

Conference Report

B2B: Five Practice-Changing Advances on the Horizon Summary

Simon Tanguay ^{1,*†}, Kilian M. Gust ², Axel Bex ^{3,4}, Robert Jones ⁵ and Peter C. Black ^{6‡}

¹ Department of Surgery, Division of Urology, McGill University, Montreal, QC H4A 3J1, Canada

² Department of Urology, Comprehensive Cancer Center, Medical University of Vienna, 1090 Vienna, Austria; kilian.gust@meduniwien.ac.at

³ Department of Urology, University College London, London W1G 8PH, UK

⁴ Department of Urology, The Netherlands Cancer Institute, 1006 BE Amsterdam, The Netherlands

⁵ School of Cancer Sciences, Beatson West of Scotland Cancer Centre, University of Glasgow, Glasgow G12 0YN, UK

⁶ Department of Urologic Sciences, University of British Columbia, Vancouver, BC V6T 1Z4, Canada

* Correspondence: simon.tanguay@mcgill.ca

† Co-Chair, B2B Scientific Programme Committee.

The 7th Bench-to-Bedside Uro-Oncology: GU Cancers Triad Meeting, organized in conjunction with the 45th Annual Congress of the Société Internationale d'Urologie, was held on 31 October 2025, in Edinburgh, Scotland, and transmitted live on the *SIU@U* Congress app. The fourth session, on the five practice-changing advances in genitourinary cancers, took place in the afternoon and was moderated by Dr. Simon Tanguay (Canada) and Dr. Peter C. Black (Canada).

First, Dr. Kilian M. Gust (Austria) spoke about the 5 practice-changing advances in bladder cancer (BCa). In the last 10 years, he pointed out, much progress has been made into developing strategies for local and distant staging, as well as novel therapies, although there is an ongoing need for additional molecular markers to better direct treatment selection. Ultimately, he said, all of these advances may lead to better strategies to evaluate patients for clinical complete response (cCR) and result in the development of a true bladder-sparing approach.

Since 2018, magnetic resonance imaging (MRI) has been used for local BCa staging, for the initial diagnosis as well as for disease monitoring [1]. It will likely become a cornerstone for the evaluation of localized bladder tumours. An algorithm for evaluating MRI images of bladder tumours is available [1], and it will be important for urologists to learn how to read these images.

Evaluating tumour response to therapy can be accomplished not just by examining the tumour but also through biomarkers. An example of a biomarker-driven approach was seen in the SunRISe-4 trial. This was a phase 2 trial in which patients with muscle-invasive BCa (MIBC) were randomized to neoadjuvant TAR-200 intravesical therapy with gemcitabine plus cetrelimab—or cetrelimab monotherapy. Subsequently, all patients underwent radical cystectomy (RC) [2]. This trial used urinary tumour DNA (utDNA) to monitor response to therapy, representing a new age of biomarker-driven trials. When patients responded to therapy, utDNA diminished. More importantly, >80% of patients with no visible tumour in their bladder remained utDNA-positive. It appears that utDNA is a useful disease marker because 81.5% of patients who achieved a pathological CR (pCR) at week 12 were utDNA-negative, compared with 21.2% who were utDNA-negative but did not achieve pCR [3].

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license.

Fluorodeoxyglucose (FDG) positron emission tomography (PET)/computed tomography (CT) is not an ideal approach for distant staging of MIBC because of the potentially high rate of false positive lesions that may lead to unnecessarily aggressive therapy. As demonstrated in the PURE-1 trial, FDG PET/CT has a high specificity but low sensitivity for detecting bladder tumours, which does not justify its routine use in practice [4]. However, novel imaging modalities are being developed. These include NECTIN4 targeting PET, fibroblast activation protein inhibitor PET (which examines the tumour microenvironment), and PD-1/PD-L1 PET/CT. These methods may even aid in the detection of metastatic disease [5].

Another promising biomarker is circulating tumour DNA (ctDNA). Tumour-naïve ctDNA testing is useful for “quick and dirty” screening but has a low specificity. Conversely, tumour-informed ctDNA testing is more specific but has a higher cost [6]. In the IMvigor011 trial, patients were monitored with ctDNA testing every 6 weeks, and results helped determine whether patients received adjuvant therapy [7]. Unfortunately, cost remains a major hurdle to bringing this technology into the clinical setting. The cost may be offset, however, by avoiding overtreatment of patients.

Several recent trials have investigated novel therapies for non-muscle-invasive BCa (NMIBC), including TAR-210 for intermediate-risk disease (MoonRISe-1) [8], TAR-200 in bacillus Calmette-Guérin (BCG) unresponsive NMIBC (SunRISe-1) [9], TAR-200 in BCG-naïve NMIBC (SunRISe-3) [10], and TAR-200 in BCG-relapsed NMIBC [11]. In the MIBC setting, SunRISe-2 [12] and SunRISe-4 [13] are again exploring the efficacy and safety of TAR-200. Other promising approaches are combination therapies of immune checkpoint inhibitors (ICI) with BCG in high-risk NMIBC [14–17]. While some improvements were observed, it is yet uncertain if this approach can be brought into clinical practice.

For MIBC, the field is advancing quickly, with novel combinations, such as durvalumab with gemcitabine–cisplatin demonstrating the advantages of systemic therapy in the neoadjuvant or perioperative setting. Several ongoing trials in this setting, including KEYNOTE-866 (NCT03924856), ENERGIZE (NCT03661320), and EV-304/KEYNOTE-B15 (NCT04700124) in cisplatin-eligible patients as well as EV-303/KEYNOTE-905 (NCT03924895) and VOLGA (NCT04960709) in cisplatin-ineligible patients, may again change the current standard of care for MIBC. A dramatic improvement of pCR has already been observed, from historical rates of about 10% with transurethral resection of bladder tumour (TURBT) alone to up to 60% in the EV-303/KEYNOTE-905 trial [18]. Response rates that high raise the possibility of sparing the bladder and keeping patients on maintenance therapy. In metastatic BCa, several novel combination therapies have also been investigated [19–21], of which the combination of enfortumab vedotin (EV) plus pembrolizumab is practice-changing in this setting [19].

For several years, PD-L1 was investigated as a potential marker to drive treatment selection for metastatic urothelial carcinoma (mUC). While PD-L1 is probably not an ideal marker in the mUC setting, other markers under investigation have started to show promise. FGFR3 has been implemented in mUC, but it is now being targeted in NMIBC for the first time. NECTIN4 is being targeted with EV, and some antibody–drug conjugates (ADCs) are targeting TROP2 and HER2. When monitoring these biomarkers, it remains unclear what to look for, with options that include protein level and expression [22].

The development of novel biomarkers leads to novel therapies, such as the combination of disitamab-vedotin plus toripalimab, which targets HER2, in HER2-positive mUC [23], with results similar to those seen with EV–pembrolizumab in the first-line setting [19].

An attractive and effective way to deliver chemotherapy is via ADCs. The challenge is to select the correct payload for each target, using the correct linker. Despite the straightforward delivery system to the tumour cell, unexpected side effects have emerged.

Today, there is real potential for a true bladder-sparing approach. Local staging with TURBT, MRI, and utDNA as well as distant staging with novel PET/CT or ctDNA could potentially support a move beyond the current treatment paradigm, in which patients with MIBC are selected for chemotherapy/radiation or RC, to one in which these patients undergo one of many systemic therapy options. Good responders with no evidence of tumour on imaging and negative ctDNA biomarkers could potentially be spared RC and remain under active surveillance or maintenance therapy instead. Patients who have a local tumour with no metastatic disease after systemic therapy may require salvage therapy or RC, if the disease is muscle invasive. For patients who can be downstaged to NMIBC, TURBT or intravesical therapy with novel devices such as TAR-200/210 might be considered as a bladder-sparing approach. Patients with true metastatic disease, with or without bladder tumours, following systemic therapy could undergo salvage systemic therapy and may not reach the point where RC is necessary.

During the Q&A session, Dr. Black noted that the bladder-sparing approach proposed by Dr. Gust will require clinical trials, and such trials are already ongoing. He also asked about the recommended management of patients with a pCR who remain utDNA-positive or patients who did not attain a pCR but were utDNA-negative. Dr. Gust replied that this is evidence of how no single test is 100% reliable, but it is promising that these markers of response are usually concordant. Currently, however, multiple tests are still necessary. An audience member discussed his experience with the BladderPath trial, where patients were randomized to MRI or TURBT for staging of new BCa. Patients assigned to MRI got to treatment faster and had better survival [24]. The surgeons involved in the trial indicated that they did not always perform a full TURBT if they knew the patient had MIBC, suggesting that better imaging can result in less need for full TURBT. He also noted that patients who do not respond to neoadjuvant chemotherapy have some of the worst outcomes, and he suspects that clinical trials exploring novel therapies will show a similar pattern.

Next, Dr. Axel Bex (the Netherlands) presented on the 5 advances in kidney cancer (KCa). The first advance was the use of stereotactic body radiotherapy (SBRT) for renal masses. The TROG 15.03 FASTRACK II trial demonstrated a local control rate of 100% at 1 year with SBRT among 70 patients with inoperable primary KCa [25]. Despite the extremely positive results, these findings have been heavily debated, he said, as it is unclear whether radiologists can demonstrate conclusively that the tumour is really gone. As the trial matures and new studies are added, however, there is the possibility that SBRT will provide a local treatment alternative to systemic therapy.

Dr. Bex then discussed the use of semi-automated robotic systems and telesurgery. In the United Kingdom, it has been predicted that by 2035, 90% of surgeries will be key-hole surgeries performed with robotic assistance [26]. This will be assisted with 3-D virtual modeling [27,28]. Dr Bex predicted that in the next few years, some of these procedures may be semi-automated. In 2024, Bordeaux-based surgeons performed kidney surgery on a patient located in Beijing, with a latency of 131.9 milliseconds [29]. The potential to treat patients without moving them over large distances to centres with high levels of expertise could widely expand the population of patients with access to these procedures in highly skilled hands.

Tools for better assessment of risk and minimal residual disease (MRD) was the next advance Dr. Bex described. Understanding of how KCa progresses has improved over the past few years. One of the hallmarks is the loss of 9q and 14q chromosomes [30]. At the Crick Institute, researchers are currently evaluating whether biopsy results are representative enough in the small renal masses to identify patients who are good candidates for active surveillance [31]. This remains a missing element in decision-making.

There is currently a strong focus on KIM-1, which has been shown to have prognostic value following nephrectomy. A low baseline KIM-1 level following surgery has been linked with a reduced risk of recurrence [32]. A recent trial also suggested ctDNA could be a helpful biomarker in KCa. Patients were treated with serial SBRT for oligometastatic disease. ctDNA was measured in patients at baseline and after 3 months of therapy. Baseline ctDNA molecular residual disease was associated with shorter systemic therapy-free survival (median 28.1 months [95% confidence interval (CI) 23.5–53.1] vs. 54.1 months [95% CI 35.4–not estimable], with a hazard ratio (HR) of 2.75 [95% CI 1.29–5.86]) [33]. Similarly, when ctDNA was higher after therapy, the risk of recurrence was greater [33].

Artificial intelligence is now being used for risk assessment. The UroPredict machine learning model has shown promise in research undertaken in France. The investigators fed standard clinical factors into the system, resulting in a highly predictive model, with an area under the curve of ≥ 0.8 [34]. In another study undertaken in China, researchers calculated a multimodal recurrence score by combining the Leibovich score, a 6 single nucleotide polymorphism score, and a histopathological whole-slide image-based score using deep learning allied to digital scanning of tumour tissue sections [35]. For T3 tumours selected on the basis of the eligibility criteria for adjuvant therapy with pembrolizumab in the KEYNOTE-564 trial, the multimodal recurrence score was highly predictive of recurrence (HR 5.59, 95% CI 3.12–10.04) [35]. While this particular algorithm may not see widespread clinical use, Dr. Bex said that it demonstrates a proof of concept.

The presence of residual disease may also be detected via molecular imaging. Most of the trials on molecular imaging have been conducted in small non-metastatic renal masses, with one showing a sensitivity of 85.5% and specificity of 87.0% using [^{89}Zr] Zr-girentuximab PET/CT imaging [36]. Nevertheless, said Dr. Bex, there were some signals in these trials that molecular imaging could also be used to identify metastasis, and this is currently under investigation in an ongoing trial. Novel imaging modalities are being developed in China and PET imaging sensitivity is also improving.

The next advance discussed by Dr. Bex was the development in metastasis-directed therapy. In a single-arm study of 121 patients with oligometastatic clear-cell renal cell carcinoma (ccRCC) with ≤ 5 metastases treated with serial SBRT alone, median progression-free survival (PFS) was relatively short at 17.7 months (95% CI 14.9–22.4), which is similar to surveillance. However, median systemic therapy-free survival time was 34.0 months (28.3–54.1 months). According to Dr. Bex, this solidifies the role of radiotherapy for this patient group, who are very likely to experience disease recurrence [33]. He expects further trials of SBRT in this setting to take place soon.

The final advance that Dr. Bex discussed was personalizing systemic therapy. He said this is important given that we have reached a ceiling with the currently available therapeutic agents. While he has hope for doublet therapy, Dr. Bex does not expect triplet and quadruplet therapy to be useful for KCa, as studies to date have largely demonstrated a substantial increase in toxicity with only a very small PFS benefit. For the ongoing CARE1 trial, treatment-naïve patients with metastatic ccRCC are being stratified by PD-L1 expression and then randomized to the combination of 2 ICIs or an ICI plus a tyrosine kinase inhibitor [37].

New therapeutic concepts also hold promise, such as neoantigen vaccination, in which a vaccine is produced using material from the tumours of individual patients. An early study of this approach demonstrated long-term remission rates up to and beyond 48 months in some patients [38]. The INTerpath-004 randomized trial (NCT06307431) is examining the effect of pembrolizumab with or without the addition of the experimental vaccine V940 (mRNA-4157). It includes patients with intermediate- or high-risk papillary or ccRCC.

During a Q&A session, Dr. Tanguay asked what the limiting factor is to move forward in the use of KIM-1 as a biomarker in the clinical setting. Dr. Bex replied that there is no universally agreed-upon threshold for KIM-1 as yet for use in clinical practice, but it continues to be used in research. It could be useful in the adjuvant setting because it may be predictive of disease volume. KIM-1 may also be an on-treatment marker, as has been demonstrated in the JAVELIN trial with avelumab [39]. Before it is used clinically, it is necessary to determine whether KIM-1 can help identify patients who are most likely to benefit from adjuvant therapy, similar to what was done in the TOMBOLA trial for BCa [40].

Next, Dr. Black asked about SBRT becoming standard of care for small renal masses. Dr. Bex said there is a good possibility it will, but a limiting factor is the ability to design randomized controlled trials (RCTs) comparing SBRT with other treatments, as patients are usually ineligible for one of the treatment options being compared. In a few years, if the update of the TROG 15.03 FASTER II trial as well as the results of other trials confirm the safety of this modality, it will probably be increasingly used clinically even without RCTs, as occurred with cryotherapy. Uptake will be facilitated by an RCT in a setting different from that of TROG 15.03 FASTER II, where patients were offered SBRT only if they did not want or were ineligible for surgery. Conducting an RCT with an SBRT arm in younger, potentially curable patients may have ethical limitations, but once safety has been established, there will be no reason to consider SBRT any differently from any other form of ablation. Dr. Tanguay added that a small trial was conducted in Ontario, looking at the feasibility of randomizing patients between radiofrequency ablation and SBRT, and feasibility was established [41]. The limiting factor was a mandatory biopsy at 1 year, as almost all the biopsies of the irradiated patients were positive. This dampened the enthusiasm to move on to a larger trial until long-term outcomes with these patients are known.

The final presentation was given by Dr. Robert Jones (United Kingdom) on advances in prostate cancer (PCa). Dr. Jones recalled that the current standard of care for advanced PCa is androgen deprivation therapy (ADT) and, when metastases occur in hormone-sensitive disease, androgen receptor pathway inhibitors (ARPI). Some patients may also receive chemotherapy. When patients become castration resistant, there are a few chemotherapy options as well as poly ADP-ribose polymerase (PARP) inhibitors and radionuclides, with little evidence to guide optimal treatment sequencing.

Research over the past 15–20 years has had a major impact in 2 areas. First is the recognition that castration-resistant disease is not “hormone resistant” as, in most cases, the cancer continues to be driven by an activated androgen receptor (AR) pathway. Second is the recognition that systemic therapies, such as ARPIs, have the greatest impact when given in the hormone-sensitive state, as efficacy generally wanes considerably once patients become castration resistant.

Currently, PARP inhibitors are used in patients with castration-resistant disease. The TALAPRO-3 trial is investigating whether greater benefits can be obtained by moving them up to the hormone-sensitive population. In this trial, patients with hormone-sensitive PCa are treated with ADT and then randomized to the ARPI enzalutamide with or without the addition of the PARP inhibitor talazoparib [42]. In this trial, patients are selected for the presence of a homologous recombination deficiency (HRD) mutation. In clinical practice, about 20–25% of patients are HRD-positive, with the *BRCA2* mutation being the most common.

TALAPRO-3 comes on the heels of TALAPRO-2, which revealed an improved overall survival in the HRD-positive, castration-resistant population with the addition of talazoparib to enzalutamide (HR 0.622, 95% CI 0.475–0.814). This translated to a 14-month median OS advantage compared with the control arm [43]. TALAPRO-3 will help

determine whether similar, or perhaps greater, benefits can be obtained with the same treatment when given in the hormone-sensitive population.

Currently, some patients with metastatic PCa are undoubtedly being overtreated, with some remaining many years on doublet endocrine therapy. This can have a significant negative impact on general health, with adverse effects in the bones, muscles, and cardiovascular system over the long term. A de-escalation trial in Europe is randomizing patients with hormone-sensitive metastatic PCa (mHSPC) who have a prostate-specific antigen (PSA) ≤ 0.2 ng/mL after 6–12 months of ARPI doublet, to continuous treatment (arm 1) or to an intermittent doublet therapy regimen (arm 2), in which therapy is paused with PSA ≤ 0.2 ng/mL and reinstated at investigator discretion [44]. Since there is strong evidence that disease outcome is good with PSA ≤ 0.2 ng/mL, Dr. Jones is hopeful this trial will identify a group of patients who can de-escalate therapy and avoid or minimize the morbidities that occur with continuous therapy.

Resistance to ARPIs in patients with advanced PCa is a common problem in the clinical setting. This usually indicates that the patient has missed an opportunity to be treated with an ARPI in the past, said Dr. Jones. He explained that ARPI resistance is often driven by the activated androgen receptor pathway, through mutations that occur in the ligand binding domain of the AR. This results in the AR no longer binding exclusively to androgens but also to other steroid hormones. As a result, current AR-targeted therapies are no longer effective, since the cancer can now be driven by other steroid hormones.

Thus, a number of drugs that target the AR in novel ways are under investigation. The one that Dr. Jones highlighted as being most advanced in development is opevesostat, a CYP11A1 inhibitor. This enzyme is essential for metabolizing cholesterol into multiple steroid hormones, including adrenocortical steroid hormones. Thus, patients taking a CYP11A1 inhibitor will require essential steroid hormone replacement therapy, as is done in patients with bilateral adrenalectomies or Addison's disease.

For the OMAHA-004 trial, patients with castration-resistant, ARPI-resistant metastatic PCa are being randomized to opevesostat combined with dexamethasone and fludrocortisone or abiraterone/enzalutamide plus prednisone. Patients who received abiraterone in the past are given enzalutamide and vice versa. This trial includes patients with and without AR ligand binding domain (AR-LBD) mutations [45].

Dr. Jones said he is excited to see the results of the OMAHA-004 trial in light of the findings from the CYPIDES study, in which patients with AR-LBD mutations had high rates of PSA response with opevesostat. While responses were lower in patients without detectable AR-LBD mutations, they were still significant [46]. Thus, opevesostat may be the first hormonally active treatment that is effective in patients who are ARPI resistant.

Next, Dr. Jones discussed immunotherapy, noting that ICIs have been disappointing in the context of PCa, despite their success in other solid tumours. He does not expect any to come into routine use in this setting, except perhaps for a small group of patients with mismatch repair deficiency. A promising alternative is bispecific T-cell engagers (BiTE), which represent a novel way of engaging the immune system and may be effective in immune complex environments, such as PCa.

BiTEs are composed of the tips of 2 different monoclonal antibodies. One of them binds to a surface antigen on the T cell, usually CD3. The other binds to something specific to the cancer cell. Thus, the molecule creates an artificial immune synapse between the T cell and the tumour, activating the immune system to target the cancer [47]. Drugs of this class are already in use in haematological cancers and melanoma.

Several phase 3 RCTs are examining the role of BiTEs in advanced PCa. In the XALute trial, patients with metastatic castration-resistant PCa (mCRPC) who have already received ARPIs and chemotherapy are being randomized to either the BiTE xaluritamig, which binds to the STEAP1 tumour cell antigen, or investigator's choice of cabazitaxel or

a second ARPI [48]. Phase 1/2 data have already indicated that this drug does appear to be quite active, with even low doses producing a significant PSA response in most patients [49].

Importantly, BiTEs have a novel and not trivial toxicity profile. It will be important for clinicians to learn how to manage this appropriately.

Finally, said Dr. Jones, the most important advance will be in preventing men from developing metastatic disease in the first place. He believes this can best be achieved with early detection and rapid treatment. He highlighted the forthcoming TRANSFORM trial as a potential route to more effective screening in the future [50].

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Abbreviations

ADC	antibody–drug conjugate
AR	androgen receptor
AR-LBD	androgen receptor ligand binding domain
ARPI	androgen receptor pathway inhibitor
BCa	bladder cancer
BCG	bacillus Calmette-Guérin
BiTE	bispecific T-cell engager
ccRCC	clear-cell renal cell carcinoma
CI	confidence interval
CT	computed tomography
ctDNA	circulating tumour DNA
EV	enfortumab vedotin
HR	hazard ratio

HRD	homologous recombination deficiency
ICI	immune checkpoint inhibitor
KCa	kidney cancer
MIBC	muscle-invasive bladder cancer
MRD	minimal residual disease
MRI	magnetic resonance imaging
mUC	metastatic urothelial carcinoma
NMIBC	non-muscle-invasive bladder cancer
PARP	poly ADP-ribose polymerase
PCa	prostate cancer
pCR	pathological complete response
PET	positron emission tomography
PFS	progression-free survival
PSA	prostate-specific antigen
RC	radical cystectomy
RCC	renal cell carcinoma
RCT	randomized controlled trial
SBRT	stereotactic body radiotherapy
TURBT	transurethral resection of bladder tumour
utDNA	urinary tumour DNA

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