



2025 SAU Annual Meeting Report: Unraveling the Molecular Complexities of Urologic Malignancies

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The 2025 Society of Academic Urologists (SAU) annual meeting was held during Jun 15-18, 2025, and showcased the latest discoveries in urologic oncology with a particular emphasis on prostate cancer biology, epigenetic regulation, immunotherapy, and novel therapeutic targets. Featuring an exceptional lineup of keynote speakers, scientific symposia, and interactive forums, the meeting provided a dynamic platform for the exchange of ideas among researchers, clinicians, and trainees dedicated to advancing urologic science and improving patient outcomes.

Opening Session: Keynotes on Epigenetics and Transcriptional Reprogramming

The conference commenced with an evening reception and opening remarks by Dr. Benyi Li (University of Kansas Medical Center), who welcomed attendees and set the tone for a highly engaging program. The keynote session featured two pioneering researchers.

Dr. Qi Cao (Northwestern University) delivered a compelling keynote on the non-canonical function of EZH2 in prostate cancer. He presented data showing that EZH2, widely known as a histone methyltransferase, also directly interacts with the RNA-editing enzyme ADAR1. This interaction modulates A-to-I RNA editing, thereby influencing transcript stability and oncogenic signaling. Through its ability to reshape substrate selectivity of ADAR1 and suppress mRNA degradation, EZH2 emerges as a pivotal regulator of post-transcriptional gene expression. Notably, ADAR1 depletion sensitized prostate tumors to EZH2-targeted therapies, suggesting a novel combinatorial strategy for treating advanced prostate cancer [1, 2].

Dr. Qianben Wang (Banks Anderson, Dr. Distinguished Professor of Pathology at Duke University) introduced another keynote speaker, Dr. Changmeng Cai (University of Massachusetts Boston). Dr. Cai's presentation focused on the role of super-enhancers in defining molecular subtypes of metastatic castra-

tion-resistant prostate cancer (mCRPC). His team developed the Super-Enhancer Analysis for Lineages (SEAL) pipeline to classify five distinct SE programs. Among these, the AR-1 and AR-2 subtypes were associated with epithelial-to-mesenchymal transition (EMT) and metabolic reprogramming, respectively. TWIST1 and HNF1A were identified as key transcriptional drivers, with HNF1A promoting a hepatocyte-like, pro-metastatic state. These findings not only deepen our understanding of CRPC heterogeneity but also nominate actionable targets for therapeutic development [3-5].

Dr. Jianfeng Xu, MD/PhD (Endeavor Health Evanston), presented a special lecture with the title of "Germline Testing in Prostate Cancer: An Evidence-Based Approach Within and Beyond NCCN Guidelines". Approximately 50% of prostate cancer (PCa) patients meet the National Comprehensive Cancer Network (NCCN) guidelines for germline testing at diagnosis. However, the specific genes recommended for testing, their various clinical utilities, and the supporting evidence remain poorly defined in clinical adoption and complicate the interpretation of results. Additional genes and SNPs that are not included in the NCCN guidelines may also provide clinical value for the management of prostate cancer patients. In this presentation, Dr. Xu reviewed current NCCN guidelines for germline testing in PCa, including their intended clinical applications, such as identifying potential genetic causes, assessing familial risk, predicting disease prognosis, and informing treatment response [6]. He also introduced novel PCa-associated genes recently identified but not yet incorporated into current guidelines and discussed the role and limitations of polygenic risk scores in germline testing. In addition, he described GoPath ProstateNow-v2, a clinical-grade and CLIA-certified germline test that includes all NCCN-recommended genes, as well as PRS and recently discovered novel PCa genes.

Session 1: Epigenetic Regulation and Cellular Plasticity in Prostate Cancer

It was co-chaired by Chunhong Yan (Augusta) and Zhenbang Chen (Meharry Medical College), explored the multifaceted roles of chromatin and RNA modifications in tumorigenesis. Dr. Yang Yi (Northwestern University) reported on the crosstalk between EZH2 and m6A RNA methylation in driving prostate cancer progression. His data revealed that EZH2 activates an autoregulatory loop via YTHDF1, reinforcing a hyper-m6A landscape that supports tumor growth. Combining EZH2 and m6A inhibitors yielded synergistic therapeutic effects in preclinical models. Dr. Di Zhao (MD Anderson Cancer Center) presented mechanistic insights into how histone methyltransferase ASH1L primes the metastatic bone niche. His work demonstrated that ASH1L rewires H3K4 and H3K36 methylation to cooperate with HIF-1 α , upregulating IGF-2 and reprogramming infiltrating monocytes into lipid-associated tumor-associated macrophages (LA-TAMs). These LA-TAMs sustain a pro-tumoral, anti-inflammatory microenvironment. Targeting the ASH1L-HIF1 α axis attenuated metastasis, offering a new epigenetic target for intervention [7]. Dr. Housheng Hansen He (University of Toronto) presented a pan-omic study of m6A dysregulation in primary prostate tumors. Germline polymorphisms were shown to influence m6A deposition, which in turn modulated disease aggression and metastatic potential. VCAN emerged as a key m6A-modified transcript linked to poor prognosis [8].

Session 2: Novel Targets and Lineage Plasticity in GU Cancers

It was chaired by Pengbo Zhou (Cornell) and Jason Luo (Johns Hopkins), highlighted lineage-specific vulnerabilities in prostate cancer. Dr. Dean Tang (Roswell Park) described a population of slow-cycling prostate cancer cells (PSCCs) enriched for stem-like features and inherent therapy resistance. Using trigenic mouse models and transcriptomic analysis, his team demonstrated that PSCCs are primed for epithelial-mesenchymal transition and preferentially survive androgen deprivation and chemotherapy [9]. Dr. Dingxiao (Jerry) Zhang (Hunan University) provided a complementary perspective on the cell-of-origin in aggressive PCa. His work dissected the basal and luminal lineage hierarchies, identifying luminal progenitors as a potential source of CRPC. He also described AR+NE- transdifferentiated subtypes sensitive to SP1 or translation inhibitors [10]. Dr. Yanquan Zhang (University of Kentucky) introduced PLK1 as a novel driver of neuroendocrine

prostate cancer (NEPC). PLK1-mediated phosphorylation of ASCL1 and NEUROD1 influenced protein stability and transcriptional activity. PLK1 inhibition suppressed NEPC gene programs, nominating it as a promising therapeutic target [11]. Dr. Meng Zhang (Emory University) discussed the emerging role of microproteins (<100 amino acids) in therapy resistance. Using ribosome profiling and transcriptomic data, she identified MMP24-AS1 as an ARSI-regulated microprotein critical for mCRPC cell survival [12].

Session 3: Novel Targets in GU Cancers

Chaired by Jing-tang Dong (SUSTech China) and Dinglan Wu (CUHK), this session covered several new targets in prostate cancer. The session started with a presentation by Dr. Jing-tang Dong, who described crosstalk between ZFX3, AR, and ER in the regulation of prostate tumorigenesis, followed by Dr. Weiran Feng (Fox Chase Cancer Center), who showed cell context dependency and vulnerabilities of ERG-driven prostate cancer [13]. Dr. Dinglan Wu discussed targeting the LRG1 protein in prostate cancer bone metastasis and its potential as a liquid biopsy biomarker [14]. Dr. Houjian Cai (U of Georgia) demonstrated to use of extracellular vesicles as a vehicle to deliver CRISPR machinery for targeting castration-resistant prostate cancer [15]. Dr. Kaifu Chen (Children's Hospital at Boston) described how to map RNA modifications in prostate cancer by AI analysis of Nanopore sequencing data [16]. The session was ended by a short presentation by Dr. Xintao Qiu (Harvard Medical School) to demonstrate how to detect small cell transformation in patients through epigenomic cfDNA profiling [17].

Session 4: Bioinformatics, AI, and New Technologies

Chaired by Xuesen Dong (UBC) and Hsin-Sheng Yang (University of Kentucky), this session showcased the integration of computational biology and experimental therapeutics. Dr. Li Liu (UT Southwestern) evaluated vascular-disrupting agents (VDAs) in kidney cancer models, highlighting their synergy with immunotherapies [18]. Dr. Zheng Xia (OHSU) presented a TCGA pan-cancer analysis linking AR activity with reduced immune cell infiltration and interferon-gamma signaling. Post-enzalutamide biopsies from mCRPC patients showed increased immune cell abundance, implying that AR inhibition may restore tumor immunogenicity. Dr. Changsheng Zhao (Emory) used Nanopore long-read sequencing to map structural variants and transposable elements in prostate cancer, revealing new layers of genome

complexity [19]. Dr. David Y. Zhang (SUNY Downstate) introduced AI-powered digital pathology platforms to enable spatial analysis, prognosis prediction, and molecular annotation in urologic cancers [20]. Dr. Wenliang Li (UTHS Houston) demonstrated how to identify and target novel kinase regulators for NEPC and cancer metastasis [21].

Session 5: Novel Therapeutic Targets in GU Cancer

It was chaired by Ming Chen (Duke) and Shang Su (LSU) and further delved into new targets. Dr. Zhiping Wang (Lanzhou University) described engineered oncolytic adenoviruses for bladder cancer, with tissue specificity and enhanced efficacy through combination with cisplatin or radiotherapy [22-24]. Dr. Jianhua Xiong (Emory) highlighted acetate as a regulator of EMT and T cell differentiation [25-28]. Dr. Jinghui Liu (Kentucky) revealed a synthetic lethal interaction between NMD components and HSP90 α , with dual inhibition showing strong anti-tumor effects [29]. Dr. Zongwei Wang (Harvard/BIDMC) reported that SRD5A2 expression correlates with prostate volume and methylation status, suggesting implications for individualized BPH treatment [30].

Session 6: Tumor Microenvironment and Immunotherapy

It was co-chaired by Su Deng (Yale) and Kexin Xu (Virginia). Dr. Zhou Wang (University of Pittsburgh) discussed the modulation of androgen signaling in the context of BPH-associated inflammation, highlighting opportunities for therapeutic intervention [31]. Dr. Wang's findings suggest that low-grade prostatic inflammation could enhance the expression of androgen-responsive genes in BPH, indicating an important role for prostate inflammation in BPH pathogenesis. Dr. Zhu Wang (UC Santa Cruz) and Dr. Chin-Lee Wu (MGH/Harvard) provided critical insights into cell-of-origin models in bladder cancer [32, 33] and pathological characterization of prostate cancer subtypes, respectively [34-36]. In particular, Dr. Wang's work supports the notion that urothelial basal cells are more competitive than intermediate or umbrella cells in producing bladder tumors and are likely the cells of origin for the more aggressive bladder cancer subtype. Dr. Wu described lessons learned from the unusual pathological features.

Session 7: Cancer Etiology, Progression, and Metastasis

Co-chaired by Yuanyuan Zhang (Wake Forest) and Xiaohong Li (U of Toledo), this session addressed the molecular heterogeneity and inflammatory signaling in urologic tumors. Dr. Sean Li (Cedars-Sinai Medical Center) explored why males

sex are more susceptible to cancers and presented solid evidence to show the biasing effects of the sex hormones with androgens increase bladder cancer risk through both tumor cell-intrinsic and extrinsic mechanisms [37, 38]. Dr. Shuai Gao (New York Medical College) showed how histone lactylation directs a distinct enhancer landscape to drive prostate cancer progression [5, 39]. While Dr. Jianmin Zhang (University of Toledo) focused on dissecting molecular mechanisms of breast cancer metastasis, Dr. Hai Wang (Roswell Park Cancer Center) described the metabolic symbiosis between tumor and bone cells in prostate cancer bone metastasis [40, 41]. Specifically, Dr. Zhang's work provides mechanistic insight into the importance of TAZ-CAMK1D-CTTN-mediated actin cytoskeletal regulation during breast cancer metastasis. Dr. Wang's work highlight MCT1 as a critical metabolic vulnerability in prostate cancer metastasis and suggest that targeting lactate uptake may offer a promising therapeutic strategy for this lethal disease.

Session 8: Therapy Resistance in Cancer

Co-chaired by Jason Liu (UTHSCSA) and Xuefeng Liu (OSU), this session focused on various drug resistance mechanisms. After Dr. Zhiguo Li (U of Kentucky) presented a new mechanism for docetaxel resistance [11], Dr. Feng Yang (Baylor College of Medicine) showed how COP1 regulates cellular response to taxane in prostate cancer [42]. Dr. Su Deng (Yale) demonstrated how to target lineage plasticity drivers to overcome therapy resistance in prostate cancer [43, 44]. Dr. Jianneng Li (Notre Dame University) described an unexpected mechanism of enzalutamide anti-tumor activity in prostate cancer [45]. Dr. Luksana Chaiswing (University of Kentucky) showed how to promote mitochondrial vulnerability with redox state modulating agents to enhance the efficacy of radiotherapy [46, 47]. Finally, Dr. Jia Li (Harvard Medical School) explored new synergies with PARP inhibitors in prostate cancer [48].

Career Development and Closing Forum

The evening professional development forum, chaired by Drs. Xiaohong Li and Li Liu featured a keynote by Dr. X. Shirley Liu (GV20 Therapeutics). Her presentation outlined GV20's AI-powered STEAD platform for antibody discovery. Their lead candidate, GV20-0251, an anti-IGSF8 monoclonal antibody-was developed through AI-guided selection and demonstrated monotherapy efficacy in early trials. This approach exemplifies the integration of computational prediction and biologics development. Roundtable discussions addressed career transitions, dual-career family dynamics, and strategies for becoming a rising star in academic research [49].

Keynote Presentation: Uncovering the Hidden Genetics of Disease Through Tandem Repeats

Dr. Qianben Wang (Banks Anderson, Dr. Distinguished Professor of Pathology at Duke University) introduced the third keynote speaker Dr. Wei Li, the Grace B. Bell Endowed Chair and Professor of Bioinformatics at the University of California, Irvine. Dr. Li delivered a visionary plenary lecture highlighting the overlooked but critical role of tandem repeat (TR) variations in human disease genetics. As a leader in computational genomics and creator of the widely-used MACS algorithm for ChIP-seq, Dr. Li emphasized the transformative potential of TRs-repetitive DNA elements previously neglected in GWAS and clinical diagnostics. His lab's recent efforts led to the development of two landmark resources: **TR-Atlas** [50], profiling ~1 million TRs from 340,000 individuals, and **TR-xQTL** [51], establishing the functional regulatory landscape of TRs across the human genome. Leveraging these platforms, Dr. Li presented compelling unpublished findings linking TR variants to a spectrum of complex and rare diseases-including cardiovascular disorders, neurodevelopmental anomalies, Alzheimer's disease, liver pathology, diabetes, and aggressive cancers. Notably, many associations have been experimentally validated, showcas-

ing the clinical utility of TRs in uncovering disease mechanisms and risk genes. This presentation underscored the urgent need to integrate TR analysis into mainstream genomics pipelines and precision medicine efforts.

Conclusion

The 2025 SAU Annual Meeting successfully highlighted the latest advances across epigenetics, lineage plasticity, tumor microenvironment, and emerging therapeutics in urologic oncology. With cutting-edge technologies and a collaborative spirit, the conference reaffirmed the critical role of the SAU community in shaping the future of precision medicine and improving the lives of patients with urologic malignancies.

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