SBUR
SOCIETY FOR BASIC UROLOGIC RESEARCH, INC.

2020 SBUR Annual Meeting

CELLULAR INTERACTIONS AND
IDENTITIES IN UROLOGIC BIOLOGY

November 11th – 14th

Welcome Colleagues and Friends!

Welcome to the Society for Basic Urologic Research Fall 2020 meeting. We are delighted to be joining with colleagues and friends in this virtual world for our annual meeting focusing on the basic science of urologic disease. Our goals for the meeting are to highlight new research areas, explore connections between diverse areas within our field, be inspired by cutting edge research, learn of new funding opportunities and, of course, enjoy some time (virtually) in the company of friends, both old and new.

The meeting begins with the Trainee Affairs Symposium on Wednesday, November 11th, led by Drs. Arun Sreekumar and Daniel Frigo, followed by our Virtual Poster Session featuring short presentations on selected abstracts. We'll also hear from our Coffey Award finalists highlighting their innovative research. All posters will be available for viewing throughout the meeting.

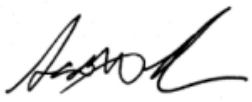
Thursday morning opens with the keynote Leland W. K. Chung lecture by Dr. Hans Clevers, followed by Plenary Sessions covering the following areas: 1) New Models and Technologies for Studying Urologic Biology; 2) Genes and Development in Urologic Health and Disease; 3) Cancer Cell Biology and Communication; 4) Cellular Identity and Lineage Plasticity; and 5) Emerging Cellular Targets and Therapies for Urologic Pathologies. We will also hear about advances in urologic pathologies from the NIDDK-funded Centers and Training Programs. In addition, the American Urological Association (AUA) has sponsored a lecture to be given by Dr. Linda Baker, a pediatric urologist and surgeon-scientist.

We are keenly aware of the financial challenges experienced by many research programs and laboratories during these challenging times. Our shared goals and priorities with the NIH in supporting the next generation of translational researchers in urologic disease are underscored by the generous support of our program by the NIDDK and NCI. In addition we recognize the ongoing support of our mission by the AUA and welcome Dr. Carolyn Best, Director of the Office of Research at the AUA who will provide an update on opportunities for research support and education.

Finally, we would like to thank the SBUR 2020 Annual Meeting Faculty members as well as Program Committee members for their invaluable assistance in planning this meeting. We would like to give special thanks to Drs. Larisa Nonn and Shawn Lupold for their leadership as Co-Chairs of the Abstracts Travel Award Selection (ATAS) Committee, and to all who have contributed time and effort to ensure the success of the meeting. We are grateful for your participation, and we hope that your time and thoughtful discussions during the meeting translate to exciting research opportunities and collaborations in your home institutions.

We look forward to interacting with you over the next few days. Enjoy the meeting!

Rosalyn M. Adam, Ph.D
President



Scott M. Dehm, Ph.D
2020 Scientific Program Chair

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Learning Objectives

- Discuss recent advances in studying the cellular basis of urologic diseases with emphasis on bioengineering, organoid technology, novel animal models, cell-cell communication, immunology, genomics, epigenomics, transcriptomics, proteomics, metabolomics, immunology, and therapeutics;
- Identify critical knowledge gaps and stimulate approaches to address them;
- Disseminate and facilitate novel discoveries in urologic diseases.

2020 Accrediation Information

In support of improving patient care, this activity has been planned and implemented by Cine-Med, Gladwell CME, and the Society for Basic Urologic Research. Cine-Med is jointly accredited by the Accreditation Council for Continuing Medical Education (ACCME), the Accreditation Council for Pharmacy Education (ACPE), and the American Nurses Credentialing Center (ANCC), to provide continuing education for the healthcare team.

Ciné-Med designates this live activity for a maximum of 18.75 AMA PRA Category 1 Credit(s)™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

All other healthcare professionals will receive a Certificate of Participation. For information on the applicability and acceptance of Certificates of Participation for activities designated for AMA PRA Category 1 Credits™, consult your professional licensing board.

CineMed
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Sponsors

Funding for this conference was made possible (in part) by:
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We also gratefully acknowledge support from:
American Urological Association
National Institutes of Health

*The views expressed in written conference materials or publications and by speakers and moderators do not necessarily reflect the official policies of the Department of Health and Human Services; nor does mention by trade names, commercial practices, or organizations imply endorsement by the U.S. Government."

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Travis Jerde, Ph.D.
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Maria Mudryj, Ph.D.
Anna Woloszynska, Ph.D.

Congratulations to the 2020 SBUR Award Winners

Distinguished Service Award

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

Aria F. Olumi, M.D.
Harvard Medical School, Boston, MA

Meritorious Achievement Award

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

Massimo Loda, M.D.
Weill Cornell Medical College, New York, NY

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 urologic research. The award represents the collaborative efforts of these two societies toward their common goal.

Carolyn J.M. Best, Ph.D.
Director of Research for the American Urological Association, Linthicum, MD

Young Investigator Award Recipients

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

Tanya Stoyanova, Ph.D.
Stanford Medicine

Omar Y. Mian, M.D., Ph.D.
Cleveland Clinic

John K. Lee, MD, Ph.D.
Fred Hutchinson Cancer Research Center

Eula and Donald S. Coffey Innovative Research Award Finalists

The Eula and Donald S. Coffey Innovative Research Award will be presented to the most innovative abstract at the SBUR Fall Annual Meeting. The top 3 finalists are asked to give a brief oral presentation of their research findings to open the Awards Presentations. The winner will be announced at the Saturday Awards Ceremony.

Ping Mu, Ph.D.
UT Southwestern Medical Center

U-Ging Lo, Ph.D.
UT Southwestern Medical Center

Patrick Brooks, MD
Missouri State University

Dong-Hoon Lee, Ph.D.
City of Hope National Medical Center

Congratulations to the 2020 SBUR Award Winners

Virtual Travel Award Winners

A primary goal of SBUR is to provide travel grants/stipends to researcher trainees. These grants support travel to/from the Fall Annual Meeting. Award recipients must be SBUR members and a recipient is not allowed to receive the award in two consecutive years. Due to the meeting being held in a virtual format, where no travel was involved, each awardee will receive a SBUR Virtual Travel Award Certificate.

Diya Binoy Joseph, Ph.D.
UT Southwestern Medical Center

Tianfang Ma
Tulane University

Shu Ning
University of California Davis

Alan Lombard, Ph.D.
University of California, Davis

Steve Kregel, Ph.D.
University of Michigan

Jason Garcia, Ph.D.
University of Illinois Chicago

Ralph White III
University of Minnesota

Jaimie Gray, M.S.
The Ohio State University

En-Chi Hsu, Ph.D.
Stanford University

Sajad A Wani, Ph.D.
The Ohio State University

Katherine Morel, Ph.D.
Dana-Farber Cancer Institute/Harvard University

Sridhar Narla, Ph.D.
University of Pittsburgh

Shekha Tahsin, Ph.D.
Phd Student

Anne E Turco
University of Wisconsin-Madison

Merve Aslan
Stanford Medical School

Qiou Wei, M.D., Ph.D.
University of Kentucky

Morgan Zenner
University of Illinois at Chicago

Wei Chen, Ph.D.
University of Pittsburgh

Renee E. Vickman, Ph.D.
NorthShore University HealthSystem

Sanghee Lee, Ph.D.
University of California San Diego

Asmaa El-Kenawi, Ph.D.
H. Lee Moffitt Cancer Center/University of South Florida

Thank You to the 2020 SBUR Distinguished Faculty

Arun Sreekumar, PhD Trainee Affairs Committee Co-Chair, Baylor College of Medicine Houston, TX
Daniel Frigo, Ph.D., MD Trainee Affairs Committee Co-Chair, Anderson Cancer Center, Houston , TX
Rosalyn Adam, Ph.D. SBUR President, Boston Children's Hospital, Boston, MA
X. Sean Li, Ph.D. Boston Children's Hospital, Boston, MA
Paula Hurley, Ph.D. Vanderbilt University, Nashville, TN
Woonyoung Choi, Ph.D. Johns Hopkins University, Baltimore, MD
Justin Drake, PhD. University of Minnesota, Minneapolis, MN
Margot Damaser, Ph.D. The Cleveland Clinic, Cleveland, OH
John Lee, M.D., Ph.D. Fred Hutchinson Cancer Research Center, Seattle, WA
Scott Hultgren, Ph.D. Washington University, St. Louis, MO
Ashleigh Theberge, Ph.D. University of Washington, Seattle, WA
William Ricke, Ph.D. University of Wisconsin, Madison, WI
Zhou Wang, Ph.D. University of Pittsburgh, Pittsburgh, PA
Cathy Mendelsohn, Ph.D. Columbia University, New York, NY
Chad Vezina, Ph.D. University of Wisconsin, Madison, WI
Donald Defranco, Ph.D. University of Pittsburgh, Pittsburgh, PA
Jonathan Barasch, M.D., Ph.D. Columbia University, New York, NY
Theresa Liu, Ph.D. University of Wisconsin, Madison, WI
Laura Pascal, Ph.D. University of Pittsburgh, Pittsburgh, PA
Eric Gonzalez, Ph.D. Duke University, Durham, North Carolina
Carolyn Best, Ph.D. AUA Director of Research, Linthicum, MD
Jason Van Batavia, M.D. University of Pennsylvania, Philadelphia, PA
Michelle Southard-Smith, Ph.D. Vanderbilt University, Nashville, TN
Margaret Vizzard, Ph.D. The University of Vermont, Burlington, VT
Gail Prins, Ph.D. University of Illinois at Chicago, Chicago, IL
Li Xin, Ph.D. University of Washington, Seattle, WA
Bethany Kerr, Ph.D. Wake Forest University, Winston-Salem, NC
A. Ari Hakimi, M.D. Memorial Sloan Kettering Cancer Center, New York, NY
Jennifer Wu, Ph.D. Northwestern University, Chicago, IL
Jelani Zarif, Ph.D. Johns Hopkins University, Baltimore, MD
Daniel Frigo, Ph.D. Anderson Cancer Center, Bethesda, MD
Yuanyuan Zhang, M.D., Ph.D. Wake Forest University, Winston-Salem, NC
Wendy Huss, Ph.D. Roswell Park Comprehensive Cancer Center, Buffalo, NY
David Rickman, Ph.D. Weill Cornell Medical College, New York, NY
Donald VanderGriend, Ph.D. University of Illinois at Chicago, Chicago, IL
Tanya Stoyanova, Ph.D. Stanford University, Stanford, CA
Ganesh Raj, M.D., Ph.D. University of Texas Southwestern Medical Center, Dallas, TX
Amina Zoubeidi, Ph.D. Vancouver Prostate Centre, Vancouver, BC, Canada
Leah Cook, Ph.D. University of Nebraska Medical Center, Omaha, Nebraska
John David Spencer, M.D. Nationwide Children's Hospital, Columbus, OH
Leigh Ellis, Ph.D. Cedars-Sinai Medical Center, Los Angeles, CA
Kerry Burnstein, Ph.D. University of Miami, Miami, FL
Daniel Gioeli, Ph.D. University of Virginia, Charlottesville, VA
Veronica Rodriguez-Bravo, Ph.D. Sidney Kimmel Cancer Center at Jefferson, Philadelphia, PA
Trinity Bivalacqua, M.D., Ph.D. Johns Hopkins University, Baltimore, MD

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2020 SBUR Annual Meeting Preliminary Agenda
November 11th-14th

Discussion topics and times are still being finalized and are subject to change.

SBUR Annual Meeting Planning Committee
Scott Dehm, Ph.D. (Chair), University of Minnesota
Larisa Nonn, Ph.D., University of Illinois at Chicago
Shawn Lupold, Ph.D. John Hopkins University
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Thomas Griffith, Ph.D., University of Minnesota
Beatrice Knudsen, Ph.D., Cedars Sinai Medical Center
LaMonica Stewart, Ph.D., Meharry Medical College
Chad Vezina, Ph.D., University of Wisconsin
Arun Sreekumar, Ph.D., Baylor College of Medicine
Daniel Frigo, Ph.D., University of Texas MD Anderson Cancer Center

Note: Times listed are Central Daylight Time

Wednesday, November 11th

1:00 - 3:00 pm	**SBUR Trainee Affairs Symposium**	Daniel Frigo, Ph.D., MD Anderson Cancer Center Arun Sreekumar, Ph.D., Baylor College of Medicine
5:00 - 7:00 pm	**SBUR Annual Meeting Kickoff** Virtual Poster Presentations by Virtual Travel Awardees and Coffey Award Finalists	Rosalyn Adam, Ph.D., SBUR President

Thursday, November 12th

9:00 - 9:15 am	**Welcome & Introduction**	Rosalyn Adam, Ph.D., SBUR President

9:15 - 10:15 am	**Leland Chung Keynote Speaker and Discussion** Organoids to model human disease	Hans Clevers, M.D., PhD., Utrecht Institute

Hans Clevers: MD and PhD from Utrecht University, Holland. He is Professor in Molecular Genetics at Utrecht University since 1991. He runs his lab in the Hubrecht Institute. Throughout his career, he has worked on the role of Wnt effector, the role of Wnt in adult stem cell biology and of Wnt pathway pathway mutations in colon cancer, Lgr5 as a marker of adult stem cells, and -finally- a method to grow ever-expanding mini-organs('organoids') from Lgr5 stem cells derived from a range of healthy or diseased human tissues. This has led to over 700 publications and >120,000 citations. He is member of the Royal Netherlands Academy of Arts and Sciences, National Academy of Sciences of the USA, Academie des Sciences (Paris) and Royal Society London, Recipient of multiple awards, including the Swiss Louis Jeantet Prize, the Heineken Prize, and the Breakthrough Prize in Life Sciences.

□

10:15 -10:30 am	**BREAK**	

New Models and Technologies for Studying Urologic Biology

Discussion Leaders:

X. Sean Li, Ph.D., Boston Children's Hospital
Paula Hurley, Ph.D., Vanderbilt University

10:30 - 11:00 am	Bladder Cancer Genomics	Woonyoung Choi, Ph.D., Johns Hopkins University
11:00 - 11:30 am	(Phospho)proteomic Approaches to Identify Targets and Biomarkers in Prostate Cancer	Justin Drake, PhD., University of Minnesota
11:30 am -12:00 pm	Regenerative Urology	Margot Damaser, Ph.D., The Cleveland Clinic
12:00pm- 12:15pm	Virtual Travel Awardee Oral Presentation Three-dimensional (3D) co-culture system for organoids plus tissue infiltrating lymphocytes (TILs) derived from patient benign normal and hyperplastic proliferative ureter specimens	Sanghee Lee, Ph.D. University of California San Diego San Diego, California
12:15 - 12:45 pm	Perturbation Screening in Bladder Cancer	John Lee, M.D., Ph.D., Fred Hutchinson Cancer Research Center
12:45 - 1:00 pm	Virtual Travel Awardee Oral Presentation A neuroanatomical mechanism linking perinatal chemical exposure to prostate smooth muscle hyperactivity and altered voiding function	Anne E Turco University of Wisconsin-Madison Madison, Wisconsin
1:00 - 1:30 pm	**LUNCH BREAK (30 minutes)**	
1:30 - 2:00 pm	Models of Urinary Tract Infection	Scott Hultgren, Ph.D., Washington University

2:00 - 2:30 pm	Microfluidics for Coculture	Ashleigh Theberge, Ph.D., University of Washington
2:30 - 2:45 pm	Virtual Travel Awardee Oral Presentation  Decoding Stromal Heterogeneity across BPH Phenotypes	Diya Binoy Joseph, Ph.D. UT Southwestern Medical Center Dallas, Texas

Advances in Urologic Pathologies by NIDDK Urology Centers and Training Programs

Discussion Leaders:

William Ricke, Ph.D., University of Wisconsin
Zhou Wang, Ph.D., University of Pittsburgh
Cathy Mendelsohn, Ph.D., Columbia University

2:45 –3:00 pm	Mouse Models of Prostatic Collagen Accumulation	Chad Vezina, Ph.D., University of Wisconsin
3:00 - 3:15 pm	Mitochondrial Dysfunction in Benign Prostatic Hyperplasia	Donald Defranco, Ph.D., University of Pittsburgh
3:15 -3:30 pm	Epithelial Cell Identity in Kidney Organogenesis	Jonathan Barasch, M.D., Ph.D., Columbia University
3:30 - 4:00 pm	Aging-mediated cellular changes lead to prostatic fibrosis a lower urinary tract dysfunction	Teresa Liu, Ph.D., University of Wisconsin
4:00 – 4:15 pm	**Break**	
4:15 - 4:45 pm	Modeling Prostate Epithelial Barrier Alterations and Lower Urinary Tract Dysfunction in BPH	Laura Pascal, Ph.D., University of Pittsburgh
4:45 - 5:15 pm	Voiding and Muscle Contractility Dysfunction in Novel Animal Model of Detrusor Underactivity	Eric Gonzalez, Ph.D. Duke University
5:15 - 5:45 pm	AUA Research 2020 Update: Opportunities for Research Support and Education	Carolyn Best, Ph.D., AUA Director of Research
Available throughout the meeting	Virtual Posters and Exhibit Hall	

Friday, November 13th

Genes and Development in Urologic Health and Disease

Discussion Leaders:

Jason Van Batavia, M.D., University of Pennsylvania
Rosalyn Adam, Ph.D., Boston Children's Hospital

9:00 - 10:00 am	AUA Lecturer- Genetics and Treatment of Congenital Birth Defects of the GU Tract	Linda Baker, M.D., University of Texas Southwestern Medical Center

Dr. Linda A. Baker is a board-certified pediatric urologist that has practiced in Dallas-Ft Worth, TX for 21 years. After University of Virginia urology residency and pediatric urology fellowship at The Johns Hopkins Hospital, she has developed a complex specialty practice in difficult genitourinary surgical reconstruction for children with prune belly syndrome, disorders of sexual development, and vaginal birth defects. Having held 20 years of NIH funding at the University of Texas Southwestern, her deep interest is using a personalized medicine approach to making new molecular discoveries for children with rare conditions. She has authored/co-authored >120 publications about genitourinary birth defects and pediatric urology. Dr. Baker loves her dear husband and two beautiful grown children.

10:00 - 10:30 am	Development of Lower Urogenital Tract Innervation	Michelle Southard-Smith, Ph.D. Vanderbilt University
10:30 - 11:00 am	Mechanosensation in the Urinary Bladder	Margaret Vizzard, Ph.D., The University of Vermont
11:00 – 11:15 am	**BREAK**	

Cancer Cell Biology and Communication

Discussion Leaders:

Gail Prins, Ph.D., University of Illinois at Chicago
Li Xin, Ph.D., University of Washington

11:15 - 11:45 am	Prostate Cancer Bone Tropism	Bethany Kerr, Ph.D., Wake Forest University
11:45 am – 12:15 pm	Clear Cell Kidney Cancer TME Transcript Profiling	A. Ari Hakimi, M.D., Memorial Sloan Kettering Cancer Center
12:15 - 12:45 pm	Immune Attach and Evasion in Urologic Cancers	Jennifer Wu, Ph.D., Northwestern University
12:45 – 1:00 pm	Virtual Travel Awardee Oral Presentation Regulation of megalin by vitamin D as the mechanism for differential levels of intra-prostatic androgens between African American and Caucasian men	Jason Garcia University of Illinois Chicago Chicago, Illinois

1:00 - 1:30 pm	**Lunch Break (30 minutes)**	
1:30 – 2:00 pm	Macrophages in Prostate Cancer	Jelani Zarif, Ph.D., Johns Hopkins University
2:00 – 2:30 pm	Targeting CAMKK2 to Counter Prostate Cancer Metabolism	Daniel Frigo, Ph.D., Anderson Cancer Center
2:30 – 2:45 pm	Virtual Travel Awardee Oral Presentation  Tristetraprolin Loss Drives the Progression of Aggressive Prostate Cancer	Katherine Morel, Ph.D. Dana-Farber Cancer Institute/Harvard University Boston, Massachusetts
2:45 – 3:00 pm	Virtual Travel Awardee Oral Presentation  Prostate tumor-derived TNFα/TGFβ down regulate AR expression in the prostate cancer stroma through TAK1, NF-κB, and p38 signaling	Shekha Tahsin, M.S. University of Arizona Tucson, Arizona

Cellular Identity and Lineage Plasticity

Discussion Leaders:

Yuanyuan Zhang, M.D., Ph.D., Wake Forest University
Wendy Huss, Ph.D., Roswell Park Comprehensive Cancer Center

3:00 – 3:30 pm	Temporal Evolution of Cellular Heterogeneity During the Progression to Advance, AR-negative Prostate Cancer	David Rickman, Ph.D., Weill Cornell Medical College
3:30 – 4:00 pm	SOX2 in Prostate Regeneration and Disease	Donald VanderGriend, Ph.D., University of Illinois at Chicago
4:00- 4:30 pm	Defining New Drivers and Therapeutic Targets for Neuroendocrine Prostate Cancer	Tanya Stoyanova, Ph.D., Stanford University
4:40 – 4:45 pm	**BREAK**	
4:45 – 5:15 pm	SBUR Awards Presentation ● Virtual Travel Awards ● Young Investigator Awards ● SWIU/SBUR Award ● Distinguished Service Award ● Meritorious Achievement Award ● Coffey Research Award Winner	
5:15 – 5:45 pm	SBUR Business Meeting	Members Only
Available throughout the meeting	Virtual Posters and Exhibit Hall	

Saturday, November 14th

Emerging Cellular Targets and Therapies for Urologic Pathologies

Discussion Leaders:

Ganesh Raj, M.D., Ph.D., University of Texas Southwestern Medical Center
Amina Zoubeidi, Ph.D., Vancouver Prostate Centre

9:00 - 9:30 am	Defining the Roles of Neutrophils in Bone Metastatic Prostate Cancer	Leah Cook, Ph.D., University of Nebraska Medical Center
9:30 - 10:00 am	The Roles of the Ribonuclease A Superfamily in Immune Protection from Urinary Tract Infection	John David Spencer, M.D., Nationwide Children's Hospital
10:00 - 10:30 am	Targeting mRNA Stabilization to Inhibit Linage Plasticity in Prostate Cancer	Leigh Ellis, Ph.D., Cedars-Sinai Medical Center
10:30 - 11:00 am	AVPR1A in Prostate Cancer	Kerry Burnstein, Ph.D., University of Miami
11:00 - 11:15 am	**BREAK**	
11:15 - 11:30 am	Virtual Travel Awardee Oral Presentation  Resistance to Olaparib is Dependent on Re-Emergence from G2/M Arrested Senescence	Alan Lombard, Ph.D. University of California Davis, California
11:30 am – 12:00 pm	The lncRNA HULLK in prostate cancer	Daniel Gioeli, Ph.D., University of Virginia
12:00 - 12:30 pm	Nuclear Pore-regulated Pathways as Novel Mechanisms of Prostate Cancer Aggressiveness	Veronica Rodriguez-Bravo, Ph.D., Sidney Kimmel Cancer Center at Jefferson
12:30 - 1:00 pm	Response and Resistance to BCG Therapy for Bladder Cancer	Trinity Bivalacqua, M.D., Ph.D., Johns Hopkins University
1:00 pm	Farewell	Rosalyn Adam, Ph.D., SBUR President

Thursday, November 12th, 2020

12:00pm- 12:15pm

Travel Award Presentation #1:
Three-dimensional (3D) co-culture system for organoids plus tissue infiltrating lymphocytes (TILs) derived from patient benign normal and hyperplastic proliferative ureter specimens

Sanghee Lee, Ph.D., University of California San Diego
San Diego, California

Background:
Three-dimensional (3D) co-culture systems recapitulates in vivo-like autocrine and paracrine signaling in culture systems which retain the cellular heterogeneity of the original tissue. However, such systems have not been established for benign inflammatory changes in ureter. We established a 3D organoids and tissue infiltrating lymphocytes (TILs) co-culture system to model host-immune interplay in normal ureter and ureter with benign proliferative changes in human.

Methods:
10 mg of normal tissue and 10 mg of tissue with benign proliferative changes (abnormal) were harvested from upper ureter from a 78-year-old female patient. 3D organoid and TILs cultures were prepared and maintained for three weeks separately. Host-immune interplay of benign ureter was modeled in co-culture of 3D ureter organoids and 480,000 TILs. The TILs from abnormal tissues were not proliferating as much as the TILs from benign normal, therefore, TILs were added as a 1:1 mixture of TILs from benign normal and abnormal tissues to have total of 480,000 TILs, a pre-determined number for successful co-culture. Four areas within each coculture plate were imaged by EVOS microscope at 0, 4, 28, 56, 84 and 91 hr. 3D organoids alone, co-cultured organoids plus TILs were processed for H&E staining and Immunohistochemistry using antibodies specific to Keratin 5 (CK5), P63 and Uroplakin III (UpkIII).

Results:
The abnormal tissues were later determined by the pathologist to be benign hyperplasia. 3D organoid cultures of benign normal vs benign hyperplastic proliferation of ureter contained a mix of cell masses. Strikingly, microscopic imaging of co-cultures revealed that TILs migrated into the matrigel dome and finally infiltrated ureter organoids. TILs retained their original ability to infiltrate host benign normal ureter organoids even after their surrounding environment had changed. Ureter organoids were CK5 and P63 positive but Upk III negative. A functional consequence of TILs infiltration of the organoids was a morphological change in organoids structure, an irregular boarder of organoids and an extrusion of luminal components. Both types of TILs infiltration were observed in both benign normal vs benign hyperplastic proliferation ureter.

Conclusions:
Our study provides the first patient-derived model of benign ureter organoids plus TILs which maintained functional and cellular phenotype of urothelial cells and TILs. Funding: The Leo and Anne Albert Charitable Trust Foundation, The JM Foundation.

12:45-1:00pm

A neuroanatomical mechanism linking perinatal chemical exposure to prostate smooth muscle hyperactivity and altered voiding function

Anne E Turco, University of Wisconsin-Madison
Madison, Wisconsin

Background:
The historical focus of male lower urinary tract dysfunction (LUTD) has been benign prostatic enlargement and other aging-related processes. Little attention has been directed towards the influence of early life events on urinary physiology in advanced age. Here, we identify the intrauterine environment as a modifier of adult voiding function and risk factor for male LUTD.

Methods:
To model environmental chemical exposures, we exposed pregnant mice to the environmental contaminant 2,3,7,8 tetrachlorodibenzo-p-dioxin (TCDD, 1 µg/kg), coinciding with initiation of lower urinary tract development in male fetuses. We aged male pups to embryonic day (E) 17.5, postnatal (P) day 9, and 14 weeks of age and collected prostate tissue to stain for noradrenergic axons via immunohistochemistry. RNAseq was performed on E16.75 fetal prostates to identify dysregulated neurotrophic factors. Prostate muscle sensitivity was measured using genetically encoded calcium receptors and tissue bath. Urinary frequency was measured using cystometry.

Results:
Fetal TCDD exposure incites abnormal urodynamics in adult male mice, including increased urinary voiding frequency. TCDD also enhances adult prostate sensitivity to electrically evoked muscle contraction, suggesting increased autonomic tone. IUL TCDD exposure stably increases noradrenergic axon density beginning in the fetal period and persisting into adulthood. These changes are accompanied an increase in the abundance of a neurotrophin, Artemin (Artn), in the fetal prostate.

Conclusions:
This is the first evidence that intrauterine chemical exposures can reprogram prostate neuroanatomical development and drive prostatic smooth muscle hyperactivity in adulthood, which may create a susceptible phenotype for aging-related male lower urinary tract dysfunction.

2:30-2:45pm

Decoding Stromal Heterogeneity across BPH Phenotypes

Diya Binoy Joseph, Ph.D., UT Southwestern Medical Center
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Background:
Benign Prostatic Hyperplasia (BPH) is a non-malignant enlargement of the prostate that occurs with aging and is associated with Lower Urinary Tract Symptoms (LUTS). Therapeutic options often fail, necessitating surgical resection of the prostate. The phenotypic and cellular heterogeneity of BPH is thought to contribute to treatment resistance. BPH patients present with multiple nodules grouped around the prostatic urethra in the transition zone. The composition of these nodules vary with some being solely comprised of stromal cells and others containing a mixture of stromal and epithelial cells. In addition, some patients present with a band of fibrotic tissue around the prostatic urethra that we term as peri-urethral fibrosis. Here, we describe stromal cell heterogeneity in the normal human prostate and across the different BPH phenotypes.

Methods:
We used unbiased single cell RNA-sequencing (scRNA-seq) to obtain transcriptomic identities of stromal cells from normal human prostates and prostates from BPH patients who underwent simple prostatectomy. Cell clusters identified from scRNA-seq were validated in situ using immunohistochemistry and RNA in situ hybridization.

Results:
We found that the stromal composition of the normal prostate consists of two major fibroblast populations, a prostate smooth muscle cell type, a vascular smooth muscle cell type and pericytes. One fibroblast sub-type, marked by expression of MFAP4, is abundant around the prostatic urethra and in the interstitial space between prostate glands. The second

fibroblast population, marked by expression of APOD, is found closely associated with the secretory epithelium of the prostate and is absent from the spaces between prostate glands. The MFAP4+ fibroblast subtype extends into the bladder and represents a lower urinary tract fibroblast whereas APOD+ fibroblasts are restricted to the prostate. MFAP4+ fibroblasts are present within stromal and glandular nodules from BPH patients and are increased in peri-urethral fibrosis. APOD+ fibroblasts are absent from stromal nodules and regions of peri-urethral fibrosis. Desmin expressing smooth muscle cells are largely absent from regions of peri-urethral fibrosis. Wisps of smooth muscle are present in stromal nodules while glandular nodules are packed with Desmin expressing cells.

Conclusions:
Our results highlight the identity and anatomical location of stromal cell types in the normal and BPH prostate. We expect that a molecular understanding of stromal cell types in the prostate will aid in a better understanding of the etiology of BPH.

Friday, November 13th, 2020

12:45-1:00pm

Regulation of megalin by vitamin D as the mechanism for differential levels of intra-prostatic androgens between African American and Caucasian men

Jason Garcia, University of Illinois Chicago
Chicago, Illinois

Prostate cancer (PCa) is a hormonally driven cancer and is currently the third most common cancer in the US. African American (AA) men are disproportionately at risk for both PCa and vitamin D (vitD) deficiency compared to white men. The numerous chemopreventative properties of vitD and epidemiological relationship of vitD status with PCa aggressiveness and mortality has led to the hypothesis that vitD deficiency is a biological contributor to PCa disparity in AA men. Our lab recently reported an unexpected relationship between serum and intraprostatic vitD metabolites 25-hydroxyvitamin D (25(OH)D) and 1,25- dihydroxyvitamin D in AA men. We also observed that Megalin, a multi-liganded endocytic membrane receptor encoded by the gene LRP2, was expressed in the prostate epithelium and is regulated by vitD. Extra-renal activity of Megalin has not been well studied as the widely accepted Free Hormone Hypothesis assumes passive diffusion of circulating free hormones into tissues. The presence of megalin suggests that globulin bound hormones from the circulation, including 25(OH)D bound to vitamin D binding protein (DBP) and testosterone (T) bound to sex hormone binding globulin (SHBG), are imported into the prostate in a regulated manner. Here we examine megalin as a potential mechanism to regulate globulin bound hormone import into the prostate. 25(OH)D decreased expression of LRP2 in primary prostate epithelial cells and fresh human prostate tissue slice explants. DBP-bound 25(OH)D and SHBG-bound T were imported into these prostate models and transcriptionally active. Lastly, we quantified T and its active metabolite dihydrotestosterone (DHT) in the patient cohort from our prior study. Prostatic DHT levels inversely correlated with serum 25(OH)D status. AA men had higher levels of DHT in prostate tissue compared to white men. These clinical findings support our hypothesis that vitD status regulates intraprostatic hormone levels. In summary, we report the presence of a negative feedback loop in which vitD deficiency increases hormone import into prostate epithelium via megalin. Therefore, the upregulation of megalin in the setting of vitamin D deficiency may facilitate increased import of circulating sex steroids into the prostate contributing to carcinogenesis in AA men.

2:30-2:45pm

Tristetraprolin Loss Drives the Progression of Aggressive Prostate Cancer

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Background:
Lineage plasticity drives therapy resistance and progression of lethal disease, so it is critical to identify actionable targets to inhibit or reverse such cellular changes. Recently, we and others have characterized the molecular landscape of androgen indifferent castrate resistant prostate cancer (CRPC-AI) and have identified and validated genetic-epigenetic mechanisms, including concurrent loss of PTEN and RB1 and/or TP53, and induction of specific epigenetic modifiers, such as EZH2, required for driving prostate cancer (PCa) lineage plasticity. Preliminary data from mouse and human samples of CRPC-AI indicate significant reduction in mRNA destabilizing protein known as Tristetraprolin (TTP - encoded by the ZFP36 gene) when compared to CRPC adenocarcinomas. TTP binds to AU-rich elements in the 3`-untranslated regions of target mRNAs to promote their degradation. TTP regulates NF-κB through TNF-α mRNA degradation and inhibiting p65 nuclear translocation, and is an important regulator of proper neuronal differentiation.

Methods:
RNA and protein expression in clinical and GEMM PCa data sets were analyzed. Novel genetically engineered mouse models (GEMMs) were generated to represent loss of Ttp alone or in combination with Pten. GEMM tissues were examined by IHC, IF, WB and qPCR, and human and GEMM-derived cell lines were utilized for therapy studies.

Results:
Specific deletion of Ttp in GEMM prostate tissue induces prostatic intraepithelial neoplasia. Co-deletion of Pten and Ttp shows significant acceleration of disease progression, including dissemination of tumor cells to distant tissues, compared to Pten deletion. Further, both low TTP and concurrent low PTEN/TTP expression in multiple PCa patient cohorts selects for most aggressive disease. These patients demonstrate an increased gene signature score of lineage plasticity and EZH2 gene expression. Deletion of Ttp in mouse PCa cells results in an inflammatory tumor phenotype, increased p65 (NF-κB) expression and signaling, reduced AR expression, increased expression of synaptophysin and resistance to androgen deprivation therapy (ADT) with enzalutamide. Dimethylaminoparthenolide (DMAPT), an NF-κB inhibitor, preferentially slows growth of TTP-deficient PCa cells compared to TTP-proficient cells and restores sensitivity to enzalutamide in TTP-deficient cell lines.

Conclusions:
Together, our data demonstrates that TTP is an important regulator of prostate cellular identity, and loss of TTP drives progression of an aggressive prostate cancer phenotype. Of clinical significance, TTP loss provides a novel therapeutic vulnerability to either prevent or reverse resistance to ADT due to lineage plasticity in PCa patients.

2:45-3:00pm

Prostate tumor-derived TNFα/TGFβ down regulate AR expression in the prostate cancer stroma through TAK1, NF-κB, and p38 signaling

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Tucson, Arizona

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Background:
The ability of stromal AR to secrete differentiation factors that act on the surrounding epithelial cells is a critical determinant of normal prostate gland formation. However, loss of stromal AR is associated with increasing Gleason grade and development of aggressive prostate cancer, resulting in less-differentiated tumors. The mechanisms that lead to stromal AR loss are unknown. In this study, we tested the hypothesis that tumor-secreted factors, such as TGFβ and TNFα, are responsible for AR

repression through NF-κB activation.

Methods:
Using benign human immortalized prostate stroma cells (BHPrs1) as a model, the mechanistic downregulation of AR was examined. Using cytokine array profiling, we identified TNFα and TGFβ1 as two major factors secreted by two different PCa cell lines, C4-2 and 22RV1. RT-qPCR, immunoblotting, pharmacological inhibitors and shRNA knock-down were utilized to identify the TNFα/TGFβ-mediated signaling pathways contributing AR downregulation.

Results:
Treatment of BHPrs1 cells with TNFα or TGFβ leads to loss AR expression within 18-24 hours. This was accompanied by a parallel loss of AR mRNA. TNFα-treated cells showed a time and concentrationdependent activation of NF-κB, TAK1, p38-MAPK, and Jnk, which peaked at 18-24hr. Inhibitors of TAK, p38, and NFκB, but not Jnk, blocked the ability of TNFα to downregulate AR expression. These data indicate that the activation of p38-MAPK and NF-κB via TAK1 are involved in suppressing AR expression in response to TNFα. Future experiments will determine if TGFβ uses the same pathways and whether NFκB and ATF1 binding sites on the AR promoter are involved.

Conclusions:
These results show that there are two pathways downstream of TAK1, p38 and NF-κB, that are responsible for suppressing AR expression in the prostate cancer stroma. Future experiments will explore the significance of these findings in the context of disease.

Saturday, November 14th, 2020

11:15-11:30am

Resistance to Olaparib is Dependent on Re-Emergence from G2/M Arrested Senescence

Alan Lombard, Ph.D., University of California
Davis, California

Background:
Inhibition of poly (ADP-ribose) polymerase (PARP) is an exciting treatment strategy recently approved for prostate cancer patients with homologous recombination repair defects. Despite this advance in the field, there are important unanswered questions regarding PARP inhibitor (PARPi) use; 1) How do PARPi sensitive cells respond to treatment? 2) What mechanisms give rise to PARPi resistance? To address these questions, we sought to characterize response to PARP inhibition using PARPi sensitive LNCaP and C4-2B cells and two PARPi resistant cell line derivatives.

Methods:
LN-OlapR and 2B-OlapR olaparib resistant cell lines were generated from LNCaP and C4-2B cells through chronic exposure to increasing doses of olaparib. Western blot was used to detect PARP activity, apoptosis, and DNA damage. Flow cytometry and beta-galactosidase activity assays tested response to PARPi's. CDK1 was inhibited using RNAi and small molecule drug, BMS-265246.

Results:
OlapR cells exhibit marked resistance to olaparib versus parental cells. OlapR models are also crossresistant to other clinically relevant PARPi's including rucaparib, niraparib, and talazoparib. Mechanistically, PARPi treatment inhibits PARP catalytic activity, induces DNA double strand breaks, and activates apoptosis in LNCaP and C4-2B cells. We also observed a cytostatic response in a significant proportion of cells. Flow cytometry showed a robust G2/M arrest in response to olaparib treatment, accompanied by marked increases in p21 expression and beta-galactosidase activity, suggestive of senescence. In contrast, OlapR cells do not exhibit G2/M arrest, increased p21, or senescence in response to PARP inhibition, suggesting

that resistance is dependent upon re-emergence from p21 dependent senescence. CDK1 activity governs the G2/M cell cycle phases and is a primary p21 target. Thus, we tested if CDK1 inhibition re-sensitizes OlapR cells to PARPi treatment. Indeed, we found that CDK1 inhibition by either siRNA or BMS-265246 re-sensitized OlapR cells to treatment.

Conclusions:
We find that response to PARP inhibition is characterized largely by a G2/M arrested senescence, which may give rise to resistance through re-emergence from this state. PARPi induced senescence provides an escape route from PARPi cytotoxicity, creating a repository of persistent cells which can give rise to resistance. Targeting CDK1 may prove to be an efficacious strategy for the treatment of reemerged, PARPi resistant prostate cancer.

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