SBUR 2016 FALL SYMPOSIUM:

Urologic Biology: Cell Actions and Reactions in Normal and Disease Niches

November 10-13, 2016 • Scottsdale, AZ
The Scottsdale Plaza Resort

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 2016 FALL SYMPOSIUM.

Endocrine-Related Cancer

THE SOCIETY FOR BASIC UROLOGIC RESEARCH THANKS THE FOLLOWING COMPANIES FOR PROVIDING EDUCATIONAL GRANT SUPPORT FOR THE 2016 FALL SYMPOSIUM.

Astellas Pharma Global Development, Inc. Medical Affairs, Americas

Ferring Pharmaceuticals Inc.

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(NIH/NIDDK R13 Grant)*

*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 10/26/16

Welcome Colleagues and Friends!

Welcome to the Society for Basic Urologic Research Fall 2016 meeting. It's great to see all of you here in sunny Scottsdale! Cells are the fundamental units of urologic health and disease. Therefore, the focus of our annual meeting is on the cellular mechanisms that underpin our understanding of the urologic diseases and how this knowledge will translate into rational approaches towards more effective patient care.

The Symposium will commence with the keynote Leland W. K. Chung Lecture given by Dr. Jeffrey M. Rosen, Baylor College of Medicine, followed by five Plenary Sessions covering the following areas: 1) Cellular Communication in Development 2) Signaling Networks in Urologic Cells 3) Cell-Cell Dynamics within the Niche 4) Molecular Niche Communication and 5) Inflammation and Infection as Modulators of Cell/Niche Function.

Integration of basic, translational and clinical research approaches is foundational for the success of personalized medicine. Two sessions on "The Origin of the Prostate Stem Cell and Tumor Heterogeneity" and the "Advantages and Challenges of Personalized Medicine in Urology" will provide opportunities for dynamic discussions on these complex issues.

This year we are particularly excited to include a "Trainee Affairs Career Symposium" in our program. Dr. David R. Rowley, Baylor College of Medicine, will lead the discussion and Dr. Carolyn Best, AUA Director of Research, will meet with trainees one-on-one or in small groups during Saturday's poster session. This is a great opportunity, and we encourage trainees to sign up to meet with Dr. Best. She will also be available to discuss funding opportunities and grant application strategies with both new and established investigators. For our clinician-scientist attendees, the SBUR continues to offer CME credits.

I would like to thank the SBUR 2016 Fall Meeting Program Committee for their invaluable assistance in putting this program together and for the NIH NIDDK for supporting our trainees with travel awards.

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

Hari K. Koul, MSC, PhD, FASN
President

Susan Kasper, PhD
2016 Scientific Program Chair

SBUR
SOCIETY FOR BASIC UROLOGIC RESEARCH, INC.

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Table of Contents

General Information

REGISTRATION HOURS

Conference Foyer

Thursday, November 10 . . . . . 2:00 p.m. – 8:30 p.m.

Friday, November 11 . . . . . 7:00 a.m. – 6:30 p.m.

Saturday, November 12 . . . . . 7:00 a.m. – 7:00 p.m.

Sunday, November 13 . . . . . 7:00 a.m. – 12:15 p.m.

GENERAL SESSION

Grand Ballroom ABC

Thursday, November 10 . . . . . 6:45 p.m. – 8:25 p.m.

Friday, November 11 . . . . . 8:00 a.m. – 4:50 p.m.

Saturday, November 12 . . . . . 8:00 a.m. – 4:00 p.m.

Sunday, November 13 . . . . . 8:00 a.m. – 12:15 p.m.

TRAINEE AFFAIRS CAREER SYMPOSIUM

Grand Ballroom ABC

Saturday, November 12 . . . . . 6:00 p.m. – 7:00 p.m.

LUNCH

Terraza

Friday, November 11 . . . . . Noon – 1:30 p.m.

Saturday, November 12 . . . . . Noon – 1:30 p.m.

Evening Events

NETWORKING RECEPTION, AWARDS AND POSTER SESSIONS

Join old and new friends and colleagues Friday and Saturday evenings for a light reception and scientific poster session.

Grand Ballroom DEF

Poster Session I, Young Investigator Awards and Travel Awards Presentation

Friday, November 11
4:50 – 6:30 p.m.
Business Casual
Light snacks and cash bar

Grand Ballroom DEF

Poster Session II, One-on-One/Small Group Meetings for Trainees with Dr. Carolyn Best

Saturday, November 12, 2015
4:00 – 6:00 p.m.
Business Casual
Light snacks and cash bar

2016 – 2017 Committees

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Vinata B. Lokeshwar, PhD (to 2018)

Shuyuan Yeh, PhD (to 2017)

Natasha Kyprianou, PhD (to 2017)

James Brooks, MD (to 2017)

Hongwu Chen, PhD

Vinata B. Lokeshwar, PhD

Hari K. Koul, MSC, PhD, FASN (ex officio)

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Hunter Wessells, MD

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Clayton Yates, PhD

Cindy Miranti, PhD

(Charles) Chunming Guo, PhD

Sanghee Lee, PhD

Congratulations to the 2016 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.

Timothy L. Ratliff, PhD

MERITORIOUS AWARD

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

Ralph Buttyan, PhD

SWIU/SBUR AWARD FOR EXELLENCE 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.

Donna Peehl, 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.

Christopher E. Barbieri, MD, PhD
Weill Cornell Medical College

Jeremy Paul Burton, BSc, MSc, PhD
Lawson Health Research Institute

Scott A. Tomlins, MD, PhD
Michigan Center for Translational Pathology

YOUNG INVESTIGATOR AWARD NOMINEES

David J. DeGraff, PhD
Penn State Milton S. Hershey Medical Center

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

Leigh Ellis, PhD
Roswell Park Cancer Institute

Alejandro S. Godoy, BSc, MSc, PhD
Pontifical Catholic University of Chile / Roswell Park Cancer Institute

Maria Hadjifrangiskou, PhD
Vanderbilt University School of Medicine

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

Ari Hakimi, MD
Memorial Sloan Kettering Cancer Center

Tim Jenkins, PhD
University of Utah, Division of Urology

Bethany Kerr, PhD
Wake Forest School of Medicine

Hung-Ming Lam, PhD
University of Washington

Scott R. Manson, PhD
Washington University School of Medicine in St. Louis

Mohummad Minhaj Siddiqui, MD
University of Maryland School of Medicine

Douglas W. Strand, PhD
UT Southwestern Medical Center

H. David S. Ulmert, MD, PhD
Memorial Sloan Kettering Cancer 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.

Sinead Aherne, PhD
Moffitt Cancer Center, Tampa

Himanshu Arora, PhD
University of Miami

Hannah Brechka, BA
University of Chicago

Mark T. Cadena
University of Wisconsin, Madison

Justin Cotellessa
University of Massachusetts, Boston

Dibash K. Das, MA
Hunter College & CUNY Graduate Center

Sander B. Frank, PhD
University of Arizona Cancer Center

Praveen K. Jaiswal, PhD
LSU Health Science Center

Diya B. Joseph, BS
University of Wisconsin

Alan Lombard, PhD
UC Davis

Erin McAuley, BS, MS
University of Chicago

Dalton McLean
University of Wisconsin

Xiangqi Meng, PhD
Van Andel Research Institute

Petra Popovics, PhD
University of Miami

Swathi Ramakrishnan, PhD
Roswell Park Cancer Institute

Fengtian Wang
Louisiana State University Health Sciences Center

Faculty

Arturo Araujo, PhD
H. Lee Moffitt Cancer Center & Research Institute
Tampa, FL

Darius Jehan Bägli, MD
The Hospital for Sick Children
Toronto, ON

Carolyn Best, PhD
The American Urological Association
Linthicum, MD

Lori A. Birder, PhD
University of Pittsburgh
Pittsburgh, PA

Jeremy P. Burton, PhD
Lawson Health Research Institute, University of Western Onatrio
London, ON, Canada

Angelo DeMarzo, MD, PhD
Johns Hopkins Hospital
Baltimore, MD

Martin Edwin Gleave, MD
University of British Columbia
Vancouver, BC, Canada

John T. Isaacs, PhD
Johns Hopkins School of Medicine
Baltimore, MD

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

Andrew P. McMahon, PhD
Keck School of Medicine, USC
Los Angeles, CA

Andrea Morrione, PhD
Thomas Jefferson University
Philadelphia, PA

Leonard M. Neckers, PhD
Urologic Oncology Branch, National Cancer Institute
Bethesda, MD

Vijay A.K. Rathinam, DVM, PhD
UConn Health School of Medicine
Farmington, CT

Timothy L. Ratliff, PhD
Purdue University Center for Cancer Research
West Lafayette, IN

Jeffrey M. Rosen, PhD
Baylor College of Medicine
Houston, TX

David R. Rowley, PhD
Baylor College of Medicine
Houston, TX

Michael Shen, PhD
Columbia University Medical Center
New York, NY

Arun Sreekumar, PhD
Baylor College of Medicine
Houston, TX

Zijie (ZJ) Sun, PhD, MD
Beckman Research Institute/City of Hope
Duarte, CA

Dean Tang, PhD
Roswell Park Cancer Institute
Buffalo, NY

Ashleigh Theberge, PhD
University of Washington Seattle
Seattle, WA

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

Jindan Yu, PhD
Northwestern University
Chicago, IL

Named Lecturer Bios

JEFFREY M. ROSEN, PHD

Leland W.K. Chung Lecturer

Dr. Rosen is currently a Distinguished Service Professor, the Vice-Chair and the C.C. Bell Professor of Molecular & Cellular Biology and Medicine at Baylor College of Medicine. He was the recipient of two MERIT awards from the National Cancer Institute on a grant entitled, “Hormonal Regulation of Breast Cancer” currently in its fortieth year of consecutive funding. His laboratory has authored 275 publications and book chapters dealing with hormonal regulation of gene expression, signal transduction, normal mammary gland development, breast cancer, transgenic animal models of breast and prostate cancer, mammary gland stem and progenitor cells, cancer stem cells and therapeutic resistance, and noncoding RNAs. He is the PI on the CPRIT BCM Comprehensive Cancer Training Program. Dr. Rosen has trained 33 graduate students and 45 postdoctoral fellows, many of whom are now faculty at major academic institutions in the USA and abroad.

MARTIN EDWIN GLEAVE, MD

International Lecturer

Dr. Gleave is a Distinguished Professor and Chairman of the Department of Urologic Sciences at UBC, and a British Columbia Leadership Chair. He is Director of the Vancouver Prostate Centre, now a UBC and National Centre of Excellence. He has published over 450 papers with >27,000 citations and an H-Index of 92, with >$90M in research funding. Dr. Gleave's research characterizes molecular mechanisms mediating treatment resistance in cancer, focusing on adaptive survival responses that drive acquired treatment resistance, and design of rational co-targeting strategies to create conditional lethality to improve cancer control. He generated in vivo models that mimic the course of castrate resistance that are now used worldwide for mechanistic and preclinical proof-of principle studies. He defined adaptive stress responses that promote treatment-resistance involving chaperone proteins clusterin (CLU) and Hsp27, and founded OncoGenex Pharmaceuticals to develop their inhibitors (OGX-011 and OGX-427) to potentiate anti-cancer therapies. After being the first to demonstrate significant target inhibition using an antisense drug (OGX-011) in humans, he helped lead a randomized trial that demonstrated a 7-month gain in overall survival when OGX-011 was combined with docetaxel. 3 global Phase III registration trials with OGX-011 have been completed. He also discovered and developed OGX-427, another drug targeting Hsp27 that, for the first time, demonstrated single agent activity in Phase II studies of castrate resistant prostate cancer and bladder cancer.

JEREMY P. BURTON, PHD

AUA Lecturer

Dr. Burton is currently the Miriam Burnett Chair in Urological Sciences, appointed as an Assistant Professor in the Department of Surgery (Division of Urology) / Microbiology and Immunology (Western University) and serves as the Deputy Director of the Canadian Centre for Human Microbiome and Probiotics (CCHMP). The current main focus of Dr. Burton's research program is to study the microbiota in urological diseases of increasing economic burden such as urolithiasis, kidney transplant, urological cancers and to see how probiotics, microbiome therapeutics and diet may be able to improve such conditions. He received his PhD at the University of Otago, Dunedin, New Zealand and the Nestle Research Centre, Switzerland (2000) and completed his postdoctoral fellowship at The University of Western Ontario, Canada (2001-2003), which was then followed by working in biotechnology start up companies. The impact of his work has resulted in nearly 100 peer-reviewed articles and book chapters published, mostly these are associated with probiotics and microbiome. He was the first to describe the urogenital tract microbiota by non-culture based techniques. These studies showed that Lactobacillus iners was the major constituent of the human vaginal microbiome and Atopobium vaginae was a pathogen in bacterial vaginosis. This work has changed the perception relating to the simplicity of the vaginal microbiota, its composition and its importance to women's health. Recently, their papers have gained recognition, including relating to sub therapeutic dosing of antimicrobials of urinary tract infections, published in the American Society for Microbiology's open access journal, mBIO and was featured in Nature Research Highlights. Additionally, their recent review in Nature Reviews Urology is frequently read and cited.

Scientific Program

Travel Award

Urologic Biology: Cell Actions and Reactions in Normal and Disease Niches

General Sessions will be located in Grand Ballroom ABC

THURSDAY, NOVEMBER 10, 2016

2:00 – 8:30 p.m. **Registration**

Grand Ballroom Conference Foyer

3:30 – 6:00 p.m. **Executive Committee Meeting**

6:45 – 7:00 p.m. **Welcome & Introductory Remarks**

Hari K. Koul, MSC, PhD, FASN
SBUR President
LSU Health Sciences Center, Department of Biochemistry and Molecular Biology and Feist Weiller Cancer Center
Shreveport, LA

7:00 – 8:25 p.m. **Keynote Session: Perspectives on Cell-Niche Interactions**

Discussion Leaders:

Susan Kasper, PhD
University of Cincinnati
Cincinnati, OH

Cindy Miranti, PhD
Van Andel Research Institute
Grand Rapids, MI

7:00 – 8:00 p.m. **Leland W.K. Chung Lecture**

Developmental Insights into Breast Cancer Intratumoral Heterogeneity

Jeffrey M. Rosen, PhD
Baylor College of Medicine
Houston, TX

Breast cancer is no longer considered a single disease, but instead is made up of multiple subtypes with genetically and most likely epigenetically heterogeneous tumors composed of numerous clones. Both the hierarchical cancer stem cell and clonal evolution models have been invoked to help explain this intratumoral heterogeneity. Several recent studies have helped define the functional interactions among the different cellular subpopulations necessary for the evolution of this complex ecosystem. These interactions involve paracrine interactions that include locally acting Wnt family members, reminiscent of the signaling pathways important for normal mammary gland development and stem cell self-renewal. A better understanding

of these interactions, especially in the metastatic setting, will be important for the development of improved combinatorial therapies designed to prevent relapse and to ultimately decrease mortality.

8:00 – 8:15 p.m. **Discussion**

8:15 – 8:25 p.m. **Meritorious Achievement Award**

Ralph Buttyan, PhD
Vancouver Prostate Centre
Vancouver, BC

8:25 – 9:25 p.m. **Welcome Reception**

FRIDAY, NOVEMBER 11, 2016

Breakfast on Own

8:00 a.m. – Noon

Plenary Session I: Cellular Communication in Development

Discussion Leaders:

X. Sean Li, PhD
Boston Children's Hospital
Boston, MA

Rosalyn Adam, PhD
Boston Children's Hospital
Boston, MA

8:00 – 8:25 a.m. **Regulation of Nephron Progenitors during Kidney Organogenesis**

Andrew P. McMahon, PhD
Keck School of Medicine, USC
Los Angeles, CA

The mammalian kidney assembles from a mobile niche during a lengthy period of fetal development. Cellular, genetic and molecular studies in the mouse have provided a molecular framework of key signaling pathways and transcriptional programs at play in maintaining progenitor populations and regulating their commitment to mature kidney cell types. This raises the question of how similar are these events between mouse and man? And, how can knowledge be applied to generating new kidney structures. I will discuss recent findings in our analysis of the mouse and human kidney in the light of recent reports of pluripotent stem cell derived kidney organoid cultures.

8:25 – 8:40 a.m. **Discussion**

8:40 – 9:05 a.m. **Transcription Factors that Regulate Organ-Specific Stem Cells in Development and Cancer**

X. Sean Li, PhD
Boston Children's Hospital
Boston, MA

One of the most consistent but poorly explained findings of cancer epidemiological studies is that incidence and mortality of the sex unspecific cancers including bladder cancer are higher among men. We will discuss evidence suggesting that females with an extra copy of X-chromosome are much less likely to develop bladder cancer in part due to the epigenetic regulatory mechanism. Understanding gender disparities in cancer incidence and mortality may identifed new risk factors to develop innovative prevention and treatment strategies.

9:05 – 9:20 a.m. **Discussion**

9:20 – 9:45 a.m. **Wnt/AR Signaling in Prostatic Development and Tumorigenesis**

Zijie (ZJ) Sun, PhD
Beckman Research Institute/City of Hope
Duarte, CA

We demonstrate a promotional role of the androgen receptor in prostate tumor initiation using a new and relevant mouse model. The androgen signaling pathway can also cooperate and enhance beta-catenin mediated oncogenic transformation in the prostate. These results provide new respects regarding roles of androgen and Wnt signaling in prostate development and tumorigenesis.

9:45 – 10:00 a.m. **Discussion**

10:00 – 10:20 a.m. **Break**

10:20 – 10:45 a.m. **Determining the Cell Type of Origin and the Molecular Mechanisms Involved in Neoplastic Transformation in the Prostate**

Angelo DeMarzo, MD, PhD
Johns Hopkins Hospital
Baltimore, MD

The cellular origin of human prostate cancer has remained somewhat elusive. This talk will focus on somatic genetic, epigenetic and expression-based molecular alterations occurring during the formation and progression of human prostate cancer. Arguments will be presented for a luminal cell phenotype as the cell that manifests the molecular alterations leading to carcinoma and that intermediate cells enriched in regions of proliferative inflammatory atrophy may represent an increased pool of 'at risk' cells for cancer initiation. The data used over the years to suggest that human high grade prostatic intraepithelial neoplasia (PIN) is the main precursor to prostatic adenocarcinoma will be discussed, as well as, recent molecular approaches to understand the "order of events" during early prostate cancer formation. Also, recent data will be presented showing that some lesions considered PIN morphologically may actually represent retrograde colonization of benign glands by invasive adenocarcinoma cells.

10:45 – 11:00 a.m. **Discussion**

11:00 – 11:10 a.m. **Travel Award Winner**

Loss of TßRII in Osteoblasts Promotes Prostate Cancer Bone Metastasis Through bFGF

Xiangqi (Neil) Meng, PhD
Van Andel Research Institute
Grand Rapids, MI

11:10 – 11:20 a.m. **Discussion**

11:20 – Noon **Debate: Advantages and Challenges of Personalized Medicine in Urology**

Moderator:

Ganesh V. Raj, MD, PhD
UT Southwestern Medical Center
Dallas, TX

Panel:

Linda Alford Baker, MD
UT Southwestern
Dallas, TX

Scott A. Tomlins, MD, PhD
University of Michigan School of Medicine
Ann Arbor, MI

Marja Nevalainen, MD, PhD
Medical College of Wisconsin Cancer Center
Milwaukee, WI

John Krolewski, MD, PhD
Roswell Park Cancer Institute
Buffalo, NY

Noon – 1:30 p.m. **Lunch**

1:30 – 4:30 p.m. **Plenary Session II: Signaling Networks in Urologic Cells**

Discussion Leaders:

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

Ming Lam, PhD
University of Washington Seattle
Seattle, WA

1:30 – 1:55 p.m. **Molecular Chaperone Dependence of Androgen Receptor Splice Variants in CRPC**

Leonard M. Neckers, PhD
Urologic Oncology Branch, National Cancer Institute
Bethesda, MD

Although initially effective in treating prostate cancer, ligand binding domain (LBD)-targeted therapies (e.g,, enzalutamide or abiraterone) almost invariably lead to emergence of castration-resistant prostate cancer (CRPC), an incurable condition commonly mediated by re-activation of the androgen receptor (AR) signaling axis through a variety of mechanisms and characterized by an alternative, androgen-independent but AR-driven transcriptional program. CRPC is frequently associated with upregulated expression of AR splice variants (ARv), including but not limited to ARv7, which lack the LBD. Consequently, these ARv are insensitive to anti-androgens and are constitutively active. Nuclear receptors, including AR, normally require the activity of molecular chaperones, including Hsp40, Hsp70 and Hsp90, to prevent protein aggregation and to ensure proper folding. Unlike Hsp90, which binds to the AR LBD and does not interact with ARv, we've shown that Hsp40 and Hsp70 bind to ARv and that ARv stability and transcriptional activity remain dependent on these chaperones. We provide proof of concept data showing that pharmacologic inhibition of either Hsp40 or Hsp70 may provide a novel strategy to treat CRPC.

1:55 – 2:10 p.m. **Discussion**

2:10 – 2:35 p.m. **Niche-dependent Roles of FOXA1 in Prostate Cancer**

Jindan Yu, PhD
Northwestern University
Chicago, IL

We will report niche-dependent roles of FOXA1 in prostate cancer cells. In the presence of androgen, FOXA1 defines prostate-specific AR cistrome, promotes AR signaling, and induces cell proliferation. However, under androgen-deprived conditions, FOXA1 dilutes nuclear AR and suppresses AR signaling. FOXA1 loss leads to androgen-independent AR activation, EMT, cell invasion, and prostate cancer neuroendocrine differentiation. We will delineate how FOXA1 regulates AR and AR-independent pathways and provide guidance to FOXA1-targeted therapies.

2:35 – 2:50 p.m. **Discussion**

2:50 – 3:10 p.m. **Break**

3:10 – 3:35 p.m. **Stromal Cell Plasticity and Reprogramming in Urologic Diseases**

David R. Rowley, PhD
Baylor College of Medicine
Houston, TX

Tumor growth and evolution is affected by interactions with reactive stroma. Carcinoma-associated fibroblasts and myofibroblasts are

recruited from mesenchymal stem cells. Many factors modulate mesenchymal stem cell biology and formation of differential reactive stroma phenotypes. These phenotypes are variable and affect key aspects of tumor biology including the immune landscape, extracellular matrix, and angiogenesis. Accordingly, understanding mechanisms of mesenchymal stem cell activation, recruitment and biology is important for developing new targets of opportunity in prostate cancer therapeutics.

3:35 – 3:50 p.m. **Discussion**

3:50 – 4:15 p.m. **Mathematical and Computational Modeling of Tumor Cell/Bone Microenvironment Interactions**

Arturo Araujo, PhD
H. Lee Moffitt Cancer Center & Research Institute
Tampa, FL

Integrating computational modeling with biological experimentation is a powerful means with which to rapidly assess the efficacy of therapeutic strategies on incurable bone metastatic prostate cancer. For this, we developed a Hybrid-Discreet Cellular Automata model where the interactions between key cell types and their role on the evolutionary dynamics of the tumor microenvironment can be studied. Our in silico results predict that there therapies can impact prostate cancer cell viability directly but also by restricting nutrient availability and interfering with the differentiation and maturation of recruited microenvironmental cells; results that in turn were validated in vivo. We demonstrate how this clinically relevant model can be used to predict the evolution of heterogeneous bone metastases in response to therapeutic targets.

4:15 – 4:30 p.m. **Discussion**

4:30 – 4:50 p.m. **Presentation of 2016 Young Investigator Awards**

4:50 – 6:30 p.m. **Poster Session #1**

 # P1 – P53

6:15 – 6:30 p.m. **Presentation of 2016 Travel Awards**

6:30 p.m. **Evening on Own**

SATURDAY, NOVEMBER 12, 2016

Breakfast on Own

8:00 a.m. – Noon

Plenary Session III: Cell-Cell Dynamics within the Niche

Discussion Leaders:

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

Susan Kasper, PhD
University of Cincinnati
Cincinnati, OH

8:00 – 8:25 a.m. **Progenitor Cells and the Origin of Prostate Cancer**

Michael Shen, PhD
Columbia University Medical Center
New York, NY

Elucidation of the epithelial lineage hierarchy in the prostate epithelium is of central importance for understanding the cell of origin for prostate cancer and for the molecular analysis of cellular differentiation in the normal and transformed prostate. The Shen laboratory has been investigating progenitor cells and lineage specification during prostate organogenesis and regeneration using lineage-tracing approaches in genetically-engineered mice as well as organoid culture models. I will present recent findings on the specification and differentiation of prostate epithelial cell types, and their implications for prostate tumor biology.

8:25 – 8:50 a.m. **The Cellular Basis and Molecular Determinants of Prostate Cancer Cell Heterogeniety**

Dean Tang, PhD
Roswell Park Cancer Institute
Buffalo, NY

We have been systematically studying prostate cancer cell heterogeneity since 2003 and our studies indicate that prostate cancer cell heterogeneity, e.g., with respect to AR expression, greatly influences tumor response to current stand-of-care therapies. Based on our results, a clinical trial is ongoing testing the idea that simultaneous targeting of AR+ (differentiated) and undifferentiated prostate cancer cells is expected to delay the emergence of CRPC and extend patients' survival.

8:50 – 9:20 a.m. **Discussion: The Origin of the Prostate Stem Cell and Tumor Heterogeneity**

9:20 – 9:45 a.m. **Bone Marrow Derived Mesenchymal Stem Cells as "Trojan Horses" in Drug Delivery**

John T. Isaacs, PhD
Johns Hopkins School of Medicine
Baltimore, MD

The recruitment of mesenchymal stem cells (MSCs) from the bone marrow to sites of prostate cancer is an ongoing process continuing throughout disease progression as documented by their presence in lesions from metastatic castration-resistant prostate cancer (mCRPC) patients at the time of death. This influx of MSCs from systemic circulation provides the rationale for their use as a cell-based vector to deliver therapeutic agents, which is the subject of an ongoing Phase 0 clinical trial. Additionally, prostate tissue from a subset of primary prostate cancer patients is highly enriched in MSCs, suggesting their enumeration may have prognostic value for identifying men with aggressive disease.

9:45 – 10:00 a.m. Discussion

10:00 – 10:20 a.m. Break

10:20 – 10:45 a.m. Urothelial Cell Signaling to Neighboring Neuronal Cells via Release of Nitric Oxide (NO) and ATP

Lori A. Birder, PhD
University of Pittsburgh
Pittsburgh, PA

The urothelium exhibits a number of 'neuron-like' properties including both a 'sensor' and 'transducer' function. The release of chemical mediators such as NO and ATP from urothelial cells suggests that these cells exhibit specialized sensory and signaling properties that could allow reciprocal communication with neighboring cells including bladder nerves. Further, the 'sensor', 'transducer' and 'barrier' properties of bladder urothelium are altered in a number of bladder pathologies. Taken together, findings have supported the view that urothelial cells receive and integrate multiple stimuli thus providing a link in the transfer of information from the bladder to the nervous system.

10:45 – 11:00 a.m. Discussion

11:00 – 11:25 a.m. Progranulin Action in Bladder Cancer

Andrea Morrione, PhD
Thomas Jefferson University
Philadelphia, PA

Bladder cancer is a major epidemiological issue in the United States with 74,000 estimated new cases and 16,000 estimated deaths in 2015. Compared to other cancers, bladder cancer has the highest cost per patient in the US due to disease prevalence and costs associated with long-term monitoring. We have recently demonstrated a critical role for progranulin in bladder cancer. Progranulin contributes, as an autocrine growth factor, to the transformed phenotype by modulating

Akt-and MAPK-driven motility, invasion and anchorage-independent growth. In addition, progranulin is critical for tumor growth in vivo, in both xenograft and orthotopic tumor models. Progranulin may constitute a novel target for therapeutic intervention in bladder tumors. In addition, progranulin may serve as a novel biomarker for bladder cancer.

11:25 – 11:40 a.m. **Discussion**

11:40 – 11:50 a.m.  **Travel Award Winner**

Spatial Mapping of Luminal Cell Expansion During Prostate Growth and Regeneration Using a Fluorescent Lineage Reporter

Diya B. Joseph, BS
University of Wisconsin
Madison, WI

11:50 a.m. – Noon **Discussion**

Noon – 1:30 p.m. **Lunch**

1:30 – 4:00 p.m. **Plenary Session IV: Molecular Niche Communication**

Discussion Leaders:

Conor Lynch, PhD
H. Lee Moffitt Cancer Center and Research Institute
Tampa, FL

Allen Gao, MD, PhD
UC Davis Medical Center
Sacramento, CA

1:30 – 1:55 p.m. **The Bladder Smooth Muscle-Extracellular Matrix Niche: A Potential Gateway to Epigenetic Regulation of Bladder Obstruction**

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

Obstructive myopathy and dysfunction in the bladder create considerable clinical sequelae, yet the mechanisms underlying persistence of this pathology despite medical or surgical alleviation of the primary obstructing stimulus (e.g., prostate overgrowth or urethral valves) remain elusive. Epigenetic machinery provides one mechanistic avenue through which the abnormal mechanical milieu associated with obstruction may persistently alter gene expression and organ function. The observation that the aberrant extracellular matrix that characterizes bladder wall hypertrophy in obstruction may reciprocally and actively drive epigenetic responses in smooth muscle has invited further exploration of the role of epigenetic machinery in this disease process. Our recent observations now suggest a role for methylation in in vivo bladder obstruction both dysfunctionally and in

dysregulation of gene expression.

1:55 – 2:10 p.m. **Discussion**

2:10 – 2:20 p.m. **AUA Research: Creating Opportunities for Basic Science in Urology**

Carolyn Best, PhD
AUA Director of Research
Linthicum, MD

Basic urologic research, while contributing many exciting advances that ultimately result in improvements in urology patient care, is under-resourced in many regards. To address the diversity of these needs, the American Urological Association (AUA) Research Department, through advocacy, education, and funding, creates many opportunities for basic scientists to enhance their urologic research and careers. Through its partnership with the SBUR and its members, every year the AUA Research Department 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. Through its established programs and new initiatives that address a breadth of urologic research needs, the AUA Research Department strives toward the goal of reducing the burden of urologic disease through impactful research.

2:20 – 2:40 p.m. **Break**

2:40 – 3:05 p.m. **The Hexosamine Biosynthetic Pathway in Driving Castration Resistant Prostate Cancer**

Arun Sreekumar, PhD
Baylor College of Medicine
Houston, TX

There is limited mechanistic understanding of the development and maintenance of the castrate resistant state. This knowledge gap is reflected in the lack of effective predictive markers and durable therapeutic strategies. Earlier studies from our lab integrating data from metabolomics, proteomics and gene expression compartments in both tissues and cell lines have revealed a key role for amino acid metabolism in the development of AD PCa [14, 15], potentially driven by AR signaling [2, 16]. In contrast, similar data in CRPC tissues and cell lines indicate a switch in metabolism from an amino acid-driven anabolic state to a carbohydrate and fatty acid driven bioenergetics condition. Using a novel bioinformatics-based integromics approach we have combined matched PCa-derived metabolome and transcriptome data to identify a regulatory role for the Hexose Biosynthetic Pathway (HBP) in promoting the metabolic switch between an AD and CRPC state. In androgen dependent disease, this involves mutual up regulation of HBP and AR to support anabolic processes that in turn promote tumor proliferation. In contrast, HBP down regulation in CRPC promotes transcriptional

activities that support the invasive potential of the tumor. Importantly, very little is known about how HBP regulates this metabolic switch and how HBP is regulated at the molecular level in the CRPC state. Notably, a novel therapeutic translation of our findings is centered on developing metabolic therapy in CRPC based upon reactivating the HBP. We have observed that such an approach appears to sensitize CRPC-like cells to anti-androgens like enzalutamide. Although still a preliminary finding, our results introduce the innovative concept of using metabolites to overcome therapeutic resistance in advanced tumors via the exploitation of altered metabolic pathways.

3:05 – 3:20 p.m. **Discussion**

3:20 – 3:45 p.m. **International Lecture**

Targeting the Adaptive Molecular Landscape of Castration-Resistant Prostate Cancer

Martin Edwin Gleave, MD
University of British Columbia
Vancouver, BC, Canada

Castration and androgen receptor (AR) pathway inhibitors induce profound and sustained responses in advanced prostate cancer. However, the inevitable recurrence is associated with reactivation of the AR and progression to a more aggressive phenotype termed castration-resistant prostate cancer (CRPC). AR reactivation can occur directly through genomic modification of the AR gene, indirectly via co-factor and co-chaperone deregulation, and supported by stress-driven induction of a myriad of overlapping cellular survival pathways. Direct targeting of the AR outside the androgen binding site offers new promises for prolonging control; however functional redundancy and heterogeneity of CRPC mean that targeting of a singular pathway will likely escape control. In this presentation, I will review recent successes and failures of therapeutic strategies designed to target AR reactivation and adaptive survival pathways, and how emerging genomic biomarkers may help better segment the population to direct more individualized therapeutic strategies.

3:45 – 4:00 p.m. **Discussion**

4:00 – 6:00 p.m. **Poster Session II**

 # P54 – P107

4:00 – 6:00 p.m. **One-on-One/Small Group Meetings with Trainees**

Bring Your Questions Here: Where's the Money? How Do I Get It? Can Anyone Help Me? and Other Good Ones

Carolyn Best, PhD
Director of Research, American Urological Association
Linthicum, MD

6:00 – 7:00 p.m. Trainee Affairs Career Symposium

6:00 – 6:05 p.m. Introduction

Shawn E. Lupold, PhD
Johns Hopkins University, Department of Urology
Baltimore, MD

6:05 – 6:30 p.m. Preparing for Exploration beyond Postdoctoral Training

David R. Rowley, PhD
Baylor College of Medicine
Houston, TX

6:30 – 7:00 p.m. Open Panel Discussion

Panel:

Neil Bhowmick, PhD

X. Sean Li, PhD

Shawn E. Lupold, PhD

David R. Rowley, PhD

Donald Vander Griend, PhD

Optional: Trainee Affairs Group Dinner to follow at J.D.'s Lounge

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

SUNDAY, NOVEMBER 13, 2016

Breakfast on Own

8:00 a.m. – 12:15 p.m.

Plenary Session V: Inflammation and Infection as Modulators of Cell/Niche Function

Discussion Leaders:

Ganesh V. Raj, MD, PhD
University of Texas Southwestern Medical Center
Dallas, TX

Timothy L. Ratliff, PhD
Purdue University Center for Cancer Research (TENTATIVE)
West Lafayette, IN

8:00 – 8:25 a.m. Inflammasomes: Linking Infection and Inflammation

Vijay A.K. Rathinam, DVM, PhD
UConn Health School of Medicine
Farmington, CT

Intracellular transport and sensing of lipopolysaccharides leading to inflammasome activation during infections will be presented.

8:25 – 8:40 a.m. Discussion

8:40 – 9:05 a.m. **Role of Interleukin-17-Mediated Inflammation in Cancer Initiation, Promotion and Progression**

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

Dr. Zongbing You's laboratory primarily focuses on how inflammation promotes prostate cancer initiation and progression. His laboratory originally discovered the association of interleukin-17 with cancer cell survival and castration-resistance in human prostate cancer. His work revealed the role of interleukin-17 in development of hormone-naive and castration-resistant prostate cancer in a Pten-null mouse model. Dr. You's laboratory has demonstrated how interleukin-17 acts through matrix metalloproteinase-7 to induce epithelial-to-mesenchymal transition in prostate carcinogenesis.

9:05 – 9:20 a.m. **Discussion**

9:20 – 9:45 a.m. **Characterization of Autoimmune Inflammation Induced Prostate Stem Cell Expansion**

Timothy L. Ratliff, PhD
Purdue University Center for Cancer Research
West Lafayette, IN

9:45 – 10:00 a.m. **Discussion**

10:00 – 10:20 a.m. **Break**

10:20 – 10:30 a.m.  **Travel Award**

Growth Hormone-Releasing Hormone (GHRH) Antagonists Inhibit Inflammation-induced Prostate Enlargement in Mice and Reduce the Proliferation of Prostate Epithelial Cells in Vitro

Petra Popovics, PhD
University of Miami
Miami, FL

10:30 – 10:40 a.m. **Discussion**

10:40 – 11:05 a.m. **Examining Cellular Signaling in Complex Environments with Microscale Systems**

Ashleigh Theberge, PhD
University of Washington
Seattle, WA

We have designed novel microfluidic multiculture platforms to probe the effects of cell-microenvironment interactions in prostate cancer and benign prostatic hyperplasia (BPH) disease progression. Our systems are disconnectable and reconfigurable, enabling culture of multiple cell types with precisely controlled co- and multiculture exposure times. As a proof of concept, we used this system to study

the effects of macrophages on angiogenesis (blood vessel formation), utilizing macrophages that were preconditioned with prostate cancer and benign prostate epithelial cells. Importantly our microfluidic platforms utilize ten- to one hundred-fold fewer cells than conventional systems, which will enable future investigations with limited cell samples from prostate cancer and BPH patients.

11:05 – 11:20 a.m. Discussion

11:20 - 11:45 a.m. AUA Lecture

The Potential Role of the Microbiome in Urology

Jeremy P. Burton, PhD
Lawson Health Research Institute, University of Western Ontario
London, ON, Canada

The human microbiome at different body sites is already known to play a significant role in aspects of urological health. Bacteria are now being described in humans, even at sites once considered sterile including the bladder. To determine if there were bacteria routinely present at sites in the urinary system above the bladder, we conducted microbiome analysis of ESWL stone patient's urine before and after treatment. In addition, kidney specimens were obtained from 56 patients undergoing radical nephrectomy and analyzed for the presence of bacteria by quantitative PCR, 16S rRNA gene sequencing and bacterial culture. Analysis of midstream urine from ESWL patients changed considerably immediately after treatment, we were unable to determine whether the bacteria detected were originating from the kidney or elsewhere in the urinary system. After taking considerable care to mitigate for the potential DNA contamination during surgical removal of tissue, and within the microbiome processing pipeline itself, it appeared that most samples tested had detectable bacteria present within the kidneys. We are unsure as to the origins of bacteria present in the kidney, but are most likely acquired from ascension through the urinary tract and/or via systemic sources. The results of these studies raise further questions regarding the potential role bacteria play in the urinary system. We are only just starting to comprehend the role the microbiome has and the implications for urological health.

11:45 a.m. - 12:00 p.m.
Discussion

12:00 - 12:15 p.m. Farewell

Hari K. Koul, MSC, PhD, FASN

SBUR President

To claim CME credits, an email with a link and instructions will be sent to you at the conclusion of the meeting. Thank you for attending!

Travel Awardees

Podium Presentations

Full Abstracts

FRIDAY, NOVEMBER 11, 2016 11:00 – 11:10 A.M.

Loss Of TßRII In Osteoblasts Promotes Prostate Cancer Bone Metastasis Through bFGF

Xiangqi Meng, PhD, Paul Daft, PhD, Alexandra Vander Ark, Jie Wang, Zachary Madaj, Galen Hostetter, Xiaohong Li

Van Andel Research Institute, Grand Rapids, MI, USA

Background: Seventy percent of patients who die of prostate cancer (PCa) have bone metastases. Little is known about osteoblast or osteoclast specific transforming growth factor-ß (TGF-ß) signaling in PCa bone metastasis. TGF-ß signaling occurs through ligands binding the TGF-ß type II receptor (TßRII), however, the expression of TßRII in bone cells of PCa bone metastatic tissues is not clear.

Methods: To study the role of bone cell specific TGF-ß signaling, $Tgfbr2^{Col1CreERT}$ knockout (KO) or $Tgfbr2^{LysMCre}$ KO mice, which have deletion of the TßRII gene, TGFBR2, specifically in osteoblasts or osteoclasts were used. PCa cells (PC3 or DU145 cells) were intratibially injected into the $Tgfbr2^{Flox}$ or the respective KO littermates. Bone lesion development was imaged by X-ray, and lesion areas were measured and quantified using Metmorph software. Cytokine array, in vivo rescue studies, and in vitro co-culture studies were used to identify the druggable mediator. Immunohistochemistry was used to profile TßRII expression in PCa patient bone metastasis tissue microarray.

Results: We found that PCa-induced bone lesion development was increased in the $Tgfbr2^{Col1CreERT}$ KO mice, but decreased in the $Tgfbr2^{LysMCre}$ KO mice, in relative to their respective $Tgfbr2^{Flox}$ littermates. In patient bone metastatic tissues, TßRII expression was frequently lost in the cancer-associated osteoblasts (CAOBs). Therefore, we focused on the $Tgfbr2^{Col1CreERT}$ KO mouse model, where the loss of TßRII is in osteoblasts. The increased bone lesions were associated with increased tumor cell proliferation, angiogenesis, cancer associated fibroblasts (CAFs) formation, and osteoclaststogenesis. bFGF was found up-regulated in CAOBs and mediated the increased PC3 bone lesions in $Tgfbr2^{Col1CreERT}$ KO mice relative to the $Tgfbr2^{Flox}$ mice. Functionally, bFGF mediates PC3-induced bone lesion development through indirect stimulation of tumor cell proliferation, direct promotion of osteoclastogenesis and CAFs formation, as well as through direct inhibition of osteoblastogenesis. Furthermore, we found the increased bFGF was associated with increases of parathyroid hormone receptor (PTH1R) and pCREB (transcription factor of the PKA pathway that PTH1R activates) in PC3 cells co-cultured with Tgfbr2 KO osteoblasts relative to $Tgfbr2^{Flox}$ osteoblasts.

Conclusions: TGF-ß signaling in osteoblasts inhibits, but in osteoclasts promotes PCa bone lesions. TßRII was frequently lost in CAOBs in PCa patient bone metastastic tissues. Loss of TGF-ß signaling in osteoblasts mediates bFGF upregulation, which is at least partly due to the activation of increased PTH1R in CAOBs, leading to promotion of PCa bone metastasis.

Spatial Mapping Of Luminal Cell Expansion During Prostate Growth And Regeneration Using A Fluorescent Lineage Reporter

Diya B. Joseph, BS, Ryan Trevena, BS, Anoop Chandrashekar, Lisa L. Abler, PhD, Chad M. Vezina, PhD

University of Wisconsin-Madison, Madison, WI, USA

Background: The prostate develops through budding from the fetal urogenital sinus and continues through stages of branching and canalization to form a pseudo-stratified epithelium comprised of luminal, basal and neuroendocrine cells. Genetic lineage tracing strategies using the luminal specific PSA-creErt2 driver have demonstrated that luminal cells can survive androgen deprivation and expand following testosterone supplementation. The work presented here extends these studies by using the luminal specific Pbsn-cre/Esr1 driver to develop a spatial map of expanding luminal cells in the prostate spanning the urethra proximal stem cell niche to the distal region.

Methods: The expression of the inducible Pbsn-cre/Esr1 luminal specific cre driver was induced in young adult mice at 4 weeks of age or in mature adult mice at 10 weeks of age by tamoxifen administration. Activation of the cre recombinase resulted in recombination at the R26R-EYFP allele leading to the expression of the EYFP protein in prostate luminal epithelial cells and their progeny. The multi-color R26R-Confetti reporter, which stochastically turns on expression of GFP, EYFP, RFP or CFP in a cre activated cell, was also used to distinguish between cell clusters originating from individual cells. Whole prostate sections were labeled by immunofluorescence and scanned using confocal microscopy.

Results: Prostates from 4-week tamoxifen dosed mice analyzed at 6 weeks and 8 weeks showed expansion of lineage labeled cells during pubertal growth, with similar frequency of fluorescent cell clusters in the proximal, medial and distal regions. Tamoxifen activation in 10 week old adult mice resulted in mosaic labeling of single prostate luminal epithelial cells. Several of these cells persisted after castration indicating that a fraction of the cells initially labeled by tamoxifen induction are castration resistant. Implantation of a testosterone capsule resulted in the expansion of single cells to doublets and triplets, with an increased frequency of labeled cell clusters at the distal portion of prostate ducts.

Conclusions: Multiple individual luminal cells contribute to prostate growth during puberty. In the adult, castration resistant luminal epithelial cells at the distal portion of ducts possess the ability to expand during prostate regeneration. Visualization of clonally expanding cells during prostate development and regeneration can contribute to our understanding of prostate biology by revealing uni-potent or bi-potent progenitors that might play a role in prostate cancer or benign hyperplasia.

Growth Hormone-Releasing Hormone (GHRH) Antagonists Inhibit Inflammation-induced Prostate Enlargement In Mice And Reduce The Proliferation Of Prostate Epithelial Cells In Vitro

Petra Popovics, PhD

University of Miami Miller School of Medicine, Miami, FL, USA

Background: The etiology of benign prostatic hyperplasia (BPH) is unclear, however, both chronic inflammation and epithelial-to-mesenchymal transition (EMT) have been suggested as contributing factors. We have shown, that GHRH, a hypothalamic peptide hormone, and its receptor are expressed in experimental models of BPH, in which antagonists of GHRH suppressed the levels of proinflammatory cytokines and altered the expression of genes related to EMT. These findings imply a role of GHRH in the development of prostatic inflammation, however, this has not been investigated to date.

Methods: Autoimmune prostatitis in Balb/c mice was induced by subcutaneous injections of rat prostate, seminal vesicle and coagulating gland tissue homogenate in Freund`s adjuvant. Prostate volume was measured with the VEVO® 1100 imaging system. Human BPH-1 prostatic cell line was used to generate matrigel-embedded 3D cultures in which average sphere diameters were evaluated. Inflammatory environment was modelled by treating cells with THP-1 macrophage-conditioned medium, whereas EMT was triggered with TGF-ß1 or TGF-ß2 peptides. The role of autocrine/paracrine GHRH in inflammation-induced proliferation and its systemic effect in vivo was determined by using specific GHRH antagonists developed in our lab. Expression of GHRH and EMT markers were detected by quantitative RT-PCR.

Results: During the 8-week induction of chronic prostatitis in Balb/c mice, we detected a progressive increase in prostate volume reaching 92% at week 8 compared to control. This time-point was chosen for the initiation of further treatments. A 1-month daily treatment with GHRH antagonists caused a significant, 48% reduction in prostate volume, whereas prostate enlargement in vehicle treated mice progressed further. In vitro, macrophage-conditioned medium induced a 26% increase ($P<0.001$) in the average diameter of cells and stimulated the expression of mesenchymal markers. The expression of GHRH was also increased 2.7-fold ($p<0.001$). GHRH antagonist reduced inflammation- and TGF-ß2-induced increase in diameter by 64% ($P<0.01$) and by 67% ($P<0.001$), respectively.

Conclusions: This study identifies GHRH as an important factor in prostatic inflammation and EMT and suggests the merit of further investigation to elucidate the effects of GHRH antagonists in chronic prostatitis and BPH.

Poster Session I – Friday Evening

View Full Abstracts at www.SBUR.org

Friday, November 11, 2016, 4:50 – 6:30 p.m.

Grand Ballroom DEF

P1-P53

P: 1

Androgen Receptor Co-Activates GLI Transcription by Stabilizing Transcriptionally-Active GLI Proteins

Na Li, PhD, Sarah Truong, MS Student, Mannan Nouri, PhD Student, **Ralph Buttyan.**

The Vancouver Prostate Centre, Vancouver, BC, Canada

P: 2

Interference with Gli-AR Binding Alters the AR Transcriptome and Sensitizes Androgen Growth-Independent Prostate Cancer Cells to Enzalutamide Treatment

Na Li, PhD, Sarah Truong, V6H 3Z6, MS Student, Mannan Nouri, V6H 3Z6, **Ralph Buttyan, PhD.**

The Vancouver Prostate Centre, Vancouver, BC, Canada

P: 3

Targeting the Treatment-Induced Developmental Reprogramming Process That Facilitates Progression of Prostate Cancer to Therapy Resistance

Mannan Nouri, PhD Student1, Josselin Caradec, PhD[1], Amy A. Lubik, PhD[1], Na Li, PhD[1], Sarah Truong, MS Student[1], Brett G. Hollier, PhD[2], Mandeep Takhar, PhD[3], Nicholas Erho, PhD[3], Mohamed Alshalafa, PhD3, **Ralph Buttyan[1].**

[1]The Vancouver Prostate Centre, Vancouver, BC, Canada, [2]Queensland University of Technology, Brisbane, Queensland, Australia, [3]GenomeDX Biosciences, Vancouver, BC, Canada

P: 4

Wnt/beta-Catenin Signaling In Neuroendocrine Prostate Cancer

Zachary Connelly, Shu Yang, Jiahe Li, Xiuping Yu.

LSU Health Sciences Center, Shreveport, LA, USA

P: 5

Foxa2 Activates The AR Responsive Promoters In The Absence Of Androgens

Zachary M. Connelly, Shu Yang, Jaihe Li, Robert Matusik, Xiuping Yu.

LSUHSC Shreveport, Shreveport, LA, USA

P: 6

EAF2 Mediates Androgen Regulation Of DNA Repair Through Ku70/ku80 In The Prostate

Laura E. Pascal, PhD, Junkui Ai, Leizhen Wei, Yachen Zhang, Xinpei Yu, Yanqing Gong, Satoshi Nakajima, Joel B. Nelson, Arthur Levine, Li Lan, Zhou Wang.

University of Pittsburgh, Pittsburgh, PA, USA

P: 7

EAF2 And p53 Co-regulate STAT3 Activation In Prostate Carcinogenesis

Laura E. Pascal[1], Yao Wang[1], Junkui Ai[1], Dan Wang[1], Yifeng Jing[1], Jan Pilch[2], Feng Li[1], Lora Rigatti[1], Lara Graham[1], Dalin He[3], Joel Nelson[1], Anil Parwani[1], Zhou Wang[1].

[1]University of Pittsburgh, Pittsburgh, PA, USA, [2]Saarland University Medical Center, Homburg/Saar, Germany, [3]First Affiliated Hospital of Xi'an Jiaotong University, Shaanxi, China

P: 8

An Optimized Method of Murine Prostate Trimming for Histologic Analysis

Ivan Galindo-Cardiel, DVM, Cory F. Brayton, DVM, **Brian Simons, DVM, PhD.**

Johns Hopkins School of Medicine, Baltimore, MD, USA

P: 9

Cyclin-Dependent Kinase 5 (CDK5) Controls Prostate Cancer Metastasis in Vivo and Alters Tumor Immune Response

Brian Simons, DVM, PhD, Thomas Nirschl, Angela Alme, Charles Drake, MD, PhD, Barry Nelkin, PhD.

Johns Hopkins School of Medicine, Baltimore, MD, USA

P: 10

Overcoming Chemotherapy Resistance by Targeting Igf-1 Axis in a Patient with Metastatic Castration-resistant Prostate Cancer: Killing Two Birds With One Stone

Tingrui Wang[1], Jue Wang[2].

[1]St Joseph Hospital and Medical Center, Internal Medicine, Phoenix, AZ, USA, [2]University of Arizona Cancer Center, Phoenix, AZ, USA

P: 11

Molecular Profiling of an Exceptional Responder and a Non-responder To Platinum Based Chemotherapy In Two Patients With Advanced Urothelial Cancer

Tingrui Wang[1], Zonghui Ding[2], Jue Wang[3].

[1]St Joseph Hospital and Medical Center, Internal Medicine, Phoenix, AZ, USA, [2]Mayo Clinic, Department of Biochemistry and Molecular Biology, Phoenix, AZ, USA, [3]University of Arizona Cancer Center, Phoenix, AZ, USA

P: 12

Rationale for the Use of Infiltrating Mesenchymal Stem Cells as a Cell-based Vector for Prostate Cancer

Nathaniel Brennen[1], Baohui Zhang[2], Ibrahim Kulac[1], Nelleke Kisteman[1], Lizamma Antony[1], Hao Wang[1], Alan Meeker[1], Angelo De Marzo[1], Isla Garraway[2], Samuel Denmeade[1], John Isaacs[1].

[1]Johns Hopkins, Baltimore, MD, USA, [2]UCLA, Los Angeles, CA, USA

P: 13 – TRAVEL AWARD

A High-Resolution Atlas of Developing and Mature Mouse Prostate Neuroanatomy

Mark T. Cadena, Bachelor of Science – Biology, Ryan L. Trevena, Diya B. Joseph, Lisa L. Abler, Kyle A. Wegner, Adam J. Gottschalk, Ruth Sullivan, Chad M. Vezina.

University of Wisconsin-Madison, Madison, WI, USA

P: 14

Pharmacologic Targeting of TGF-β mediated EMT in Prostate Cancer

Zheng Cao[1], Shahriar Koochekpour, MD, PhD[2], Kyprianou Natasha, PhD[1].

[1]University of Kentucky, Lexington, KY, USA, [2]Roswell Park Cancer Institute, Buffalo, NY, USA

P: 15

Ezh2 Regulation Through Lysine 63-linked Ubiquitination In Prostate Cancer

Wenfu Lu[1], Shenji Liu[1], Yingqiu Xie[1], Michael G. Izban[1], Billy R. Ballard[1], Sandeep A. Sathyanarayana[1], Samuel E. Adunyah[1], Robert J. Matusik[2], **Zhenbang Chen[1].**

[1]Meharry Medical College, Nashville, TN, USA, [2]Vanderbilt University Medical Center, Nashville, TN, USA

P: 16

Patient-specific Signaling Networks In Lethal Prostate Cancer Through Phosphoproteome-guided Multi-omic Integration

Justin M. Drake, PhD[1], Evan O. Paull[2], Nicholas A. Graham[3], John K. Lee[4], Bryan A. Smith[4], Bjoern Titz[4], Tanya Stoyanova[5], Claire M. Faltermeier[4], Uzunangelov Vladislav[2], Daniel E. Carlin[6], Daniel Teo Fleming[2], Christopher K. Wong[2], Yulia Newton[2], Ajay A. Vashisht[4], Jiaoti Huang[7], James A. Wohlschlegel[4], Thomas G. Graeber[4], Owen N. Witte[4], Joshua M. Stuart[2].

[1]Rutgers Cancer Institute of New Jersey, New Brunswick, NJ, USA, [2]University of California Santa Cruz, Santa Cruz, CA, USA, [3]University of Southern California, Los Angeles, CA, USA, [4]University of California Los Angeles, Los Angeles, CA, USA, [5]Stanford University, Palo Alto, CA, USA, [6]University of California San Diego, San Diego, CA, USA, [7]Duke University, Durham, NC, USA

P: 17

Rb1 Suppresses Prostate Cancer Metastasis And Lineage Plasticity Underlying Castration Resistance

ShengYu Ku, Spencer Rosario, **Leigh Ellis, PhD,** David W. Goodrich, PhD.

Roswell Park Cancer Institute, Buffalo, NY, USA

P: 18

PEDF Causes The Tumoricidal Activity Of Macrophages Toward Prostate Cancer Cells In Vitro.

Stephanie Filleur, PhD[1], Dalia Martinez-Marin[1], Thomas Nelius, MD, PhD[1], Olga V. Volpert, PhD[2].

[1]Texas Tech University Health Sciences Center, LUBBOCK, TX, USA, [2]Northwestern University, Chicago, IL, USA

P: 19 – TRAVEL AWARD

Human Prostate Luminal Cell Differentiation Requires NOTCH3 Induction by p38-MAPK and MYC

Sander B. Frank, PhD[1], Penny Berger[2], Mats Ljungman, PhD[3], Cindy Miranti, PhD[1].

[1]University of Arizona, Tucson, AZ, USA, [2]Van Andel Institute, Grand Rapids, MI, USA, [3]Univeristy of Michigan, Ann Arbor, MI, USA

P: 20

The Role of Prostate Cancer Derived Exosomes in Metabolism

Dometria Gilbert, Joseph Inigo, Dhyan Chandra.

Roswell Park Cancer Institute, Buffalo, NY, USA

P: 21

Paracrine Effect of the Endothelium on Prostate Cancer Cells

Verónica Torres-Estay[1], Catalina Asencio[1], Carla Cembrano[1], Patricia Fuenzalida[1], Daniela Carreño[1], Loreto Véliz[1], Julio Amigo[1], Xavier Figueroa[1], Juan Carlos Sáez[1], Paula Sotomayor[2], Viviana Montecinos[1], **Alejandro S. Godoy[1].**

[1]Pontificia Universidad Católica de Chile, Santiago, Chile, [2]Center for Integrative Medicine and Innovative Science, Universidad Andres Bello, Santiago, Chile.

P: 22

Role of PI3K/AKT Signaling Pathway in Regulation of Expression of Androgen Receptor in ABCG2+ CWR-R1 Prostate Cancer Cells

Destini Goodly.

Howard University, Washington, DC, USA

P: 23

A Specific Co-Activator Interaction Defines the Subset of ETS Transcription Factors Rearranged in Prostate Cancer

Vivekananda Kedage[1], Nagarathinam Selvaraj[1], Taylor R. Nicholas[1], Justin A. Budka[1], Travis J. Jerde[2], **Peter C. Hollenhorst, PhD[1].**

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P: 25

Secreted Hsp90 Promotes Prostate Cancer Stem Cell Heterogeneity

Jasmine Kaur, PhD, Krysal Nolan, **Jennifer Isaacs.**

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P: 26 – TRAVEL AWARD

Tet Inhibits cap-dependent mRNA Translation in Prostate Cancer Cells by Targeting 4EBP[1] and Limits Prostate Cancer Cell Growth and Proliferation

Sergey V. Slepenkov[1], **Praveen K. Jaiswal, PhD[1],** Sweaty Koul[2], Qin Dong[1], Hari K. Koul[3].

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P: 27

Switching on Prostate Cancer Metastasis: CD117 Tyrosine Kinase Activation

Bethany Kerr, PhD[1], Lihong Shi, MD[1], Koran Harris[1], Taylor Peak, MD[1], Aleksander Skardal, PhD[2].

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P: 28

The Role of TGFß Family Members in Androgen Deprivation Therapy-induced Regression of Normal and Neoplastic Prostate

John J. Krolewski, MD-PhD, Shalini Singh, PhD, Chunliu Pan, PhD, Kent L. Nastiuk, PhD.

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P: 29

Targeting GPR30 in Abiraterone- and MDV3100-resistant Prostate Cancer

Olena Tseona, Holly M. Nguyen, Jessica Heide, Rebecca de Frates, Colm Morrissey, Eva Corey, Robert L. Vessella, **Hung-Ming Lam.**

University of Washington, Seattle, WA, USA

P: 30

The Role of Estrogen Receptor Activation in Benign Prostatic Hyperplasia

Teresa T. Liu[1], Jalissa L. Wynder[1], Taryn T. James[1], Jill Macoska[2], William A. Ricke[1].

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P: 31

CXCL12-CXCR4 Axis Activation Promotes Collagen Secretion Through Cullin-RING Ubiquitin Ligase Activity

Jill Macoska, PhD.

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P: 32

Differential Actions of Estrogen Receptor α and β via Non-genomic Signaling in Human Prostate Stem-Progenitor Cells

Shyama Majumdar[1], Neha R. Malhotra[1], Jaqueline C. Rinaldi[1], Lishi Xie[1], Timothy D. Gauntner[1], Wen-Yang Hu[1], Susan Kasper[2], Gail S. Prins[1].

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P: 33

Lack of Tcf21 Disrupts Bladder Organogenesis

Elizabeth Mann, Melissa Mogle, Joo-Seop Park, Pramod Reddy.

Cincinnati Children's Hospital Medical Center, Cincinnati, OH, USA

P: 34 – TRAVEL AWARD

The Contribution ofSox2-positive Cells to Prostatic Epithelium Development and Homeostasis

Erin M. McAuley, Sophia M. Lamperis, Steve Kregel, PhD, Gladell Paner, MD, Donald Vander Griend, PhD.

University of Chicago, Chicago, IL, USA

P: 35 – TRAVEL AWARD

In Vivo Modeling of Metastasis Using a Prostate Cancer Progression Cell Line

Dalton McLean.

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P: 36

Integrin Alpha 6 Protects Castration-resistant Prostate Cancer From PI3K Inhibition by Upregulating BNIP3

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P: 37

Characterizing Models of Indolent and Aggressive Prostate Cancer

Morenike Olu, Karina Miller, Irwin Gelman.

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P: 38

Endoglin Expression in Carcinoma Associated Fibroblasts Enhances Prostate Tumorigenesis and Castration Resistance

Veronica R. Placencio, PhD[1], Manabu Kato, MD[2], Anisha Madhav, M.S.[1], Frank Duong, B.S.[1], Manisha Tripathi, PhD[1], Subhash Haldar, PhD[1], Bryan Angara, M.S.[1], Neil A. Bhowmick, PhD[1].

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P: 39 – TRAVEL AWARD

Dna Hypomethylation Inhibits Proliferation of Bladder Cancer Cells Via Notch[1] Activation

Swathi Ramakrishnan[1], Qiang Hu[1], Nithya Krishnan[1], Dan Wang[1], Evelyn Smit[1], Roberto Pili[2], Monika Rak[3], Jianmin Wang[1], Anna Woloszynska-Read[1].

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P: 40

Effects of Soy Isoflavones and Src Targeting Agents on Metastatic Activity in Prostate Cancer

Lori Rice, PhD, Dietmar W. Siemann, PhD.

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P: 41

A Contrasting Relationship Between the Levels of Vitamin D Metabolites in the Serum and Prostate Tissues of European-American and African-American Prostate Cancer Patients

Zachary Richards[1], Rachael Farhat[1], Drew Makowski[2], Peter Gann[1], Rick Kittles[3], Larisa Nonn[1].

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P: 42

Divergent Effects of Long-term Leptin Supplementation On Prostate Epithelium And Stroma In Obob Mice

Takeshi Sasaki, Omar Franco, LaTayia Aaron, Rodrigo Javier, Yana Filipovich, Susan Crawford, Simon Hayward.

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P: 43

Blockade Of Akt And Nf-κB/p65 Signaling Have Synergistic Antitumor Effect In Prostate Cancer

Eswar Shankar[1], Aditya Joshi, BA[2], Rajnee Kanwal, PhD[1], Aditi Goel, BA[2], Xiaoping Yang[2], Parameswaran Ramakrishnan, PhD[3], Sanjay Gupta, PhD[1].

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P: 44

Pretreatment Circulating MonocyteSubset Gene Expression Predicts Patient Survival Following Dendritic CellVaccination

Anand Sharda[1], Alexnader Wald[1], Mohammad Habiby Kermany[1], Katja Koeppen[2], Thomas Hampton[2], Camilo Fadul[2], Marc Ernstoff[1], Thomas Schwaab[1], Jason Muhitch[1].

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P: 45

Tetrandrine Promotes Apoptosis in Part by Up-regulation Of Death Receptors in Prostate Cancer

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P: 46

Exploring the Role of SNORD78 in Prostate Cancer Disease Progression

Alan Lombard, Chengfei Liu, Wei Lou, Allen Gao.

University of California, Davis, Sacramento, CA, USA

P: 47

Phosphorylation of TOPK At S32 By Erk2 Promotes Tumorigenesis Of Renal Cancer

Huimin Sun[1], Juanjuan Xiao[2], Juntao Yue[1], Shijie Liu[1], Lei Zhang[3], Feng Zhu[2], Chen Shao[1].

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P: 48

Expression of Housekeeping Genes across Prostate Cancer Progression

Jordan Vellky, Sean McSweeney, William Ricke, PhD.

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P: 49 – TRAVEL AWARD

PDEF Promotes Epithelial/Luminal Differentiation and Inhibits Prostate Cancer Metastasis

Fengtian Wang[1], Sweaty Koul[2], Prakash Shanmugam[1], Qin Dong[1], Hari Koul[3].

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P: 50

Androgenic To Estrogenic Switch Modulates Prostatic Growth in Silencing Of Steroid 5-α Reductase 2

Zongwei Wang, PhD[1], Libing Hu, MD[1], Rongbin Ge, PhD, MD[1], Keyan Salari, MD[1], Shulin Wu[1], Shahin Tabatabaei[1], Chin-Lee Wu[1], Chin-Lee Wu[1], Douglas Strand, PhD[2], Aria Olumi[1].

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P: 51

Non-cell-autonomous Regulation of Prostate Epithelial Homeostasis by Androgen Receptor

Boyu Zhang, Oh-Joon Kwon, Li Zhang, Xing Wei, William Brinkley, Yiqun Zhang, Chad Creighton, Michael Ittmann, **Li Xin.**

Baylor College of Medicine, Houston, TX, USA

P: 52

FOXA1, GATA3 and PPARG Cooperate to Drive Luminal Subtype in Bladder Cancer: A Molecular Analysis of Established Human Cell Lines

Hironobu Yamashita, PhD, Joshua Warrick, MD, Vonn Walter, PhD, Lauren Shuman, Vasty Osei Amponsa, Zongyu Zheng, PhD, Wilson Chan, Tiffany L. Whitcomb, DVM, Feng Yue, PhD, Tejaswi Iyyanki, Yuka I. Kawasawa, PhD, Matthew Kaag, Wangsong Guo, MD, Jay D. Raman, MD, David J. Degraff.

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P: 53

Decreased Vascular Endothelial Growth Factor in Radiation Cystitis

Bernadette MM Zwaans, PhD, Sarah Kreuger, PhD, Sarah N. Bartolone, MS, Brian Marples, PhD, Michael B. Chancellor, MD, Laura E. Lamb, PhD.

Poster Session II – Saturday Evening

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Saturday, November 12, 2016

4:00 – 6:00 p.m.

Grand Ballroom DEF

P54-P107

P: 54

Serum Free Complete Medium, an Alternative Medium to Mimic Androgen Deprivation in Human Prostate Cancer Cell Line Models

Michael V. Fiandalo, PhD, John H, Wilton, PhD, Krystin Mantione, MS, Carol Wrzosek, BS, Kristopher Attwood, PhD, Yue Wu, PhD, James L. Mohler, MD.

Roswell Park Cancer Institute, Buffalo, NY, USA

P: 55

Adrenal Androgens Facilitate Prostate Cancer Cell Resistance to Androgen Deprivation Therapy

Michael Fiandalo, PhD, John Stocking, MS, Elena Pop, MD, John Wilton, PhD, Krystin Mantione, MS, James Mohler, MD.

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P: 56

Identification of Human Prostate Stem Cells at Single Cell Resolution

Wen-Yang Hu, MD, Phd[1], Dan-Ping Hu[1], Lishi Xie[1], Ye Li[1], Larisa Nonn[1], Toshi Shioda[2], Gail Prins, Phd[1].

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P: 57

Temporal Expression and Regulation of ERα and ERβ in Human Prostate Stem-progenitor Cells

Wen-Yang Hu, MD, PhD[1], Dan-Ping Hu[1], Lishi Xie[1], Ye Li[1], Shyama Majumdar[1], Larisa Nonn[1], Susan Kasper, PhD[2], Gail Prins, Phd[1].

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P: 59 – TRAVEL AWARD

Elucidating Cross-Resistance Between Docetaxel and Cabazitaxel in Castration Resistant Prostate Cancer

Alan Lombard, Chengfei Liu, Wei Lou, Allen Gao.

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P: 60

Overcoming Renal Cell Carcinoma (RCC) TherapeuticResistance and Avoiding Renal Toxicity by Conjugating Cisplatin to a Tumor Cell Targeting Near-Infrared (NIR) Carbocyanine Dye

Stefan Mrdenovic, MD[1], Yanping Wang, MD[2], Yi Zhang, PhD[2], Yin Lijuan, MD, PhD[2], Gina Chia-Yi Chu, PhD[2], Ruoxiang Wang, MD[2], Srinivas Nandana, PhD[2], Michael Lewis, MD[2], Edwin Posadas, MD[2], Hyung L. Kim, MD[2], Robert A. Figlin, MD[2], Haiyen E. Zhau, PhD[2], Leland W.k. Chung, PhD[2].

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P: 61

Three-dimensional prostate cancer circulatingtumor cell (CTC) spheroids with chemo- and hormone antagonist resistance inculture can be resensitized by targeted near-infrared dye-drug conjugates

Stefan Mrdenovic, MD[1], Yi Zhang, PhD[2], Yin Lijuan, MD, PhD[2], Gina Chia-Yi Chu, PhD[2], Ruoxiang Wang, MD, PhD[2], Srinivas Nandana, PhD[2], Michael Lewis, MD[2], Edwin Posadas, MD[2], Haiyen E. Zhau, PhD[2], Leland W.k. Chung, PhD[2].

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P: 62

Widespread Extraprostatic Expression of Prostate-Specific Promoters in Mouse Models

Rebecca Miller[1], Edward Schaeffer, MD, PhD[2], **Brian W. Simons, DVM, PhD[1].**

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P: 63

A Murine Model of Prostate Cancer Bone Metastasis in a Syngeneic Immunocompentent Host

Brian W. Simons, DVM, PhD[1], Benjamin Benzon, MD[1], Kamyar Ghabili, MD[1], Ashley E. Ross, MD, PhD[1], Paula J. Hurley, PhD[1], Edward M. Schaeffer, MD, PhD[2].

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P: 64

NOD Mice as a Model of Prostatic Hyperplasia

LaTayia Aaron[1], Susan Crawford[2], Simon Hayward[2].

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P: 65

Toward A Standardized Protocol For Urinary Function Testing And Analysis Using The Void Spot Assay

Lisa L. Abler, PhD[1], Steven R. Oakes[1], Jill A. Macoska, PhD[2], Dale E. Bjorling, DVM, MS[1], Chad M. Vezina, PhD[1].

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P: 66 – TRAVEL AWARD

miR-125b-2-3p, a Novel Prostate Cancer Tumor Suppressor miRNA

Sinéad T. Aherne[1], Fiona O'Neill[2], Stephen F. Madden[3], Martin Clynes[2], Conor C. Lynch[1].

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P: 67

Reconditioning the Microbiome to Reduce Stone Burden Assessed Using a Drosophila Model of Nephrolithiasis

Kait Al, Ryan Chanyi, PhD, Gregor Reid, PhD, Hassan Razvi, MD, Jeremy P. Burton, PhD.

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P: 68 – TRAVEL AWARD

Leydig Stem Cell Isolation and Differentiation From Human Testis Biopsies: Potential Modality To Increase Serum Testosterone

Marilia Sanches Santos Rizzo Zutti, Masters, **Himanshu Arora, PhD,** Bruno Nahar, M.D, Joshua M. Hare, MD, Ranjith Ramasamy, MD

University Of Miami, Miami, FL, USA

P: 69

A Feed-forward Mechanism Within the oncomiRic miR-183 Family Cluster May Contribute to its High Levels in Prostate Cancer

Bethany Baumann, LaTonya Williams, Larisa Nonn.

University of Illinois, Chicago, IL, USA

P: 70

Type 2 Cytokines Mediate Microbial Induced Prostate Fibrosis

Ashlee Bell-Cohn, B.S.

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P: 71 – TRAVEL AWARD

Novel Tumor Suppressor Role for the Meis/Hox Axis in Prostate Cancer

Hannah Brechka, Marc Gillard, Raj Bhanvadia, Calvin Van Opstall, Donald J. Vander Griend, PhD.

University of Chicago, Chicago, IL, USA

P: 72

Hypoxia Facilitates Neuroendocrine Prostate Cancer Transdifferentiation

Mia J. Broughton, Sheng Yu Ku, Spencer Rosario, David Goodrich.

Roswell Park Cancer Institute, Buffalo, NY, USA

P: 73 – TRAVEL AWARD

Predicting Risk of Recurrence in Patients with Clear Cell Renal Cell Carcinoma from Urine Supernatant RNA

Justin Cotellessa, Amy Avery, M.S., Todd Riley, PhD, Jill Macoska, PhD.

University of Massachusetts Boston, Boston, MA, USA

P: 75 – TRAVEL AWARD

PVT1-derived Non-coding RNAs in Prostate Cancer in Men of African Ancestry

Dibash K. Das, BS, MA.

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P: 76

Estrogen Utilization of Igf-1r To Signal In Human Prostate Stem/progenitor Cells

Jaqueline de Carvalho Rinaldi[1], Wen-Yang Hu[1], Shyama Majumdar[1], Dan-Ping Hu[1], Lishi Xie[1], Susan Kasper[2], Gail S. Prins[1].

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P: 77

Role of Pro-inflammatory Peri-prostatic Fat Secretome In BPH

Omar Franco[1], Rodrigo Javier[1], Ashleigh Theberge2, Matthew Brady3, Susan Crawford[1], Simon Hayward[1].

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P: 78

Aberrant Bladder Reflexes Can Drive Hind Limb Locomotor Activity Following Complete Suprasacral Spinal Cord Injury

Brian M. Inouye, MD[1], Jillene M. Brooks, MS[2], Danielle J. Degoski, BS[2], Francis M. Hughes, Jr., PhD[1], J. Todd Purves, MA, PhD[1], **Matthew O. Fraser, PhD[3].**

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P: 79

Stable ERα-Expressing Benign Primary Prostate Stromal Cells Share Morphological Features, Molecular Characteristics and a Clinically-Predictive Gene Expression Signature with Cancer-Associated Fibroblasts

Timothy D. Gauntner, PhD[1], Larisa Nonn, PhD[1], Donna M. Peehl, PhD2, Gail S. Prins, PhD[1].

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P: 80

Erbb3-mediated Resistance To Androgen Deprivation Therapy In Castration-resistant Prostate Cancer

Maitreyee K. Jathal[1], **Paramita M. Ghosh, PhD[2].**

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P: 81

RNA-Seq of Prostate Tumor, Stroma, and Benign Glands using PAXgene Fixed Human Prostatectomy Specimens

Marc Gillard, PhD[1], Baizhen Zhu[1], Gladell Paner, MD[1], David VanderWeele, MD, PhD[2], Donald Vandergriend, PhD[1].

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P: 82

The Emerging Role of EZH2 Inhibition in Therapy for Muscle Invasive Bladder Cancer (MIBC)

Victoria Granger[1], Monika Rak[2], Swathi Ramakrishnan[1], Nithya Krishnan[1], Qiang Hu[1], Jianmin Wang[1], Anna Woloszynska-Read[1].

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P: 83

Effects of Combinatorial Drug Therapy on Human Prostate Cell Lines in 2 and 3-Dimensional Culture Environments

Yana Filipovich, MS, Omar E. Franco, MD, PhD, **Simon W. Hayward, PhD.**

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P: 84

Deconstructing BPH Phenotypes: Molecular Pathogenesis of Basal Hyperplasia

Gervaise H. Henry, MSc, Alicia Malewska, MSc, Ryan Mauck, MD, Claus Roehrborn, MD, Douglas Strand, PhD.

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P: 85

Asporin in the Prostate Tumor Microenvironment is a Heritable Mediator of Metastasis

Paula H. Hurley[1], Robert M. Hughes[1], Brian W. Simons[1], George J. Netto[1], Steven

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P: 86

Genomic Analysis of a Longitudinal Series of Surgical Prostate Cancer Bone Metastases and Xenografts from the Same Patient Revealed Selection of a Progressively Therapy Resistant Metastatic Clone

Christina A.M. Jamieson[1], Michelle T. Muldong[1], Abigail Gallegos[1], Christina N. Wu[1], Theresa Mendoza[1], Jin Sung Park[2], Michael A. Liss[3], Omer Raheem[1], Seung Chol Park[4], William Zhu[1], Elana Godebu[1], Jason R. Woo[1], Danielle Burner[1], Amy Strasner[1], Olga Miakicheva[1], Nicholas A. Cacalano[5], Catriona H.M. Jamieson[1], Christopher J. Kane[1], Anna A. Kulidjian[1], Terry Gaasterland[1].

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P: 87

The Role of GRP/GRP-R signaling in Castration Resistant Prostate Cancer Progression

Renjie Jin.

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P: 88

Bisphenol A Effects On Bladder Contraction

Neil S. Lamarre, PhD, Alexandra Waldman, Zunyi Wang, PhD, Dale E. Bjorling, DVM.

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P: 89

Long-term Follow-up Reveals Differential Phenotypes Of Neurologic Impairment And Voiding Dysfunction In A Viral Murine Model Of Multiple Sclerosis

Sanghee Lee, Balachandar Nedumaran, Joseph Hypolite, Randall Meacham, Anna Malykhina.

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P: 90

Enhancing The Efficacy of Olaparib In Castration-resistant Prostate Cancer

Jack Li, Ricky Wang, Evan Kong, **Xiaoqi Liu.**

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P: 91

Identification Of mir-30b-3p and mir-30d-5p As Direct Regulators of Androgen Receptor Signaling in Prostate Cancer by Complementary Functional Microrna Library Screening

Binod Kumar, Salar Khaleghzadegan, Brian Mears, Koji Hatano, Tarana A. Kudrolli, Wasim H. Chowdhury, David Yeater, Charles Ewing, Jun Luo, William B. Isaacs, Luigi Marchionni, **Shawn E. Lupold.**

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P: 92

Drosha Knockdown As A Model to Mimic miRNA Reduction in Prostate Carcinogenesis

Tara N. McCray.

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P: 93

The CRM1 Inhibitor Selinexor Attenuates Bladder Tumor Growth

Maria Mudryj, PhD, Alan P. Lombard, PhD, Stephen J. Libertini, Han Bit Baek, Rachel Nakagawa, Jane Tian, Kathleen Vidello, Robert Weiss, MD, Paramita Ghosh, PhD.

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P: 94

Myokine Signaling Blockade Prevents Androgen Deprivation Therapy Induced Sarcopenia And Promotes Tumor Regression

Chunliu Pan, PhD, Shalini Singh, PhD, Yanni Zulia, B.S., **Kent L. Nastiuk, PhD.**

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P: 95

High Throughput Computer-Based Analysis of Void Spot Assay Data

Steven R. Oakes, Kyle A. Wegner, Lisa A. Abler, Kevin W. Eliceiri, Chad M. Vezina.

University of Wisconsin – Madison, Madison, WI, USA

P: 96

A Combined Loss of FOXA1 and PTEN Accelerates Squamous Differentiation and Progression in a Carcinogen Induced Bladder Cancer

Vasty Osei Amponsa, MS[1], Zongyu Zheng, Ph.D[1], Soumar Bouza[1], Joshua Warrick, MD[1], Cathy Mendelsohn, PhD[2], Klaus Kaestner, Ph.D[3], Xue-Ru Wu, PhD[4], David DeGraff, Ph.D[1].

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P: 97

Growth Hormone-Releasing Hormone (GHRH) Antagonists Inhibit Inflammation-induced Prostate Enlargement in Mice and Reduce the Proliferation of Prostate Epithelial Cells in Vitro

Petra Popovics, PhD.

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P: 98

Pelvic Pain and Voiding Sensory Pathways in Murine Models of Bacterial Prostatitis

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

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P: 99

Inflammation, Androgens And Macrophages In The Prostate: Are We Missing The Link?

Camila Rosat Consiglio, MSc, Sandra Gollnick, PhD.

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P: 100

Urothelial Regeneration of Surgical Defect Sites is mediated by Cytokeratin 5-positive Basal Cells

Frank-Mattias Schafer[1], Khalid Algarrahi[1], Debra Franck[1], Alyssa Savarino[1], Catherine Seager[1], Xuehui Yang[1], Kyle Costa[1], Stefan Lukianov[1], Zarine Balsara[1], Xue Sean Li[1], Rosalyn Adam[1], Cathy Mendelsohn[2], Joshua R. Mauney[1].

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P: 101

Antagonizing the heterogeneity of prostatecancer: Targeting androgen signaling, aerobic glycolysis and epithelial-mesenchymaltransition by human-specific microRNA-644a

Jey Sabith Ebron[1], Jagit Singh[1], Eswar Shankar[2], Kavleen Sikand[1], Crystal M. Weyman[1], Daniel Lindner[3], Sanjay Gupta[2], **Girish C. Shukla[1].**

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P: 102

Foxa1 Knockout is Associated with Increased Carcinogenic Susceptibility and Androgen Receptor Expression in Murine Bladder Cancer

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P: 103

Deep Prostate Tissue Immunohistochemical Staining and Fluorescent Confocal Scanning For 3-Dimmensional Microenvironment Analyses

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P: 104

Genetic Background Influences Susceptibility of Male Mice to Hormone-induced Urinary Dysfunction

Kyle A. Wegner, Kimberly P. Keil, Zunyi Wang, Peiqing Wang, Dale E. Bjorling, Chad M. Vezina.

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P: 105

DNA Methylation, Disease Aggressiveness and Prostate Cancer in African American Men

Anna Woloszynska-Read, PhD, Swathi Ramakrishnan, PhD, Nithya Krishnan, MS, Dan Wang, PhD, Li Yan, PhD.

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P: 106

Arsenic Drives Transformation By Activating p62-Keap1-Nrf2 Pathway Through Autophagy Flux Blockade In Prostate Stem-progenitor Cells

Lishi Xie, PhD, Wen-Yang Hu, Dan-Ping Hu, Jianfu Yang, Ye Li, Gail S. Prins, PhD

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P: 107

Changes in Arginine Metabolism May Be Associated With Androgen Signaling in Prostate Cancer Progression

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Educational Needs and Objectives

STATEMENT OF NEED

Researchers need to keep up with the rapid changes in personalized medicine, urologists and urology residents would need:

a. Education on the components underlying genomic medicine and how these could be integrated to develop more effective, evidence-based approaches to patient care.

b. 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.

c. Education regarding the requirement of a multidisciplinary team approach across a wide range of disciplines to advance personalized medicine.

LEARNING OBJECTIVES

At the conclusion of the meeting, attendees should be able to:

- The impact of hormones and transcription factors on the regulation of urologic organ development.
- The cell type of origin involved in normal organ development and in neoplastic transformation.
- Signaling networks that regulate angiogenesis and metastasis; and processes which might provide novel "druggable" targets.
- Cell-cell interactions during development and disease; and their potential use as "Trojan horses" to deliver drugs to interacting cells.
- Personalized medicine.
- Cell-matrix interactions during wound healing in the lower urinary tract.
- The roles of inflammasomes, immune cells, and bacteria in the regulation of urologic stem cell expansion and the development of disease.

CONTINUING MEDICAL EDUCATION

Accreditation: This activity has been planned and implemented in accordance with the accreditation requirements and policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint providership of the American Urological Association (AUA) and the Society for Basic Urologic Research, Inc. (SBUR). The AUA is accredited by the ACCME to provide continuing medical education for physicians.

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- 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.

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Listed below are disclosures of individuals who are in control of content for the SBUR 2016 Fall Symposium (faculty, planners, authors, etc.).

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Darius Jehan Bägli, MD	Advances in Urology – Journal: Other
Linda A. Baker, MD	None
Carolyn Best, PhD	None
Neil Bhowmick, PhD	None
Lori A. Birder, PhD	Astellas Pharma: Meeting Participant or Lecturer, Scientific Study or Trial
Jeremy P. Burton, PhD	None
Marc Cox, PhD	None
Angelo DeMarzo, MD, PhD	Janssen Research & Development: Scientific Study or Trial; Myriad Genetics: Scientific Study or Trial
Allen Gao, MD, PhD	None
Martin E. Gleave, MD	Sanofi-Aventis: Consultant or Advisor, Scientific Study or Trial; Astra-Zeneca: Consultant or Advisor, Scientific Study or Trial; OncoGenex Technology: Leadership Position, Consultant or Advisor, Scientific Study or Trial, Investment Interest, Owner, Product Development; Pfizer: Scientific Study or Trial; Takeda: Scientific Study or Trial; Astellas: Consultant or Advisor, Scientific Study or Trial; Bayer: Consultant or Advisor
Donald Vander Griend, PhD	None
John T. Isaacs, PhD	Wiley Publishing: Health Publishing; Genspera: Consultant or Advisor, Investment Interest; Sophiris: Investment Interest
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Susan Kasper, PhD	None
John J. Krolewski, MD, PhD	None
Hung-Ming Lam, PhD	None
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Conor Lynch, PhD	None
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Cindy Miranti, PhD	None
Andrea Morrione, PhD	None
Leonard Neckers, PhD	None
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Ganesh V. Raj, MD, PhD	Ptares: Owner, Product Development; C-diagnostics Corp: Consultant or Advisor, Investment Interest; Amgen: Meeting Participant or Lecturer; Johnson and Johnson: Consultant or Advisor, Meeting Participant or Lecturer; Astellas: Meeting Participant or Lecturer, Consultant or Advisor; Janssen: Owner, Product Development; Bayer: Consultant or Advisor, Meeting Participant or Lecturer; Sanofi: Consultant or Advisor, Meeting Participant or Lecturer; Gaudium: Leadership Position, Investment Interest, Owner, Product Development; Medivation: Consultant or Advisor, Meeting Participant or Lecturer, Scientific Study or Trial
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Notes

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SBUR members include molecular biologists, immunologists, epidemiologists, andrologists, oncologists, biochemists and clinical urologic scientists. Members are experts in the study of urologic cancers (prostate, bladder, kidney, testis, penis), the biology of prostate growth, kidney and bladder function, autoimmune urologic diseases, infectious diseases, neurourologic diseases, male reproductive biology, infertility and erectile dysfunction.

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