsheed A. Ganaie**[1], Teri Johnson[1], Reihana Maqbool[1], Badrinath R. Konety[2], Mohammad Saleem[1].

[1]The Hormel Institute University of Minnesota, Austin, MN, USA, [2]University of Minnesota, Minneapolis, MN, USA.

Background: NE-PCa an aggressive variant of prostate cancer (PCa) is non-responsive to androgen deprivation therapy (ADT) and shows poor prognosis in patients. We recently showed that S100A4 is an oncoprotein that drives the development of NE-PCa in transgenic GEM model. We showed that genetic targeting of S100A4 inhibits the development of NE-CaP. These data formed the basis of our hypothesis that S100A4 is a drug amenable protein, and could be exploited as a drug-target for treating NE-PCa in patients. We hypothesize that inhibition of S100A4 protein using small molecular inhibitors is an ideal approach to treat NE-PCa disease.

Results: We show that S100A4 secreted by prostatic tumors confers metastatic and NE-PCa characteristics to indolent tumor cells. We show that serum-S100A4 levels highly elevated in PCa patients positively correlate to tumor stage. Next, we screened a library of 5000 molecules in-silico for S100A4 binding, and identified inhibitors (SMI1 and SMI2). Using isothermal titration Calorimetry (ITC) and Surface Plasmon Resonance (SPR) assays, we show that SMI1 and SMI2 physically bind to recombinant S100A4 protein. Using Myosin-II protein disassembly and turbidity as indices of S100A4 activity, we showed that SMI1 and SMI2 inhibit S100A4 activity. We previously showed that soluble S100A4 induces the invasiveness via RAGE receptor on tumor cells. Using an ELISA assay we show that SMI2 inhibits the binding of soluble S100A4 protein to RAGE protein. We developed TdT/luc-expressing stable NE-CaP models from PC3M, PC3M-LN4 and TRAMPC2 and tested efficacy of inhibitors using these models in-vitro and in-vivo. SMI1 and SMI2 treatment decreased the activation of downstream targets (MMP9 and NFκB) of S100A4 in NE-PCa models. PCa cells are known to cross endothelial barrier of blood vessels and home at bones. Next using transendothelial and bone homing/alizarin assay, we tested the anti-metastatic efficacy of the inhibitors and show that SMI2 treatment inhibited the (i) potential of NE-PCa cells crossing the endothelial barrier and (ii) attachment of NE-PCa cells to bone marrow-derived hMSCs. Notably, SMI1 and SMI2 therapies inhibited the growth, prostatosphere formation, migration and invasion of NE-PCa models. Finally, SMI1 and SMI2 therapy caused a significant reduction in the growth/proliferation of NE-PCa tumors in syngeneic and athymic xenograft mouse models.

Conclusion: S100A4 inhibition is a novel therapeutic approach NE-PCa treatment and S100A4 inhibitors are the potential drug candidates for treating NE-PCa in humans.

Poster Session I – Friday Evening

View Full Abstracts at www.SBUR.org

Friday, November 10, 2017, 5:00 pm – 7:00 pm

Grand Foyer

P1 – P38

P1

Effects of Phthalates on Steroidogenesis in Prostate Cancer Cells

Clara Jeong, William Ricke.

University of Wisconsin-Madison, Madison, WI, USA.

P2

Genetic Regulation of Prostate Cancer by the Androgen Receptor and the Class E Basic Helix-Loop-Helix Transcription Repressor BHLHE40

Paramita M. Ghosh, Ph.D., Zsofia Kiss, Ph.D.

VA Northern California Health Care System, Mather, CA, USA.

P3

Sensitizing Castration-Resistant Prostate Cancer to Anti-androgens By Targeting Gastrin-releasing Peptide Receptor

Renjie Jin.

Vanderbilt University, Nashville, TN, USA.

P4

BCG Intravesical Therapy Modulates Immune Response against Bladder Cancer by Suppressing Pd-L1 Expression

Junhyeok Heo[1], Kyung Seok Han[2], Kang Su Cho[2], Woong Kyu Han[1], Joon Chae Na[2], Sook Young Kim[2], Sung Joon Hong[1].

[1]Department of urology and urological science institute, Brain Korea 21 PLUS Project for Medical Science, Yonsei University, Seoul, Korea, Republic of, [2]Department of Urological Science Institute, Yonsei University College of Medicine, Seoul, Korea, Republic of.

P5

Progranulin/Epha2 Axis, a Novel Driver of Bladder Cancer

Shi-Qiong Xu[1], Simone Buraschi[1], Thomas Neill[1], Leonard G. Gomella[1], Antonino Belfiore[2], Renato V. Iozzo[1], Andrea C. Morrione[1].

[1]Thomas Jefferson, Philadelphia, PA, USA, [2]Universita' Della Magna Graecia, Catanzaro, Italy.

P6

Combined Targeting of EZH2 and Androgen Receptor in Castration-Resistant Prostate Cancer Cells.

Eswar Shankar, Ph.D.[1], Daniel Franco, BS[2], Omair Iqbal, BS[2], Stephen Moreton, BS[3], Rajnee Kanwal, Ph.D.[1], Sanjay Gupta, Ph.D.[4].

[1]Department of Urology, Case Western Reserve University, The Urology Institute, University Hospitals Cleveland Medical Center, Cleveland, OH, USA, [2]Department of Urology, Case Western Reserve University,, Cleveland, OH, USA, [3]Louis Stokes Veterans Administration Medical Center, Cleveland, OH, USA, [4]Department of Urology, Case Western Reserve University, The Urology Institute, University Hospitals Cleveland Medical Center, Louis Stokes Veterans Administration Medical Center, Cleveland, OH, USA.

P7 - TRAVEL AWARD

Using Metabolic Pathways to Improve Diagnosis and Risk-Stratification of Prostate Cancer

Aymen Alqazzaz[1], Dexue Fu, PhD[1], Arman

Karimi, PhD[2], Gustavo Ferreira, PhD[2], Mary C. McKenna, PhD[2], Mohummad Minhaj Siddiqui, MD[1].

[1]Division of Urology, Department of Surgery, University of Maryland School of Medicine, Baltimore, MD, USA, [2]Department of Pediatrics, University of Maryland School of Medicine, Baltimore, MD, USA.

P8

Androgen Regulates Arginine Metabolism in Prostate Cancer

Dexue Fu, MD., Ph.D[1], Min Xu[1], Aymen Alqazzaz[1], Jee-Hoon Song, Ph.D[1], Hubert Huang[1], Lingbu Wang[1], Mohammad Afnan Khan[1], Krish Chandrasekaran[1], Arif Hussain[1], Ganesh Sriram, Ph.D[2], Mohummad Minhaj Siddiqui, MD[1].

[1]University of Maryland School of Medicine, Baltimore, MD, USA, [2]University of Maryland College Park, Baltimore, MD, USA.

P9

ABCB1 Remains as a Dominant Factor Driving Acquired Cabazitaxel Resistance

Alan Lombard, PhD, Chengfei Liu, PhD, Cameron Armstrong, PhD, Vito Cucchiara, MD, Xinwei Gu, MD, Wei Lou, MD, Christopher Evans, MD, Allen Gao, MD, PhD.

University of California, Davis, Sacramento, CA, USA.

P10

WITHDRAWN

P11

A Selective LSD1 Inhibitor Enhanced Anti-prostate Cancer Effect of Enzalutamide by Induction of the Nur77 Mediated Apoptotic Pathway in Prostate Cancer Cells

Noriko Yokoyama, PhD[1], Victor Pham, BS[1], Yi-Chao Zheng, PhD[2], Hongmin Liu[2], Xiaolin Zi[1].

[1]University of California, Irvine, Orange, CA, USA, [2]Zhengzhou University, Zhengzhou, China.

P12 - TRAVEL AWARD

Macrophage Plasticity in Bone Metastatic Prostate Cancer

ChenHao Lo, MS, Etienne Baratchart, PhD, David Basanta, PhD, Conor Lynch, PhD.

Moffitt Cancer Center, Tampa, FL, USA.

P13 - TRAVEL AWARD

P53 and Rb Are Required In Ell2 Suppression of Prostate Cancer Cell Proliferation and Migration

Mingming Zhong, PhD[1], Laura E. Pascal[1], Xiaonan Qiu[2], Yibin Zhou[3], Yao Wang[4], Zhou Wang[1].

[1]University of Pittsburgh, Pittsburgh, PA, USA, [2]Tsinghua University, Beijing, China, [3]The Second Affiliated Hospital of Soochow University, Soochow, China, [4]China-Japan Union Hospital of Jilin University, Changchun, China.

P14

Novel Small Molecule Inhibition of The Function and Level of Androgen Receptor In Castration-resistant Prostate Cancer

Zhenyu Yang[1], James Johnson[2], Laura Pascal[1], Dan Wang[1], Jianhua Zhou[1], Joel Nelson[1], Peter Wipf[3], Zhou Wang[1].

[1]Department of Urology, University of Pittsburgh, Pittsburgh, PA, USA, [2]Department of Chemistry, University of Pittsburgh, Pittsburgh, PA, USA, [3]Department of Pharmaceutical Sciences, School of Pharmacy, University of Pittsburgh, Pittsburgh, PA, USA.

P15 - TRAVEL AWARD

Regulation of Androgen Receptor of Dhx15 in Prostate Cancer

Yadong Xu[1], Laura E. Pascal[1], Fangming Deng[2], Zhou Wang[1].

[1]UPMC Shadyside, Pittsburgh, PA, USA, [2]Department of Pathology, NYU School of Medicine, New York, NY, USA.

P16

Radiation Induced Endoglin Mediates Prostate Cancer Radio-resistance Via Metabolic Reprogramming

Anisha Madhav, Frank Duong, Neil A. Bhowmick.

Cedars-Sinai Medical Center, Los Angeles, CA, USA.

P17

Androgen Regulation of Fibroblast Growth Factor-5 in Prostate Cancer

Dalton McLean.

University of Wisconsin, Madison, WI, USA

P18

Megacystis-Microcolon-Intestinal Hypoperistalsis and Prune Belly Syndrome: Overlapping Genetic Variants Cause Overlapping Phenotypes

Nida Iqbal, PhD, Thomas Jascur, PhD, Shaohua Zhang, Linda Baker, MD.

UT Southwestern Medical Center, Dallas, TX, USA.

P19

Tumor Communication to Bone When SCF is Deleted

Brittni M. Foster, Lihong Shi, MD, Bethany Kerr, PhD, Phyillis Elliot, Mary E. Mobley, Koran Harris, Christina M. Snyder.

Wake Forest University School of Medicine, Winston Salem, NC, USA.

P20 - TRAVEL AWARD

Notch3 Promotes Prostate Cancer-Induced Bone Metastasis in a MMP3-dependent manner

Sourik S. Ganguly, Ph.D[1], Xiaohong LI, Ph.D[1], Cindy K. Miranti, Ph.D[2].

[1]Van Andel Research Institute, Grand Rapids, MI, USA, [2]The University of Arizona, Tucson, AZ, USA.

P21 - TRAVEL AWARD

Androgen Receptor Variants Mediate DNA Repair Following Radiation in Prostate Cancer

Yi Yin[1], Rui Li[1], Felix Y. Feng[2], Johann S. de Bono[3], Scott M. Dehm[4], Ram S. Mani[5], Ganesh V. Raj[6].

[1]Department of Urology, The University of Texas Southwestern Medical Center, Dallas, TX, USA, [2]Departments of Radiation Oncology, Urology, and Medicine, University of California at San Francisco, San Francisco, CA, USA, [3]Drug Development Unit and Prostate Cancer Targeted Therapy Group, The Royal Marsden NHS Foundation Trust and The Institute of Cancer Research, London, United Kingdom, [4]Masonic Cancer Center and Departments of Laboratory Medicine and Pathology and Urology, University of Minnesota, Minneapolis, MN, USA, [5]Department of Pathology and Urology, The University of Texas Southwestern Medical Center, Dallas, TX, USA, [6]Department of Urology and Pharmacology, The University of Texas Southwestern Medical Center, Dallas, TX, USA.

P22

The Impact of Altered Steroidogenesis on Estrogen Receptor Activation in Benign Prostatic Hyperplasia

Teresa T. Liu, Clara H. Jeong, Emily A. Ricke, William A. Ricke.

University of Wisconsin - Madison, Madison, WI, USA.

P23 - TRAVEL AWARD

Effect of CXCR6/CXCL16 Axis on Efficacy of Docetaxel in Prostate Cancer

Neeraj Kapur[1], Hina Mir[1], Guru Sonpavde[2], Shailesh Singh[1].

[1]Morehouse School of Medicine, Atlanta, GA, USA, [2]Dana Farber Cancer Institute, Boston, MA, USA

P24

WITHDRAWN

P25 - TRAVEL AWARD

Anti-cancer Activity of Novel Chalcones against Highly- Metastatic Prostate Cancer Cells

Metsiel Donate[1], Maria Sanchez, Master Degree[1], Joseph Mooney[2], Magaly Martinez Ferrer, Ph.D[1], David Sanabria, Ph.D[3].

[1]CCC UPR, San Juan, Puerto Rico, [2]Inte American University of Puerto Rico, San Juan, Puerto Rico, [3]Inter American University of Puerto Rico, San Juan, Puerto Rico.

P26

The Co-alteration Landscape Of ARID1A Mutated High Grade Bladder Cancer

Eugene J. Pietzak, III, Victor McPherson, Sumit Isharwal, Shawn Dason, Francois Audnet, Ahmed Zehir, Niki Schultz, Michael Berger, Jonathan Rosenberg, Dean Bajorin, Hikmat Al-Ahmadie, Gopa Iyer, Bernard Bochner, David Solit.

Memorial Sloan Kettering Cancer Center, New York, NY, USA.

P27

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P28

Role of Eukaryotic Translation Initiation Factor 4 Gamma 1 (eif4g1) In Prostate Cancer

Praveen K. Jaiswal, Ph.D., Sweaty Koul, Hari K. Koul, PhD.

Louisiana Health Science Center, Shreveport, La, USA.

P29

Mesenchymal Compartmentalization Of Mir-1 And Mir-143 In Prostate Tissue And Loss Of Expression In Tumor-associated Stroma

Binod Kumar, Avi Rosenberg, Su Mi Choi, Karen Fox-Talbot, Angelo De Marzo, Larisa Nonn, W Nathaniel Brennen, Luigi Marchionni, Marc K. Halushka, Shawn E. Lupold.

Johns Hopkins University School of Medicine, Baltimore, MD, USA.

P30

Chromosomal Inequality Causes the Sex Disparities in Bladder Cancer through a KDM6A-dependent Epigenetic Mechanism

Satoshi Kaneko, Ph.D., Xue Li, Ph.D.

Boston children's hospital, Boston, MA, USA.

P31

Genomic Alterations In The cfDNA Of Castrate Resistant Prostate Cancer Patients

Elisa M. Ledet, PhD, Joshua Schiff, BS, Bryce Christensen, BS, Marcus Moses, MS, Lynne Chapman, BS, Ashkan Shahbandi, BS, Peter Steinwald, MS, Patrick Cotogno, MS, Jodi Layton, MD, Brian Lewis, MD, Oliver Sartor, MD.

Tulane University, New Orleans, LA, USA.

P32 - TRAVEL AWARD

Mesenchymal Stem Cells Promote Osteogenesis and the Evolution of Apoptosis Resistant Bone Metastatic Prostate Cancerv

Jeremy J. McGuire[1], Leah Cook, PhD[2], Jeremy Frieling, PhD[2], Conor Lynch, PhD[2].

[1]Tumor Biology / Cancer Biology PhD Program, Moffitt Cancer Center / University of South Florida, Thonotosassa, FL, USA,

[2]Moffitt Cancer Center / University of South Florida, Tampa, FL, USA.

P33

Activated Gli is the Primary Driver of Prostate Cancer Cell Growth

Na Li, PhD, Sarah Truong, Mannan Nouri, B.S., Jackson Moore, Ralph Buttyan, PhD.

The Vancouver Prostate Centre, Vancouver, BC, Canada.

P34

Fibroblast Heterogeneity and Inflammatory Cell Recruitment in Prostate Cancer

Nadia M. Attalah[1], Renee E. Vickman[2], Omar E. Franco[2], Timothy L. Ratliff[1], Simon W. Hayward[2].

[1]Purdue University Center for Cancer Research, West Lafayette, IN, USA, [2]NorthShore University Health System, Evanston, IL, USA.

P35

PEDF Acts as a Bridging Protein in a Novel Lipid-Centrosomal Signaling Axis in Prostate Cancer-Associated Fibroblasts

Susan E. Crawford, Francesca Nardi, Philip Fitchev, Omar E. Franco, Lijun Huang, Jelena Ivanisevic, Adrian Scheibler, Simon W. Hayward.

North Shore Univ. Research Institute, Evanston, IL, USA.

P36

Nuclear Factor I/B Interacts with Androgen Receptor to Regulate Response to Anti-androgens

Wisam N. Awadallah[1], Robert J. Matusik, PhD[2], Magdalena M. Grabowska, PhD[1].

[1]Case Western Reserve University, Cleveland, OH, USA, [2]Vanderbilt University Medical Center, Nashville, TN, USA.

P37

Thrombospondin-1 Regulates Lipolytic Activity in Prostate Cancer Cells

Susan Wcislak[1], Ayesha Chawla[1], Nizar Khamjan[1], Beth A. Plunkett[2], Jennifer A. Doll[1].

[1]University of Wisconsin Milwaukee, Milwaukee, WI, USA, [2]NorthShore University Health System, Evanston, IL, USA.

P38

Novel Race-specific Genetic and Epigenetic Determinants of Enzalutamide-resistance In Prostate Cancer

Arsheed A. Ganaie[1], Tabish Hussain[1], Marina Ferrari[1], Matteo Astone[2], Luke Hoeppner[1], Todd Schuster[1], Paari Murugan[3], Badrinath R. Konety[3], Mohammad Saleem[2].

[1]Hormel Institute, University Of Minnesota, Austin, MN, USA, [2]Hormel Institute, University Of Minnesota, Austin, MN, USA, [3]University Of Minnesota, Minneapolis, MN, USA.

Poster Session 2 – Saturday Evening

View Full Abstracts at www.SBUR.org

Saturday, November 11, 2017, 5:10 pm - 7:00 pm

Grand Foyer

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P39

Concurrent Deletion of Eaf2 and Ell2 Induces Murine Prostate Intraepithelial Neoplasia

Laura E. Pascal, PhD, Mingming Zhong, Uma Chandran, Khalid Masoodi, Yachen Zang, Lora H. Rigatti, VMD, Zhou Wang.

University of Pittsburgh, Pittsburgh, PA, USA.

P40

PDEF Inhibits Prostate Cancer Metastasis by Promoting Luminal Differentiation

Fengtian Wang[1], Sweaty Koul[2], Prakash Srinivasan Timiri Shanmugam[2], Qin Dong[2], Hari K. Koul[2].

[1]Louisiana State University Health Sciences Center-Shreveport, Shreveport, LA, USA, [2]Louisiana State University Health Sciences Center-Shreveport, Shreveport, LA, USA.

P41

Members of the Transcription Factor Activator Protein 2 Family Are Markers of the Basal Molecular Subtype of Human Bladder Cancer and Repressed By Peroxisome Proliferator Activated Receptor Gamma

Hironobu Yamashita[1], Zongyu Zheng, PhD[1], Lauren Shuman1, Truc Tran[1], Osei-Amponsa Vasty[1], Joshua Warrick[1], Jay Raman, MD[2], Trevor Williams[3], David Degraff[1].

[1]The Departments of Pathology and Surgery, Division of Urology, Penn state Hershey, Hershey, PA, USA, [2]The Division of Urology, Penn state Hershey, Hershey, PA, USA, [3]The University of Colorado Medical Center, Aurora, CO, USA.

P42

A Better Alternative for Prostate Cancer Diagnosis by Using Organic Compounds in Urine

Qin Gao, MS[1], Xiaogang Su, PhD[1], Heinric Williams, MD[2], Thomas Prince, PhD[2], Brielle R Schreiter, B.S.[2], Wen-Yee Lee, PhD[1].

[1]University of Texas at El Paso, El Paso, TX, USA, [2]Urologic Oncology Geisinger Medical Center, Danville, PA, USA.

P43

Endodermal Dnmt1 Maintains an Endoderm-Mesoderm Junction in the Developing Urogenital Tract

Diya Binoy Joseph, BS, Anoop Chandrashekar, Lisa L. Abler, PhD, Chad M. Vezina, PhD.

University of Wisconsin-Madison, Madison, WI, USA.

P44

Addition of Stroma Enhances Branching Morphogenesis and AMACR Expression in a 3D Organoid Co-Culture Model of Prostate Cancer

Zachary Richards[1], Morgan Zenner[1], Joseph Marsili[2], Jacob Manlucu[2], Tara McCray[1], Klara Valyi-Nagy[3], Larisa Nonn[4].

[1]University of Illinois at Chicago, Chicago, IL, USA, [2]Northeastern Illinois University, Chicago, IL, USA, [3]University of Illinois Biorepository, Chicago, IL, USA, [4]University of Illinois Cancer Center, Chicago, IL, USA.

P45

Challenging the Free Hormone Hypothesis for Vitamin D in the Prostate Has Implications for the Prostate Cancer Disparity in African American Men

Zachary Richards[1], Jason Garcia[1], Ryan Deaton[2], Andres Acosta[1], Klara Valyi-Nagy[3], Virgilia Macias[1], Peter H. Gann[4], Larisa Nonn[4].

[1]University of Illinois at Chicago, Chicago, IL, USA, [2]University of Illinois at Chicago, Research Histology & Tissue Imaging, Chicago, IL, USA, [3]University of Illinois at Chicago, UI Biorepository, Chicago, IL, USA, [4]University of Illinois at Chicago, UI Cancer Center, Chicago, IL, USA.

P46

Characterization of Androgen Response in Organoids Derived from a Novel Metastatic Castration Recurrent PDX

Diamonds A. Banks[1], Shruti Shah, M.S.[2], Barbara Foster, Ph.D.[2].

[1]Howard University, Washington, DC, USA, [2]Roswell Park Cancer Institute, Buffalo, NY, USA.

P47

Inactivation of Foxa1 and Pten Results in Development of Carcinoma in Situ and Basal Subtype of Muscle Invasive Bladder Cancer

Vasty O. Amponsa[1], Jenna M. Buckwalter, Ph.D.[1], Vonn Walter, Ph.D.[1], Joshua Warrick, M.D.[1], Xue-Ru Wu, M.D.[2], Jay D. Raman, M.D.1, David J. DeGraff, Ph.D.[1].

[1]Penn State Hershey College of Medicine, HERSHEY, PA, USA, [2]New York University, New York, NY, USA.

P48

Pro-fibrotic Signaling in Prostate Stroma Increases Prostate Collagen Fiber Density and Disrupts Normal Urinary Function in Male Mice

Kyle A. Wegner[1], Jinjin Guo[2], Jill A. McMahon[2], Nobuyo Maeda[3], Peiqing Wang[1], Dale E. Bjorling[1], Paul C. Marker[1], Andrew P. McMahon[2], Chad M. Vezina[1].

[1]University of Wisconsin - Madison, Madison, WI, USA, [2]University of Southern California, Los Angeles, CA, USA, [3]University of North Carolina at Chapel Hill, Chapel Hill, NC, USA.

P49

Delayed Treatment of Tcf4 Inhibitor, Pkf118-310, Attenuated Enzalutamide Resistant Prostate Cancer Cell Growth in Xenograft Mice

Geuntaek Lee, Ph.D., Isaac Y. Kim, M.D., Ph.D.

Rutgers, the Cancer Institute of New Jersey, new brunswick, NJ, USA.

P50

Pathogenesis of Inflammation in Human BPH

Gervaise Henry, MS, Alicia Malewska, MS, Ryan Mauck, MD, Ryan Hutchinson, MD, Jeff Gahan, MD, Franto Francis, MD, Jose Torrealba, MD, Claus Roehrborn, MD, Douglas W. Strand, PhD.

UT Southwestern Medical Center, Dallas, TX, USA.

P51

CREB1 and ATF1 Differentially Regulate Terminal Prostate Luminal Cell Differentiation by Controlling the Timing of ING4 Expression, while CREB1 Prevents ING4 Expression upon PTEN Loss in Prostate Cancer

McLane Watson[1], Penny Berger[1], Sander B. Frank, PhD[2], Mary Winn, PhD[1], Cindy Miranti, PhD[2].

[1]Van Andel Research Institute, Grand Rapids, MI, USA, [2]University of Arizona, Tucson, AZ, USA.

P52

Hormonal Modulation of DDX3 and its Implications on Translational Regulation in Prostate Cancer

Jordan Vellky.

University of Wisconsin-Madison, Madison, WI, USA.

P53 - TRAVEL AWARD

Protocadherin 7 is Overexpressed in Advanced Prostate Cancer and Promotes Prostate Cancer Progression by Modulating MEK and AKT Signaling

Gauri Shishodia, Ph.D., Prakash Srinivasan Timiri Shanmugam, Ph.D., Sweaty Koul, MS, Hari Koul, Ph.D..

LSUHSC-S, Shreveport, LA, USA.

P54

A Prostate Tumor Neo-Antigen Polyionic Virus-Like Particle Vaccine Shows Immunogenicity and Efficacy in a Mouse Model of Advanced Prostate Cancer

Brian Simons, Fabiana Cannella, Ashley Ross, Raphael Viscidi.

Johns Hopkins School of Medicine, Baltimore, MD, USA.

P55

Oncogenic Properties of Tumor Suppressor Protein STAG2 in Muscle-invasive Bladder Cancer

Lanni Aquila, Nithya Krishnan, Monika Rak, Swathi Ramakrishnan, Kristopher Atwood, Carl Morrison, Candace Johnson, Khurshid Guru, Jianmin Wang, Anna Woloszynska-Read.

Roswell Park Cancer Institute, Buffalo, NY, USA.

P56

DNA Methylation Alterations Contribute To Differences in Prostate Cancer Biology in African American and European American Men

Swathi Ramakrishnan, Xuan Peng, Qianya Qi, Qiang Hu, Emily Ellman, Gissou Azabdaftari, Elena Pop, James Mohler, Kristopher Attwood, Li Yan, Jianmin Wang, Anna Woloszynska-Read.

Roswell Park Cancer Institute, Buffalo, NY, USA.

P57

CD117 Tyrosine Kinase Activation Drives Prostate Cancer Aggressiveness

Lihong Shi, Koran Harris, Brittni Foster, Aleksander Skardal, Bethany Kerr, Ph.D.

Wake Forest School of Medicine, Winston Salem, NC, USA.

P58 - TRAVEL AWARD

Stromal-Epithelial Interactions Promote Resistance to Androgen-Targeted Therapy in the Context of Fatty Acid Oxidation

Krizia Rohena Rivera, PhD, Anisha Madhav, MS, Veronica Placencio, PhD, Frank L. Duong, BS, Neil Bhowmick, PhD.

Cedars-Sinai Medical Center, Los Angeles, CA, USA.

P59

A Temporal and Spatial Map of Axons in the Developing Mouse Prostate

Anne E. Turco[1], Mark T. Cadena[1], Helen L. Zhang[1], Richard E. Peterson, Ph.D[1], Janet R. Keast, Ph.D[2], Chad M. Vezina, Ph.D[1].

[1]University of Wisconsin Madison, Madison, WI, USA, [2]University of Melbourne, Melbourne, Australia.

P60

Andrographolide Inhibits Epithelial to Mesenchymal Transition In Prostate Cancer

Hina Mir[1], Neeraj Kapur[1], Guru Sonpavde[2], Shailesh Singh[1].

[1]Morehouse School of Medicine, Atlanta, GA, USA, [2]Dana Farber Cancer Institute, Boston, MA, USA.

P61 - TRAVEL AWARD

A Proteomic Fingerprint of Hormone-Induced Lower Urinary Tract Dysfunction in Mice

Samuel Thomas, Ling Hao, Seth Hyman, Laura Steinke, Kellen Delaney, William Ricke, Lingjun Li.

University of Wisconsin - Madison, Madison, WI, USA.

P62

Myokine Signaling Blockade Prevents Androgen Deprivation Therapy Induced Sarcopenia in a Mouse Model of Prostate Cancer

Chunliu Pan, Ph.D., Yanni Zulia, B.S., Kent L. Nastiuk, Ph.D.

Roswell Park Cancer Institute, Buffalo, NY, USA.

P63 - TRAVEL AWARD

FOXA2 Promotes Prostate Cancer Bone Colonization

Zachary M. Connelly[1], Shu Yang[1], A. Wayne Orr[1], Ranjie Jin[2], David DeGraff[3], Xiaotun Zhang[4], Colm Morrissey[4], Eva Corey[4], Robert J. Matusik[2], Xiuping Yu[1].

[1]LSUHSC Shreveport, Shreveport, LA, USA, [2]Vanderbilt University Medical Center, Nashville, TN, USA, [3]Penn State College of Medicine and Milton S. Hershey Medical Center, Hershey, PA, USA, [4]University of Washington, Seattle, WA, USA.

P64

Vitamin D Promotes Differentiation of Human Prostate Organoids in a microRNA-Dependent Manner

Tara McCray, Giovanni Lugli, PhD, Larisa Nonn, PhD.

University of Illinois, Chicago, Chicago, IL, USA.

P65 - TRAVEL AWARD

Examination Of The Small RNA Landscape In Prostate Epithelium Reveals Abundance Of PIWI-interacting RNAs and Regulation by Vitamin D

Bethany Baumann, PhD, Shang Gao, Giovanni Lugli, PhD, Zachary Richards, Larisa Nonn, PhD.

University of Illinois, Chicago, IL, USA.

P66

Collagen Architecture of Canine and Murine Prostate: Impact of Age and Androgen Deprivation

Hannah Ruetten, Kyle Wegner, Diya Joseph, Sara Colopy, Ruth Sullivan, Chad Vezina.

University of Wisconsin- Madison, Madision, WI, USA.

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Can Androgens Have Significant Effects on Mesenchymal Stem Cells

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Tracing Cell Movement in the Bladder during Regeneration after Subtotal Cystectomy

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P69 - TRAVEL AWARD

Optogenetic Stimulation of Corticotropin-releasing Hormone Expressing Neurons in Barrington's Nucleus Recapitulates the Social Stress Voiding Phenotype in Mice

Jason Van Batavia, Stephan Butler,

Eleanor Lewis, Joanna Fesi, Rita Valentino, Stephen Zderic.

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Growth Hormone-Releasing Hormone Antagonists Exert Multiple Beneficial Effects in a Rat Model of Non-Bacterial Prostatitis

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Developmental Origins of Prostate Neuroendocrine Cells

Mark T. Cadena, B.S. Biology[1], Ryan L. Trevena[1], Adam Gottschalk[1], Diya B. Joseph[1], Michelle Southard-Smith[2], Chad M. Vezina[1].

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Heterogeneity and Drug Resistance in 3D Cultures of Patient Prostate Cancer Bone Metastases and Primagrafts

Christina A.M. Jamieson, PhD, Theresa Mendoza, MS, Michelle Muldong, BS, Abigail Gallegos, BS, Christina N. Wu, PhD, Danielle Burner, William Zhu, BS, Olga Miakicheva, BS, Elana Godebu, MD, Omer Raheem, MD, Jason R. Woo, MD, Nicholas A. Cacalano, PhD, Christopher J. Kane, MD, Anna A. Kulidjian, MD, Terry Gaasterland, PhD.

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Beta-Arrestins regulate basal cell and cancer stem cell phenotype in muscle-invasive bladder cancer.

Georgios Kallifatidis, Diandra K. Smith, Jie Gao, James J. Hoy, Richard Pearce, Jiemin Li, Vinata Lokeshwar, Ph.D., Bal L. Lokeshwar, Ph.D.

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Roles For Tumor-derived Matrix Metalloproteinase 3 (MMP-3) In Prostate Cancer Growth in Bone

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Mouse Bladder is a De Novo Organ

Liguang Xia, MD, Nehizena S. Aihie, Haochuan Zhang, Xue Sean Li, PhD.

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Defining New Drivers of Castration-resistant Prostate Cancer

En-chi Hsu[1], Meghan Rice[1], Rosalie Nolley[1], Abel Bermudez[1], Jiaoti Huang[2], Donna Peehl[1], Christian Kunder[1], Sharon Pitteri[1], James Brooks[1], Tanya Stoyanova[1].

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Increased Tumor Induction by Prostate Fibroblasts from African American Men Compared To Caucasians

Marc Gillard[1], Rodrigo Javier[2], Susan Crawford, MD, PhD[2], Donal Vander Griend, PhD[1], Omar Franco, MD, PHD[2].

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The Prostatic Inflammatory Environment of the NOD/ShiltJ Mouse

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Targeting Androgen Receptor in Combination with Cisplatin: Effective Treatment Strategy for Muscle Invasive Bladder Cancer.

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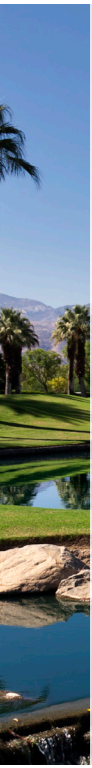

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