Making Education Easy

28–30 June, 2026

In this review:

Abbreviations used in this review:

BCG = bacillus Calmette-Guérin; **ctDNA** = circulating tumour DNA; **mCRPC** = metastatic castration-resistant prostate cancer; **mHSPC** = metastatic hormone-sensitive prostate cancer; **(N)MIBC** = (non-)muscle-invasive bladder cancer; **OS** = overall survival; **PFS** = progression-free survival; **PSA** = prostate-specific antigen; **QALY** = quality-adjusted life-year; **QoL** = quality of life; **TME** = tumour microenvironment.

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Welcome to our review of the 2026 ANZUP (Australian & NZ Urogenital and Prostate Cancer Trials Group) Annual Scientific Meeting held in Adelaide, Australia.

The 2026 ANZUP Annual Scientific Meeting was held at the Adelaide Convention Centre from 28–30 June under the theme "*What matters most.*" This theme was reflected throughout the programme, which examined not only whether treatments improve cancer outcomes, but also how they affect QoL, function, treatment burden, affordability and equitable access to care. The meeting highlighted the importance of multidisciplinary collaboration across prostate, bladder, kidney, testicular and other genitourinary cancers. Sessions incorporated translational research, biomarker development, real-world evidence, supportive care and consumer perspectives. This review focuses on selected presentations with potential relevance to clinical practice and future research. These include spatial biomarkers in prostate cancer, strategies to reduce BCG-related bladder toxicity, metabolic vulnerabilities in castrate-resistant prostate cancer, use of ctDNA alongside PSA for dynamic prognostication, treatment decision-making based on life expectancy and functional outcomes, financial and health-system consequences of cancer care, emerging biomarkers for selecting prostate cancer treatments, the appropriate use of PSMA-PET and the potential role of Cxbladder Triage Plus in reducing unnecessary cystoscopy. Several findings were preliminary, preclinical or unpublished at the time of presentation, and should be interpreted in that context.

I trust that you will find this review clinically valuable, and I look forward to your feedback. The programme handbook and abstracts are available online here.

Kind Regards,

Dr Umbreen Hafeez
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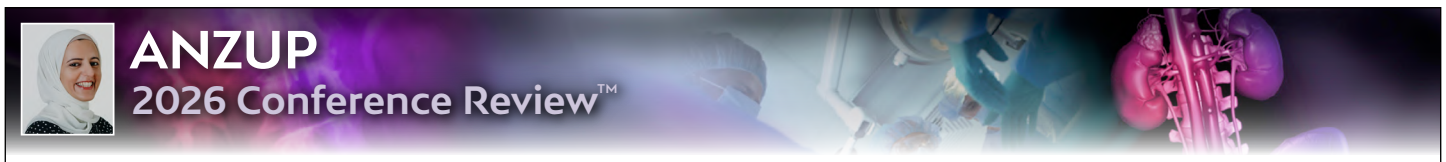
Spatial single-cell proteomics defines multicellular niches in primary prostate cancer

Speaker: Professor Marianna Kruithof-de Julio (Department for BioMedical Research, Department of Urology, Inselspital, Bern, Switzerland)

Summary: This study examined whether the spatial organisation of epithelial, stromal and immune cells could provide prognostic information not captured by conventional histopathology. The analysis included 195 patients, 34 protein markers and approximately 2.1 million individual cells. 18 recurrent multicellular niches were discovered. Three major biological insights were identified. An ERG-positive, p53-positive tumour epithelial state, that was independently associated with worse PFS, not explained by Gleason score. A cumulative score incorporating three immune-rich niches also stratified survival, suggesting that the overall burden of coordinated immune architecture may be more informative than any single immune-cell population. A separate peri glandular myCAF CD105-high niche was associated with worse PFS, while the abundance of these cells without their spatial context was not prognostic.

Comment: This work illustrates an important transition from describing which cells are present to understanding how those cells are engaged. The finding that the same stromal cell can have different prognostic implications depending on its cellular neighbourhood, is biologically plausible. The study remains exploratory. It used a fixed 34-marker panel, and a localised disease cohort with relatively few events. Before it can be used clinically, it will require simplification into assays that can be applied to routine pathology, potentially through H&E-based AI models. Nevertheless, spatial biomarkers may eventually improve risk assessment beyond the Gleason score.

Translational Highlights Session



Independent commentary by Dr Umbreen Hafeez

Dr Umbreen Hafeez is a medical oncologist and clinician scientist at the Olivia Newton-John Cancer and Research Centre in Melbourne. Dr Hafeez's translational research focuses on developing and characterising novel anti-ErbB3 antibodies to combat cancer. She is also passionate about patient-centred care and committed to promoting and enhancing high-quality cancer care. She is a strong supporter of health equity, advocating for equal access to healthcare, treatment outcomes, and opportunities for all individuals, regardless of their background.

Unravelling mechanisms and identifying therapies to relieve BCG induced bladder side effects

Speaker: Dr Luke Grundy (Flinders Health and Medical Research Institute, Flinders University, Adelaide, Australia)

Summary: Intravesical BCG remains an effective treatment for high-risk NMIBC, but urinary frequency, urgency, dysuria and bladder pain can lead to treatment interruption or discontinuation. These symptoms are driven partly by hypersensitisation of bladder sensory nerves. Can we inhibit bladder-innervating sensory nerves to relieve or prevent BCG-induced sensitisation, and improve both QoL and treatment adherence? In mouse models, BCG produces substantial bladder immune-cell recruitment, and increases sensory-nerve firing in response to bladder pressure. BCG-treated animals also demonstrate a lower withdrawal threshold, consistent with heightened pelvic sensitivity. Phenazopyridine, an FDA-approved urinary tract analgesic, reduces afferent nerve firing in a dose-dependent manner. Importantly, the preclinical experiments did not demonstrate a major adverse effect on BCG-related direct tumour-cell cytotoxicity or the overall immune-cell response within the bladder. These findings have led to the RELIEVE-BCG trial, a randomised study evaluating oral phenazopyridine for BCG-induced lower urinary tract symptoms. Planned outcomes include the EORTC NMIBC24 and QLQ-C30 QoL instruments.

Comment: BCG toxicity is challenging to manage, with its potential to compromise treatment adherence and QoL. The concept of targeting the sensory pathway without suppressing the antitumour immune response is attractive. Reduction in nerve firing in a mouse model may not ensure clinically meaningful improvement in urinary symptoms, treatment completion or oncological outcomes. The RELIEVE-BCG trial is therefore essential. A positive study could provide a simple intervention that helps patients receive an adequate course of BCG, without impairing efficacy.

Translational Highlights Session

Aspartate metabolism is an androgen-regulated vulnerability in castration- and enzalutamide-resistant prostate cancer

Speaker: Professor Jeff Holst (University of NSW, Australia)

Summary: Cancer metabolism is commonly studied using culture media containing nutrient concentrations substantially different from those found in human plasma. However, the conventional RPMI media contain supraphysiological concentrations of several metabolites, including glucose, glutamine, arginine, asparagine and aspartate. When prostate cancer models were examined in human plasma-like medium Plasmax, castrate-resistant/enzalutamide-resistant cells demonstrated substantial changes in aspartate, purine, pyrimidine and urea cycle metabolism. Aspartate availability appeared particularly important because it is critical for purine and pyrimidine synthesis, and links mitochondrial metabolism with the urea cycle. Physiological culture conditions showed a reduction in intracellular aspartate following enzalutamide treatment that was less apparent in conventional media. Collaborative work is undergoing targeting dihydroorotate dehydrogenase with leflunomide, a key enzyme in *de novo* pyrimidine synthesis.

Comment: The most important message is that a drug target identified in nutrient-rich laboratory media may not remain relevant in the physiological environment. Targeting aspartate utilisation is biologically compelling, particularly in castrate-resistant disease. Clinical translation will nevertheless require demonstration of a therapeutic window, as nucleotide synthesis is also essential for normal proliferating tissues.

Translational Highlights Session

Added value of ctDNA testing with primary treatment for metastatic prostate cancer prognosis

Speaker: Professor Gert Attard (Cancer Institute Director, University College London Cancer Institute, England)

Summary: PSA response is strongly prognostic in metastatic prostate cancer, but PSA does not fully capture tumour burden or treatment sensitivity. The aim of this study was to use circulating biomarkers for dynamic monitoring of treatment sensitivity and improve outcomes, to allow targeted interventions. The STAMPEDE data showed that achieving a PSA of ≤0.2ng/mL at 24 weeks was associated with favourable long-term outcomes. In this study, ctDNA positivity at cycles 3 or 4 was associated with shorter OS than ctDNA negativity, with a hazard ratio of 2.72. Patients with both detectable ctDNA and a PSA above 4ng/mL had the poorest outcomes. For prediction of 12-month OS, combining ctDNA and PSA improved the true-positive rate compared with PSA alone.

Comment: Persistent ctDNA despite an apparently satisfactory PSA response may identify patients with biologically-resistant disease. The analyses should not yet be used to switch or intensify treatment outside a trial. Several subgroups were small, confidence intervals were wide, and prognostic association does not prove that changing therapy based on ctDNA will improve survival.

Translational Highlights Session

What matters most: weighing trade-offs in urologic care and aligning treatment decisions with patient's priorities

Speaker: Sarah Psutka (Associate Professor of Urology, University of Washington, US)

Summary: Sarah Psutka highlighted that complex decision-making is now routine across genitourinary oncology. Patients with localised prostate cancer, low-risk prostate cancer, metastatic prostate cancer, small renal masses, high-risk renal-cell carcinoma, MIBC and BCG-unresponsive NMIBC may each face several reasonable treatment options, with differing effects on cancer control, toxicity, function, independence and QoL. Treatment discussions should begin by establishing the relevant time horizon. Estimation of life expectancy provides a framework for determining whether a patient is likely to live long enough to experience the oncological benefit of an intervention, while also considering its immediate burden. The Lee Schonberg Index was presented as a practical tool for estimating medium- and long-term all-cause mortality. The ACS NSQIP calculator can be used as a method of estimating perioperative morbidity and mortality, including outcomes relevant to older and frail patients. Treatment-specific decision aids may go further by simultaneously estimating cancer mortality, other-cause mortality and complications for each management option. The small renal mass model provided a useful example. Using age, sex, BMI, renal function, performance status, Charlson comorbidity index and tumour diameter, the model compared radical nephrectomy, partial nephrectomy, thermal ablation and active surveillance. In the example presented, predicted death from renal cell carcinoma was only 1–2% across treatment strategies, while the risk of death from other causes was 47%. Psutka also emphasised that decision-making should not end once treatment has been selected. The BounceBack programme is prospectively studying mobility, physical function, frailty, resilience and QoL before and after surgery. She also highlighted that standard patient-reported outcome measures may overlook important issues such as falls, cognition, nutrition, independence and financial burden.

Comment: This was a highly practical presentation that reframed treatment selection around a simple but frequently overlooked question: will this patient live long enough, and remain well enough, to benefit from the treatment being proposed? Personalised risk tools can improve shared decision-making, particularly when the expected cancer treatment benefit is small relative to complications or loss of independence. However, prognostic calculators depend on the populations from which they were developed, and may be less accurate when applied to different health systems, ethnic groups or contemporary treatments.

Keynote Session One: What Matters Most? When Outcomes Compete

From costs to consequences: what defines value in healthcare?

Speaker: Dan Lindsay (Group Lead, Cancer Economics, Cancer Council QLD, Australia)

Summary: Dan Lindsay discussed the growing financial burden of healthcare for both patients and the health system. Financial toxicity following a cancer diagnosis is associated with poorer QoL, psychological distress, treatment non-adherence, delays in care and widening health inequities. Healthcare budgets are limited; hence, new treatments must be assessed according to their clinical benefit and cost compared with existing care. Cost-effectiveness analysis commonly uses quality-adjusted life-years (QALYs) and the incremental cost-effectiveness ratio, and approximately AU$50,000 per QALY is commonly used as an Australian benchmark. However, value should also account for disease severity, rarity and equity. The Australian public may place greater value on health gains for disadvantaged, Indigenous and rural populations.

Comment: The presentation highlighted that value in cancer care cannot be judged by clinical efficacy alone. High-value care requires balancing survival and QoL benefits against treatment cost, patient financial burden and the opportunity cost of diverting funding from other services. Cost-effectiveness frameworks are important for sustainable decision-making, but equity must be incorporated so that disadvantaged populations are not further excluded from access to effective care.

Keynote Session One: What Matters Most? When Outcomes Compete

[177Lu]Lu-PSMA-617 treatment of high-risk localized prostate cancer selects clones with increased neuro-endocrine differentiation

Speaker: Professor Paul Neeson (Academic Researcher, Peter MacCallum Cancer Centre, Australia)

Summary: This study investigated Lu-PSMA changes in the tumour microenvironment (TME) and explored the effect of Lu-PSMA on prostate cancer cell differentiation. The investigators observed a reduction in PSMA-positive tumour cells after treatment, accompanied by heterogeneous changes in the tumour inflammation signature. Gene ontology analysis demonstrated enrichment of pathways associated with neuroendocrine differentiation, interferon signalling, IL-6/JAK/STAT3 activity, epithelial–mesenchymal transition and inflammatory responses after treatment. Neuroendocrine gene expression increased within the TME after Lu-PSMA treatment. It was proposed that radioligand therapy may preferentially remove PSMA-high adenocarcinoma cells while selecting survival of PSMA-low, neuroendocrine-like populations.

Comment: The findings provide a plausible explanation of resistance to PSMA-directed radioligand therapy. Loss of PSMA expression and emergence of neuroendocrine features could reduce the effectiveness of subsequent PSMA-targeted treatment, and may explain discordance between PSA, PSMA imaging and clinical progression in some patients. It is not yet clear whether treatment induces lineage plasticity or simply selects pre-existing resistant clones. The study is hypothesis-generating and should not discourage appropriate use of lutetium-PSMA.

Abstract #1

Evaluation of the Plasmalogen Score as a prognostic lipid biomarker in patients with metastatic prostate cancer

Speaker: Liam Dwyer (PhD candidate, University of Sydney, Australia)

Summary: This study evaluated whether variations in plasmalogen scores were associated with survival outcomes across multiple metastatic prostate cancer cohorts, including ENZAMET, an observational mCRPC cohort, IMPROVE and TheraP. Higher plasmalogen scores were associated with longer survival in both hormone-sensitive and mCRPC patients. Patients with a high score appeared to derive greater benefit from lutetium-PSMA, with a median survival of 19.3 months with lutetium-PSMA and 17.5 months with cabazitaxel. In contrast, patients with a low score had a median survival of 13.7 months with lutetium-PSMA and 20.4 months with cabazitaxel.

Comment: A blood-based metabolic score capable of predicting the relative benefit of lutetium-PSMA and cabazitaxel would be clinically valuable. However, prospective validation and standardisation of the assay will be required before it can be used to select treatment or justify de-intensification in clinical practice.

Abstract #24

Beyond the androgen receptor: decoding master regulators of aggressive prostate cancer

Speaker: Natalie L Lister (Research Fellow, Monash University, Australia)

Summary: This study looked at whether high transcription-factor network activity is associated with increased risk of disease progression in mHSPC patients receiving standard-of-care treatments. Gene expression profiles from primary prostate tumours were used to derive 53 transcription-factor network signatures, and their relationship with clinical outcomes was examined. High transcription-factor network activity was associated with an increased risk of progression in patients receiving standard treatment for mHSPC. MYBL2 network activity emerged as an independent adverse prognostic marker across the CHAARTED and ENZAMET datasets. Authors demonstrated that MYBL2 was required for growth and survival in prostate cancer organoid models. This study identified MYBL2 not only as a prognostic biomarker, but also as a potential therapeutic target.

Comment: Aggressive cancer is unlikely to be driven by a single dominant pathway in every patient. MYBL2 is an attractive target. However, transcription factors have traditionally been difficult drug targets.

Abstract #9

Earn CPD

Royal Australasian College of Physicians (RACP) MyCPD participants can claim the time spent reading and evaluating research reviews as CPD in the online MyCPD program. Please contact MyCPD@racp.edu.au for any assistance.

Royal Australian & New Zealand College of Radiologists (RANZCR) members can claim reading related to their practice as a CPD activity under the category 'journal reading and web based no certificate *reflection required'. More info.

Nursing and Midwifery Board of Australia (NMBA) Journal reading and watching videos (including Research Reviews') may be considered a self-directed activity set out in the NMBA Registration Standard: Continuing Professional Development. One hour of active learning will equal one hour of CPD. Details at NMBA CPD page.

PSMA-PET use in low to favourable intermediate risk prostate cancer patients

Speaker: Mrunal Hiwase (PhD student/Urology registrar, Adelaide University, Southern Adelaide Health Network, Australia)

Summary: This real-world analysis used the statewide INSPIRATION repository to examine PSMA-PET staging in patients with low- or favourable-intermediate-risk prostate cancer. Among 1690 publicly-imaged patients, 894 had undergone PSMA-PET for initial staging. The study identified 107 patients with low- to favourable-intermediate-risk disease. Twelve had PSMA avid findings outside the prostate, but only one represented confirmed metastatic disease. The remaining findings produced a false-positive rate of 91.7% among patients with an extra-prostatic abnormality. False-positive findings prompted multidisciplinary review and additional investigations or treatment changes. Overall changes to treatment happened in approximately 4.7% of the cohort. The estimated cost of scanning, investigation and altered management was AU$142,352, with unclear or limited overt benefit.

Comment: PSMA-PET has transformed staging in higher-risk prostate cancer, but greater sensitivity does not mean that indiscriminate use is beneficial. In populations with a very low pre-test probability of metastatic disease, false-positive results can outnumber true-positive findings and lead to anxiety, biopsies and unnecessary treatment changes.

Abstract #50

The AUSSIE study: preliminary evaluation of Cxbladder Triage Plus for urothelial carcinoma detection in patients with haematuria

Speaker: Weranja Ranasinghe (Urologist, Monash Health and Monash University, Australia)

Summary: The AUSSIE study prospectively evaluated Cxbladder Triage Plus in an independent Australian cohort referred for haematuria assessment. Among 691 evaluable patients, 424 had visible haematuria and 267 had non-visible haematuria. At the prespecified 0.15 cut-point, sensitivity for urothelial carcinoma was 89%, specificity 79%, PPV 27% and NPV 99%. In patients with visible haematuria, sensitivity was 89%, specificity 77%, PPV 31% and NPV 98%; in patients with non-visible haematuria, sensitivity was 90%, specificity 83%, PPV 17% and NPV 100%. There were six false-negative results: five low-grade Ta tumours and one urachal adenocarcinoma. The adenocarcinoma was detected on imaging. Based on the proposed pathway, ~73% of patients could potentially avoid cystoscopy. Compared with American Urological Association risk criteria, Triage Plus produced a 3.4-fold increase in PPV. Its AUC was 0.90 compared with 0.84 for the earlier Triage assay.

Comment: Cystoscopy is invasive, resource-intensive and low-yielding in many patients with haematuria. A test with a 99% NPV could substantially reduce unnecessary procedures, particularly when combined with appropriate imaging and safety-net follow-up. The principal concern is the consequence of a false-negative result. Most missed tumours were small, low-grade lesions, but one urachal adenocarcinoma was also missed. Longitudinal follow-up and health-economic evaluation are needed before widespread implementation.

Abstract #59