

## MEETING REPORT

# SBUR 2025 ANNUAL MEETING REPORT

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This report summarizes the scientific presentations from the 33rd Annual Meeting of the Society for Basic Urological Research (SBUR), held from November 13 to 16, 2025, in Orlando, Florida, USA. This year's meeting gathered researchers, clinicians, and trainees from the United States and other countries to discuss new discoveries that are shaping the future of urologic research. Sessions showcased advances from basic molecular mechanisms to clinical applications, focusing on prostate cancer biology, treatment resistance, metabolic weaknesses, and new treatment strategies. The presentations highlighted the need to combine basic research with various clinical approaches to improve patient outcomes and further the field of urologic oncology.

## PLENARY SESSION I: INNOVATIVE MODELS AND TECHNOLOGIES IN UROLOGICAL RESEARCH

Nathan R. Tykocki, PhD (Michigan State University), presented "Get Up and Stretch: Compliance, Remodeling and the Sensation of Bladder Fullness." He explained that traditional methods for measuring bladder compliance ignore structural differences. His team developed a mirror-based, high-speed imag-

ing system that creates detailed 3D and 4D reconstructions of bladder shape and behavior during filling. This platform allowed the lab to uncover rapid inflammation-induced remodeling, irregular pressure events, and changed compliance partly driven by neurogenic signaling. These findings highlight the close connections between tissue mechanics, sensory pathways, and the behavior of the extracellular matrix.

Zohreh Izadifar, PhD (Boston Children's Hospital), followed with "Human Urogenital Organ Chips: Innovative *In Vitro* Models for Urological Research." She emphasized that neuro-genital organs have complex mechanics, diverse cell types, and changing biological environments that standard *in vitro* or animal models do not capture. She described organ-on-chip technologies that recreate human-like systems, showcasing cervix and vagina chips that imitate hormonal responses, mucus behavior, barrier function, and microbiome dynamics, with ongoing development of urinary tract and fertility models.

Leigh Ellis, PhD (Uniformed Services University), then discussed "Spatial Mapping of Accessible Chromatin Landscapes in Prostate Cancer." He outlined an emerging spatial-epigenomics framework to better understand prostate cancer evolution. He noted a rising incidence of the disease among Black men

and explained how tumors transition through various treatment-driven states, influenced by the loss of tumor suppressors and potentially reversible with EZH2 inhibition. His group's single-cell and spatial multi-omics analyses show shared cell-type compositions across races and distinct molecular differences, such as increased EMT programs and unique CTCF motifs, alongside significant heterogeneity within the prostate. Future integration of chromatin, transcriptional, and histological data aims to identify early resistance-related epigenetic features. Finally, Stephen A. Kaplan, MD, FACS (Icahn School of Medicine at Mount Sinai), presented "AUA – Advancing Urology Through Research and Innovation." He shared how his career in clinical practice, basic science, and entrepreneurship led to the creation of the AUA Innovation Nexus. He was motivated by funding challenges, declining physician-scientist pathways, and unmet clinical needs. He explained how turning daily clinical frustrations into practical solutions resulted in successful company launches and inspired a platform for education, mentorship, consulting, startup showcases, and future grant programs aimed at fostering ongoing urologic innovation and improving patient care.

## PLENARY SESSION II: TRANSCRIPTIONAL, POST-TRANSCRIPTIONAL, AND EPIGENETIC REGULATION IN UROLOGICAL DISEASES

David P. Labbé, PhD (McGill University), presented "MYC-driven Vulnerabilities in Prostate Cancer." He emphasized that diet significantly affects prostate cancer risk, progression, and treatment response. He demonstrated that high saturated-fat intake can rapidly push early lesions towards more aggressive states, promote invasion, increase angiogenesis, cause DNA damage, and lead to immunosuppression in established tumors, while omega-3s provide protective effects. Brief dietary changes before radiotherapy can also enhance treatment sensitivity, which relies on a functional immune system, and human data support these stage-specific dietary influences.

Katherine Xu, PhD (Columbia University), then presented "Genome-Wide Association Study Across Biobanks Identifies New Susceptibility Loci for Urinary Tract Infections." This study is the largest UTI genetic analysis to date, involving over 1.8 million

individuals and identifying 36 significant loci, including a strong new signal near *PSCA*. Her team found that *PSCA* is expressed throughout the urinary tract, secreted into urine, binds directly to *E. coli*, inhibits bacterial growth, and offers protection against infection in mouse models. Protective *PSCA* variants also lower the risk of other infection-related diseases, although they showed opposite associations in some urogenital cancers.

Feng Yang, PhD (Baylor College of Medicine), delivered a talk titled "MAPK4, an Emerging Oncogenic Driver to Promote Prostate Cancer." He detailed how specific structural regions of MAPK4 are essential for activating AKT. Disruption of these regions weakens the interaction and reduces downstream signaling. He also highlighted GATA2's role as a transcription factor regulating androgen receptors linked to aggressive, treatment-resistant disease. His group discovered an enzyme that targets GATA2 for degradation, suggesting a potential therapeutic strategy to limit AR-driven tumor growth.

## PLENARY SESSION III: MICROBIOME, IMMUNITY, AND IMMUNE THERAPEUTICS IN UROLOGICAL DISEASES

Di Zhao, PhD (MD Anderson Cancer Center), presented "Decipher Actionable Genetic Alterations for Personalized Immunotherapy in Advanced Prostate Cancer." He explained that tumors with *PTEN* and *TP53* loss often increase B7-H3 levels to suppress immune activity, making B7-H3 an attractive therapeutic target. While blocking B7-H3 alone offers limited benefit due to compensatory immune pathways, combining its inhibition with PD-L1 or CTLA-4 blockade results in stronger and longer-lasting anti-tumor responses. Dr. Zhao also highlighted *ASH1L* as a newly identified driver of metastasis, particularly in bone, where it promotes invasion and reshapes the microenvironment by influencing macrophages to support metastatic growth.

Nicole J. De Nisco, PhD (The University of Texas at Dallas), followed with "Microbiome After Menopause: Impact on Urinary Microbiome, Metabolome and Recurrent UTI Susceptibility." She emphasized that recurrent UTIs, especially common in postmenopausal women, result from bacterial invasion of deep bladder tissues, persistent inflammation, and changes in the urinary microbiome due to age and hormones. Her group found that premenopausal

women usually carry protective *Lactobacillus*, while postmenopausal women often display dysbiosis and lingering uropathogens linked to recurring infections. Estrogen use restored *Lactobacillus* dominance, and metabolomic profiling identified lipid-related signatures and a possible prognostic biomarker related to future UTI risk.

Conor C. Lynch, PhD (Moffitt Cancer Center), then discussed “Driving  $\gamma\delta$  CAR-T Cells into the Bone Metastatic Prostate Microenvironment.” He described developing  $\gamma\delta$  CAR-T cells for bone-metastatic castration-resistant prostate cancer, which responds poorly to standard immunotherapies.  $\gamma\delta$  T cells naturally recognize tumors without MHC and migrate to epithelial tissues. Their activity is enhanced by zoledronate, a drug often given to these patients. In preclinical studies,  $\gamma\delta$  CAR-T cells showed strong tumor-killing capabilities that improved when combined with zoledronate. Spatial transcriptomics indicated that stromal cells in the bone niche support CAR-T function. These promising findings have advanced the therapy into a first-in-human clinical trial.

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## PLENARY SESSION IV: BIG DATA AND ARTIFICIAL INTELLIGENCE IN UROLOGY

Liang Cheng, MD, MS (Brown University), presented “Harnessing Artificial Intelligence for Prostate Cancer Care.” He discussed the increasing global burden of prostate cancer and the challenges of late diagnosis and a shrinking pathology workforce. He highlighted how AI is changing detection, biopsy interpretation, grading accuracy, and outcome prediction, achieving performance levels nearly equal to pathologists in Gleason grading, molecular prediction, and reducing errors. While AI offers improvements in efficiency, cost reduction, and better integration of histology, imaging, and clinical data, challenges remain related to generalizability, regulation, and dataset quality.

Housheng Hansen He, PhD (University of Toronto), followed with “Transcriptional and Functional Landscape of Circular RNA in Prostate Cancer.” He showed that circRNAs are widely expressed in prostate tumors and influence key pathways that drive aggressive disease, suggesting their potential as biomarkers and therapeutic targets.

Victor Jin, PhD (Medical College of Wisconsin), then presented “Learning Multi-Omics-Seq Data to Predict Pioneer Factors-Mediated Nucleosome Repo-

sitioning in Lethal Prostate Cancer.” He explained how prostate cancer evolves from androgen-dependent to castration-resistant forms through lineage plasticity driven by pioneer transcription factors like FOXA1, GATA2, and HOXB13. His team used computational tools such as ePEST and NucHMM to show that FOXA1 and GATA2 play distinct roles in chromatin remodeling. GATA2’s binding at canonical AR enhancers may drive the transition from ADPC to CRPC, highlighting it as a promising therapeutic target.

Finally, Zheng Xia, PhD (Oregon Health & Science University), presented “Single-Cell Transcriptome Atlas to Reveal Heterogeneity and Cellular Ecosystem Dynamics During Prostate Cancer Progression.” He introduced PCCAT, a detailed atlas created from hundreds of integrated single-cell datasets from normal and malignant prostate tissues. The atlas revealed significant heterogeneity among tumor, stromal, and immune cells and identified specific fibroblast and immune populations that encourage aggressive disease. It also includes an interactive portal to aid in discovering biomarkers and development strategies for advanced prostate cancer.

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## PLENARY SESSION V: CELLULAR HETEROGENEITY AND PLASTICITY IN UROLOGICAL BIOLOGY

David Goodrich, PhD, from Roswell Park Comprehensive Cancer Center, discussed his work on “Molecular Determinants of Prostate Cancer Lineage Plasticity.” He explained bipolar androgen therapy (BAT) as a method intended to improve differentiation. This approach alternates patients between low-androgen states and short bursts of high-dose testosterone. The goal is to limit cellular plasticity and reduce the chances of tumors becoming highly resistant forms like CRPC or NEPC. His research indicates that BAT helps maintain an androgen receptor-dependent epithelial phenotype, slows tumor growth, limits the emergence of more aggressive variants, and fosters an immune environment that helps control tumors. Overall, his findings suggest that epigenetic changes, rather than lasting genetic mutations, play the main role in prostate cancer progression. Dean Tang, PhD (Roswell Park Comprehensive Cancer Center), followed with “Epigenetic Basis and Therapeutic Targeting of Prostate Cancer Heterogeneity and Plasticity.” He talked about how prostate cancer stem cells and castration-resistant tumors arise due

to therapy-induced plasticity. He highlighted miR-34a as a strong inhibitor of PCSC activity and metastatic behavior. Although initial attempts to apply this clinically faced challenges, newer delivery methods for miR-34a using ligands and nanoparticles have shown promising preclinical activity against aggressive, treatment-resistant prostate cancer.

Jung Wook Park, PhD (Duke University), presented "Multi-Functional Neuronal Protein Drives the Growth, Differentiation, and Metastasis of Advanced Prostate Cancer." He discussed how therapeutic pressure from agents like enzalutamide can encourage neuroendocrine differentiation. He identified NPTX1 and HDAC6 as critical regulators of NEPC proliferation, differentiation, and metastatic potential. Both molecules represent promising paths for future treatment development.

Finally, Ana Aparicio, MD (MD Anderson Cancer Center), in "Dissecting the Heterogeneity within Aggressive Variant Prostate Cancers," emphasized the diverse biology of androgen-indifferent tumors. These tumors often harbor defects in *RB1*, *TP53*, and *PTEN* and respond variably to existing treatments. While some patients benefit from platinum chemotherapy or PARP inhibitors, others depend significantly on metabolic adaptations, especially arginine metabolism, to sustain progression and dodge immune responses. Current clinical efforts that target these metabolic weaknesses highlight the urgent need to better categorize aggressive variants and personalize treatments accordingly.

## PLENARY SESSION VI: NOVEL AND EMERGING THERAPEUTIC APPROACHES IN UROLOGICAL RESEARCH

Isaac Kim, MD, PhD (Yale University), in "Leveraging the Surgical Trial in Metastatic Prostate Cancer Research," reflected on his career, mentorship, and the development of his department while stressing the importance of combining surgery with translational research. He discussed the challenges of treating *de novo* metastatic prostate cancer, noting that despite advancements in systemic therapy, survival improvements remain modest. Drawing on his clinical experience, he emphasized that cytoreductive radical prostatectomy can be safe and potentially beneficial for select patients, particularly those with low metastatic burden. This sometimes yields deep PSA responses or lasting disease control. Ongoing

phase I and randomized trials are assessing the impact of combining surgery with standard therapies to identify which patients benefit most. Early results suggest potential improvements in local control, quality of life, and possibly survival, although careful patient selection remains critical.

Kexin Xu, PhD (University of Virginia), in "Targeting RNA Methylation for Castration-Resistant Prostate Cancer," presented new evidence that M6A RNA modifications play a significant role in prostate cancer progression and resistance. These modifications regulate key genes related to androgen signaling and tumor growth. Manipulating this pathway in preclinical models slows cancer growth, making RNA methylation machinery a promising target for therapy.

Roberto Pili, MD (University at Buffalo), in "Integrating Dietary Interventions with the Treatment of Urological Malignancies," discussed how nutrition affects cancer risk, tumor biology, and treatment response. He shared data showing that cutting down on animal protein or using caloric or time-restricted diets changes tumor metabolism, hormone signaling, and immune responses. Clinical studies suggest that dietary changes can improve treatment tolerance, increase therapeutic effectiveness, and may enhance long-term outcomes, supporting diet as a helpful addition to standard cancer treatments.

## POSTER SESSION #1 AND #2

On November 14 and 15, 2025, the conference featured evening poster sessions with a total of 88 selected presentations. Among these, Vrunda Satsiya presented our ongoing research titled "Characterization of FAM120A as a Novel Effector in the Progranulin/EphA2 Axis in Bladder Cancer." In her study, she showed that FAM120A acts as a new progranulin-dependent EphA2 interactor in bladder cancer and contributes to increased cellular motility, invasion, clonogenicity, spherogenesis, anchorage-independent growth, and *in vivo* tumor progression. Additionally, FAM120A is upregulated in bladder cancer tissues compared to normal tissues, suggesting it may serve as a biomarker for bladder cancer.

## CONCLUSIONS

These presentations highlight the growing complexity of urological diseases and the rapid progress in how

they are studied and treated. Research on prostate cancer is uncovering how lineage plasticity, neuroendocrine differentiation, metabolic changes, and epigenetic alterations drive resistance. New therapeutic strategies, including immunotherapy, RNA-modifying targets, and dietary interventions, are emerging in response. Innovative technologies such as organ-on-chip systems, spatial epigenomics, AI-assisted diagnostics, and single-cell atlases are offering deeper insights into tissue biology, microenvironment interactions, and individual patient differences. Beyond cancer, new tools are enhancing our understanding of bladder mechanics and the role of the microbiome in recurrent UTIs. This collective work emphasizes the importance of interdisciplinary collaboration and tailored approaches to improve outcomes across urology.

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## COMPLIANCE WITH ETHICAL STANDARDS

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### Conflict of interests

The Authors have declared no conflict of interests.

### Availability of data and materials

The data underlying this article are available in the article.

### Author's contributions

VS attended the meeting and drafted and revised the manuscript. MNT revised the manuscript. AM provided the guidance and revised the manuscript. AG provided funds to attend the meeting and revised the manuscript.

### Ethical approval

N/A.

### Publication ethics

#### *Plagiarism*

Authors declare no potentially overlapping publications with the content of this manuscript and all original studies are cited as appropriate.

#### *Data falsification and fabrication*

All the data correspond to the real.