Podium Session 3: Oncology – Prostate
Sunday, June 28, 2026 • 14:15–15:25

Cite as: *Can Urol Assoc J* 2026;20(6Suppl1):S11-4. http://dx.doi.org/10.5489/cuaj.9810

POD 3.1

Real-world experience with ^{177}Lu-PSMA-617 in metastatic castration-resistant prostate cancer (mCRPC): Initial Canadian experience from Princess Margaret Cancer Centre

Di Maria Jiang[1], Patrick Veit-Haibach[2], Ur Metser[2], Seyed Ali Mirshahvalad[2], Eshetu Atenafu[3], Judy Gabrys[4], Mihaela Ginj[5], Esmail Al-ezzi[1], Nazanin Fallah-Rad[1], Vikaash Kumar[1], Sheeraz Ali[1], Khaled Abdulalem[1], Rebecca Wong[6], Neil E. Fleshner[7], Srikala S. Sridhar[1]

[1]Medical Oncology, Princess Margaret Cancer Center, University of Toronto, Toronto, Canada; [2]Joint Division of Medical Imaging (JDMI), University Health Network, University of Toronto, Toronto, Canada; [3]Biostatistics, University of Toronto, Toronto, Canada; Nuclear Medicine, University Health Network, Toronto, Canada; [5]Radiopharmacy, Universtity Health Network, Toronto, Canada; [6]Radiation Medicine Program, Princess Margaret Hospital, University Health Network, Toronto, Canada; [7]Urologic Oncology, Princess Margaret Cancer Centre, University Health Network, Toronto, Canada

Introduction: ^{177}Lu-PSMA-617 is a PSMA-targeted radioligand therapy (RLT) that improves survival in mCRPC post-ARPI and taxane chemotherapy. Public reimbursement became available in December 2024 at select Ontario centers following Health Canada approval based on the VISION trial. We report the initial Canadian real-world experience of delivering ^{177}Lu-PSMA-617 at Princess Margaret Cancer Centre (PMCC).

Methods: We retrospectively reviewed patients with mCRPC treated with ^{177}Lu-PSMA-617 (7.4 GBq q6w, up to six cycles) at PMCC between December 2024 and September 2025. Eligibility was assessed using PSMA PET/CT per VISION criteria; FDG PET/CT was not required. Outcomes included PSA and radiographic response, progression-free survival (PFS), overall survival (OS), and adverse events (AEs).

Results: Forty-three patients received 128 doses. Median age was 72 years (range 54-87), median PSA was 152 (range 4–4677); 49% had de novo metastases, 33% visceral disease, and 23% had baseline grade 2 anemia. All patients received ARPI and docetaxel; other prior treatments included cabazitaxel (30%), radium-223 (16%), platinum (7%), PARP inhibitors (2%), and other PSMA RLTs (2%). Median followup was 4.1 months (IQR 2.3–5.2); 86%, 37%, and 5% completed ≥2, ≥4, and six cycles. Among 42 evaluable patients, 15 (36%) achieved PSA50 response, 30 (71%) had any PSA response, and 4/29 (4%) had radiographic response. The six-month PSA PFS, radiographic PFS, and OS rates were 59% (95% CI 37–76), 75% (95% CI 56–87), and 79% (95% CI 50–92), respectively. Common AEs were xerostomia (40%), anemia (35%), fatigue (28%), nausea (21%), and thrombocytopenia (14%). Grade 3/4 AEs included anemia (16%; 12% transfused) and thrombocytopenia (2%). No fractures occurred. Dose reduction occurred in 7%. No patient discontinued treatment due to AEs.

Conclusions: Despite a more heavily pre-treated population with more aggressive disease features, outcomes with ^{177}Lu-PSMA-617 in real-world Canadian practice were comparable to the VISION trial, supporting broader implementation of PSMA RLT.

Funding: Novartis for statistical support.

POD 3.2

Automating standardization of prostate cancer biopsy and histopathology reports with privacy-preserving local large language models

Rohit Malyala[1], Takeshi Namekawa[2], Anna J. Black[1], Martin E. Gleave[1,2], Miles P. Mannas[1,2]

[1]Department of Urologic Sciences, University of British Columbia, Vancouver, Canada; [2]Vancouver Prostate Centre, Mohseni Institute of Urologic Sciences, Vancouver, Canada

Introduction: Histology and radiology reports are often saved in unstructured or poorly structured free-text formats. Large language models (LLMs) demonstrate promise in automatically structuring this data, but most deployments are not privacy-preserving. We hypothesized that tailored, private deployments of LLMs can extract structured data from unstructured, plain-language reports.

Methods: We used Mistral 7B, an open-source, small-parameter LLM that can be run on local consumer-grade hardware to ensure privacy, to extract information from transrectal ultrasound-guided prostate biopsy procedures and pathology reports. We tested "single-stage" (a single LLM prompt only) approaches for short ultrasound procedure reports, and a vertically integrated "multi-stage" LLM and natural language processing (NLP) approach for the US-procedure and histopathology reports, with iterative error resolution via recursive LLM prompting. Fifty records were for development, and 100 used for validation. Errors were qualitatively assessed to determine failure modes.

Results: LLM-structured outputs demonstrated high concordance with human-extracted data. Single-stage analysis of procedure reports achieved 95.3% accuracy (991 correct of 1040 discrete data points) across extracted data fields. The multistage LLM–NLP pipeline reached 98.0% accuracy (1314/1341) for ultrasound procedure reports. Applied to histopathology reports, the vertically integrated approach achieved 99.5% accuracy (9110/9150) across diagnosis, grade, key histologic features, and per-core location mapping. Errors clustered in ambiguous cases involving vague descriptors or uncommon reporting structures differing from institutional documentation culture.

Conclusions: A carefully deployed LLM, tailored to local practices in prostate biopsy procedure and pathology reporting, can be employed to automatically and privately structure data from unstructured radiology and pathology reports.

Acknowledgements: A modified version of this abstract was presented at the AUA 2026 Annual Meeting.

POD 3.3

Major cardiovascular event risk with androgen receptor pathway inhibitors and a luteinizing hormone-releasing hormone agonist or gonadotropin-releasing hormone antagonist: Real-world evidence

Marc B. Garnick[1], E. David Crawford[2], Zuzanna Szmukier[3], Christopher J.D. Wallis[4]

[1]Beth Israel Deaconess Medical Center, Havard Medical School, Boston, United States; [2]Koman Family Outpatient Pavilion, University of California, San Diego, San Diego, United States; [3]Medical Affairs, Tolmar Pharmaceuticals Canada, Inc., Toronto, Canada; [4]Department of Surgery, Division of Urology, University of Toronto, Toronto, Canada

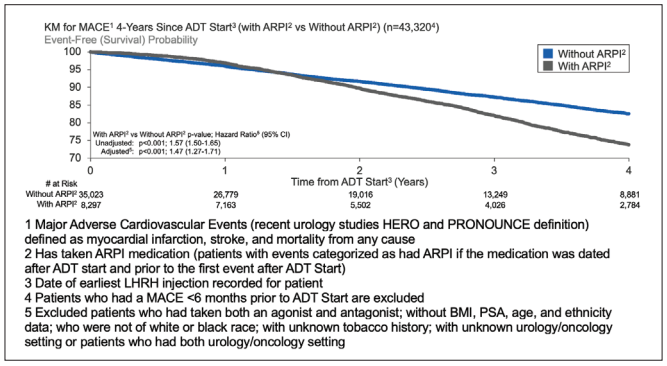
Introduction: The current standard of care for advanced prostate cancer (PCa) includes an androgen deprivation therapy (ADT) backbone with the addition of androgen receptor pathway inhibitors (ARPIs). While ARPIs prolong cancer survival, they have a known cardiovascular (CV) risk that may be additive to the risk associated with ADT. This study evaluated major adverse cardiovascular event (MACE) risk with ADT+ARPI doublet therapy, using real-world clinical data, comparing luteinizing hormone-releasing hormone (LHRH) agonists+ARPI vs. gonadotropin-releasing hormone (GnRH) antagonist+ARPI.

Methods: Data were collected from the Decision Resources Group (now Clarivate) Real World Evidence repository (>300 million U.S patients). Eligible patients received ≥1 ADT (99% started ADT within 2010–2020). Exclusions included missing ADT start date or MACE within the prior six months. MACE includes stroke, myocardial infarction, and all-cause mortality. Kaplan-Meier and Cox regression analyses assessed MACE-free survival and hazard ratios for ADT+ARPI vs. ADT alone, and agonists+ARPI vs. antagonist+ARPI.

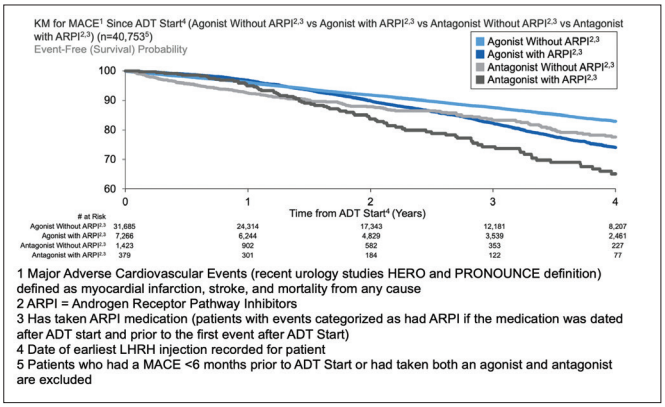
Results: A total of 43 320 patients were included. MACE risk for patients on ADT+ARPI vs. ADT without ARPI was 26.2% vs 17.5% at four years after ADT start (adjusted HR 1.47, 95% CI 1.27–1.71, p<0.001) (Figure 1, Table 1). At four years, unadjusted MACE risk among patients on doublet therapy (ADT+ARPI) was 34.9% with antagonist+ARPI vs. 25.9% with agonist+ARPI, and among patients on ADT alone was 22.4% with antagonist vs. 17.1% with agonists (Figure 2).

Conclusions: Based on observational data, ARPI-based doublet therapy, while guideline-recommended, notably increases MACE risk vs ADT alone, consistent with prior trial findings. Additionally, in this real-world cohort of approximately 45 000 patients, MACE risk was lower with agonists+ARPI vs. an antagonist+ARPI, although this may be attributable to residual confounding. Our findings underscore the need to adhere to CV risk mitigation to balance oncologic benefits of ARPI-based doublet therapy with its elevated CV risk.

Funding: Tolmar, Inc.



POD 3.3. Figure 1. KM for MACE1 4 years since ADT start[3] (with ARPI[2] vs. without ARPI[2]) (n=43 3204).



POD 3.3. Figure 2. KM for MACE[1] since ADT start[4] (agonist without ARPI[2,3] vs. agonist with ARPI[2,3] vs. antagonist without ARPI[2,3] vs. antagonist with ARPI[2,3]) (n=40 7535).

POD 3.3. Table 1. Univariable and multivariable Cox proportional hazard regression of MACE2

Univariable and Multivariable Cox Proportional Hazard Regression of MACE[2]

Factors	Univariable (n=43,320)[3,4]		Multivariable (n=6,701)[3,5]	
	HR (95% CI)	P-Value	HR (95% CI)	P-Value
With vs Without Metastasis (Baseline)	2.69 (2.40-3.03)	<0.001	2.42 (1.77-3.30)	<0.001
With vs Without Personal History of MACE[2]	2.76 (2.49-3.06)	<0.001	2.25 (1.70-2.97)	<0.001
Oncology vs Urology (Setting)	2.22 (2.03-2.43)	<0.001	2.04 (1.73-2.42)	<0.001
Without vs With Statin[1]	1.77 (1.68-1.87)	<0.001	1.49 (1.27-1.74)	<0.001
Antagonist vs Agonist	1.54 (1.38-1.72)	<0.001	1.49 (1.10-2.00)	0.009
With vs Without ARPI[1]	1.57 (1.50-1.65)	<0.001	1.47 (1.27-1.71)	<0.001
With vs Without Tobacco History	1.03 (0.95-1.12)	0.4	1.31 (1.09-1.57)	<0.005
White vs Black (Race)	1.68 (1.56-1.82)	<0.001	1.24 (1.02-1.50)	0.03
Increasing Age per Year (Older vs Younger)	1.07 (1.06-1.07)	<0.001	1.08 (1.07-1.09)	<0.001
Decreasing ADT Exposure per Year	1.00 (0.99-1.01)	0.7	1.04 (1.01-1.07)	0.01
Decreasing BMI per kg/m^2 (Baseline)	1.03 (1.03-1.04)	<0.001	1.02 (1.01-1.04)	<0.005
Increasing PSA per ng/mL (Baseline)	1.00 (1.00-1.00)	<0.001	1.00 (1.00-1.00)	<0.005
Non-Hispanic vs Hispanic	1.41 (1.24-1.62)	<0.001	1.46 (0.95-2.22)	0.08
With vs Without Family History of MACE[2]	1.14 (1.04-1.25)	0.006	1.22 (0.97-1.54)	0.09
With vs Without Hypertension (HTN)[6]	1.07 (1.01-1.14)	0.03	1.17 (0.99-1.38)	0.07
With vs Without Diabetes[6]	0.97 (0.92-1.03)	0.4	1.07 (0.92-1.25)	0.4

1 Has taken statins/ARPI medication (patients with events categorized as statins/ARPI if the medication was dated after ADT start and prior to the first event after ADT Start)
2 Major Adverse Cardiovascular Events (recent urology studies HERO and PRONOUNCE definition) defined as myocardial infarction, stroke, and mortality from any cause
3 Excluded patients who had a MACE <6 months prior to ADT Start
4 Largest N out of all factors is shown for univariable analysis; Ns for each factor vary
5 Excluded patients who had taken both an agonist and antagonist; without BMI, PSA, age, and ethnicity data; who were not of white or black race; with unknown tobacco history; with unknown urology/oncology setting or patients who had both urology/oncology setting
6 Has taken hypertension/diabetes medication or diagnosed with hypertension/diabetes disease (patients with events categorized as hypertension/diabetes if the medication/diagnosis was dated prior to the first event after ADT Start)

POD 3.4

Transperineal MRI-ultrasound fusion biopsy accurately detects clinically significant prostate cancer but underestimates adverse histologic features

Rocio Roldan-Testillano[1], Sepehr Niakani[1], Aïnhoa Olazabal[1], Hend Alshamsi[1], Alexis Rompré-Brodeur[1], Rafael Sanchez-Salas[1]

[1]Division of Urology, McGill University, Montreal, Canada

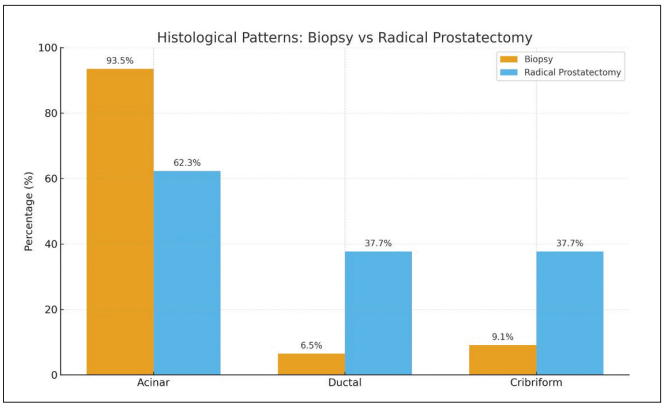
Introduction: MRI-ultrasound fusion biopsy has improved prostate cancer (PCa) detection and risk stratification; however, concordance with radical prostatectomy (RP) pathology remains imperfect. Accurate identification of adverse histologic patterns is critical for treatment selection, particularly for focal therapy and active surveillance. We evaluated agreement in Gleason grade group (GGG) and histologic subtypes between MRI-targeted transperineal (TP) fusion biopsy and RP.

Methods: Patients undergoing TP MRI-ultrasound fusion-guided biopsy followed by RP between January 2023 and April 2025 were analyzed. All biopsies were performed using the Koelis Trinity™ platform. Agreement in GGG was assessed using Spearman's rank correlation and concordance rates. Detection of acinar, ductal, and cribriform patterns was compared using McNemar's test, with inter-method agreement evaluated by Cohen's kappa.

Results: Among 716 TP biopsies, 76 patients proceeded to RP with available pathologic correlation. At biopsy, 5.3% were GGG1, 61.3% GGG2, and 33.3% GGG ≥3, compared with 1.3%, 72.7%, and 26.0% at RP, respectively. Overall GGG concordance was 63.6%, with upgrading in 13.0% and downgrading in 20.8%. A moderate correlation was observed (ρ=0.384, p<0.001). Acinar morphology was detected in 93.5% of biopsies vs. 62.3% of RP specimens, ductal features in 6.5% vs.37.7%, and cribriform architecture in 9.1% vs. 37.7% (p<0.001 for cribriform) (Figure 1). Agreement was moderate for acinar (κ≈0.55), weak-to-moderate for ductal (κ≈0.42), and weak for cribriform patterns (κ≈0.35).

Conclusions: MRI-targeted TP fusion biopsy provides good detection of clinically significant PCa and moderate agreement in GGG with final RP pathology; however, aggressive histologic features, particularly ductal and cribriform architecture, are frequently underdetected. This limitation may impact risk stratification and patient selection for focal therapy and active surveillance, supporting the need for complementary diagnostic strategies to improve identification of biologically aggressive disease.

Acknowledgements: This study was presented at the AUA 2026 Annual Meeting.



POD 3.4. Figure 1. Histologic pattern detection at transperineal biopsy vs. radical prostatectomy. Comparison of acinar, ductal, and cribriform histologic patterns etected at MRI-ultrasound fusion biopsy and final radical prostatectomy pathology.

POD 3.5

Effect of prostate cancer screening with prostate-specific antigen testing on long-term prostate cancer-specific mortality: A systematic review and meta-analysis

David-Dan Nguyen[1,2], Borna Tadayon[1,3], Lauren Lin[1,4], Jethro C.C. Kwong[1], Earl Miller[5], Alexandre R. Zlotta[1,5,6], Neil E. Fleshner,[1,5,7], Amanda E. Hird[1,7], Aisha K. Lofters[8], Christopher J.D. Wallis[1,5,6], Laura C. Rosella[9], Philipp Dahm[10], Gordon Guyatt[3,11], Girish S. Kulkarni[1,2,5]

[1]Division of Urology, Department of Surgery,, University of Toronto, Toronto, Canada; [2]Institute of Health Policy, Management and Evaluation, Dalla Lana School of Public Health, University of Toronto, Toronto, Canada; [3]Department of Health Research Methods, Evidence, and Impact, Faculty of Health Sciences, McMaster University, Hamilton, Canada; [4]Institute of Medical Sciences, Temerty Faculty of Medicine, University of Toronto, Toronto, Canada; [5]Divisions of Urology and Surgical Oncology, Department of Surgery, Toronto General Hospital & Princess Margaret Cancer Center, University Health Network, University of Toronto, Toronto, Canada; [6]Division of Urology, Department of Surgery, Mount Sinai Hospital, University of Toronto, Toronto, Canada; [7]Division of Urology, Department of Surgery, Sunnybrook Health Sciences, University of Toronto, Toronto, Canada; [8]Department of Family and Community Medicine, Women's College Hospital, University of Toronto, Toronto, Canada; [9]Department of Epidemiology, Dalla Lana School of Public Health, University of Toronto, Toronto, Canada; [10]Department of Urology, University of Minnesota, Minneapolis, United States; [11]Department of Medicine, Faculty of Health Sciences, McMaster University, Hamilton, Canada

Introduction: Interpretations of the body of evidence on the long-term mortality effect of prostate-specific-antigen (PSA)-based screening have varied. Extended and mature followup across all major trials of conventional PSA-based screening now allows for a more definitive synthesis. As such, we conducted a systematic review and meta-analysis to estimate the effect of PSA-based screening compared with no screening on prostate cancer-specific mortality.

Methods: We searched PubMed, MEDLINE, EMBASE, and CENTRAL through October 2025. We included randomized controlled trials (RCT) if they compared PSA-based screening with no screening or usual care among adults with prostates. Two reviewers independently screened citations and extracted data. Risk of bias was assessed with ROBUST-RCT. The primary outcome was prostate cancer-specific mortality at the longest followup. Secondary outcomes included mortality beyond 12 years and mortality closest to 10 years of followup. Incidence rate ratios (IRRs) were pooled using random-effects models. Certainty of evidence was assessed with the GRADE approach.

Results: We included five RCTs analyzing 721 607 participants with 11–23 years of followup. RCTs differed in screening protocols and adherence (control arm PSA screening range: 7–90%). PSA-based screening, with a high certainty in the evidence based on the GRADE approach, demonstrated a reduced risk of prostate cancer-specific mortality at the longest followup (IRR 0.92, 95% CI 0.87–0.97; ARR nine fewer per 10 000, 95% CI 3–14 fewer; I^2= 6.5%; τ^2=0). Secondary analyses by followup duration suggested greater relative prostate cancer-specific mortality reduction with longer followup.

Conclusions: With 11–23 years of followup, high certainty evidence demonstrates that prostate cancer screening with PSA testing reduces prostate cancer-specific mortality. This represents a departure from earlier meta-analyses, which pooled less mature trial data, did not demonstrate a prostate cancer-specific mortality benefit, and rated the certainty of evidence as low or moderate. Our finding does not in itself justify population-based screening or imply that every well-informed individual would elect to undergo screening. This prostate cancer-specific mortality benefit should be weighed against known harms of conventional PSA-based screening and consider emerging approaches under study that may mitigate these harms.

POD 3.6

Cost-benefit analysis of systematic biopsies in prostate cancer histopathology services in British Columbia

Rohit Malyala[1], Takeshi Namekawa[2], Anna J. Black[1], Martin E. Gleave[1,2], Miles P. Mannas[1,2]

[1]Department of Urologic Sciences, University of British Columbia, Vancouver, Canada; [2]Vancouver Prostate Centre, Mohseni Institute of Urologic Sciences, Vancouver, Canada

Introduction: Prostate cancer (PCa) diagnosis requires tissue biopsy. Although prostate MRI has improved lesion targeting, combined systematic and targeted biopsies remain standard practice, increasing pathology workloads. This study quantifies histopathology costs in British Columbia and evaluates the time, cost, and diagnostic tradeoffs of omitting systematic biopsies.

Methods: We conducted a retrospective, cross-sectional study of patients undergoing MRI-fusion prostate biopsy (transrectal or transperineal) at a high-volume tertiary center (Vancouver General Hospital) from 2020–2023. Technologist and pathologist workload, costs, and missed clinically significant PCa (csPCa; grade group [GG] ≥2) rates were modeled for targeted-only vs. combined biopsy strategies using institutionally-derived time and cost estimates.

Results: The cohort included 352 patients with 465 MRI targets. Combined systematic and targeted biopsy required a mean of 13.5 blocks per prostate versus 4.72 blocks per prostate (3.72 per target) for a targeted-only approach. Estimated per-block processing and interpretation times were 3.0 minutes for technologists and 3.75 minutes for pathologists. Omitting systematic biopsy reduced projected costs by 64.9%, saving $346 050 CAD and 346 hours of combined labor. The csPCa miss rate was 3.4% for ipsilateral and 1.7% for contralateral systematic cores. Contralateral extraprostatic extension was rare (0.4%). In PI-RADS 3–4 patients with PSA 0–20, contralateral biopsy omission carried <1.9% risk of upgrading to GG≥2 and 0% risk of extraprostatic extension, corresponding to approximately $135 000 CAD in savings.

Conclusions: Omitting systematic biopsy could reduce histopathology costs and labor by nearly two-thirds, saving approximately $983 CAD and approximately one hour of combined technologist and pathologist time per prostate, at the cost of missing csPCa in about 3.4% or fewer cases. Selective omission may offer a pragmatic balance between diagnostic yield and fiscal sustainability.

POD 3.7

Perioperative (neoadjuvant and adjuvant) apalutamide (APA) + androgen deprivation therapy (ADT) vs. placebo (PBO) + ADT with radical prostatectomy (RP) in high-risk localized or locally advanced prostate cancer: Final analysis of the PROTEUS phase 3 study

Martin Gleave[1], Mary-Ellen Taplin[2], Neal D. Shore[3], Angela D. Lopez-Gitlitz[4], Alexander Kretschmer[17], Eleni Efstathiou[5], Ronaldo Damião[6], Toshiyuki Kamoto[7], Ashley Ross[8], Alberto Briganti[9], Boris A. Hadaschik[10], Axel Heidenreich[11], Alvaro Juárez Soto[12], Brendan Rooney[13], Shaozhou Ken Tian[17], Lisa Wetherhold[17], Branko Miladinovic[14], Sharon McCarthy[17], Christopher P. Evans[15], Adam S. Kibel[16]

[1]University of British Columbia, Vancouver, Canada; [2]Lank Center for Genitourinary Oncology, Dana-Farber Cancer Institute, Boston, United States; [3]START Carolinas/Carolina Urologic Research Center, Myrtle Beach, United States; [4]Johnson & Johnson, Los Angeles, United States; [5]Knight Cancer Institute, Oregon Health and Science University, Portland, United States; [6]Universidade do Estado do Rio de Janeiro, Rio de Janeiro, Brazil; [7]University of Miyazaki, Miyazaki, Japan; [8]Northwestern University, Chicaco, United States; [9]Unit of Urology, Urological Research Institute (URI), IRCCS Ospedale San Raffaele, Vita-Salute San Raffaele University, Milan, Italy; [10]University of Duisburg-Essen and German Cancer

Consortium (DKTK), University Hospital Essen, Essen, Germany; [11]University Hospital of Cologne, Cologne, Germany; [12]Hospital Universitario de Jerez de la Frontera, Cádiz, Spain; [13] Johnson & Johnson, High Wycombe, United Kingdom; [14] Johnson & Johnson, San Diego, United States; [15]University of Nevada, Reno, United States; [16] Mass General Brigham, Boston, United States; [17]Johnson & Johnson, Spring House, United States

Introduction: RP is potentially curative for patients (pts) with high-risk localized or locally advanced prostate cancer (HR LPC/LAPC), yet approximately 50% of pts relapse. PROTEUS evaluated whether APA + ADT vs. PBO + ADT before and after RP with pelvic lymph node dissection (henceforth, RP) improves pathologic complete response/minimal residual disease (pCR/MRD) and metastasis-free survival (MFS) in HR LPC/LAPC.

Methods: Pts with newly diagnosed HR LPC/LAPC (histology, prostate-specific antigen [PSA], and cN0/cN1 on conventional imaging) were randomized 1:1 to blinded APA (240 mg/d) or PBO as neoadjuvant treatment (tx) for six months + ADT, with a two-week break prior to and a four-week break post-RP, followed by six months of assigned tx. Dual primary endpoints, pCR/MRD (≤ypT2, ≤5 mm tumor diameter) and MFS based on conventional or prostate-specific membrane antigen positron emission tomography (PSMA PET) imaging, were assessed by blinded independent central review (BICR). Secondary endpoints included event-free survival (EFS), time to first subsequent tx (TTST1), time to distant metastasis (TTDM), and safety. Exploratory endpoints included residual cancer burden (RCB/MRD; ≤ypT2, ≤0.25 cm3) and investigator-assessed MFS.

Results: Of 2109 pts randomized (APA + ADT [1057] or PBO + ADT [1052]), median (range) age was 66.0 (41–89) years; PSA, 14.8 (0.0–2798.0) ng/mL; GS ≥8, 95.8%. Median followup was 61.7 mo. Both primary endpoints were met with APA + ADT vs. PBO + ADT: pCR/MRD rate was significantly higher, 8.9% vs. 1.0% (odds ratio [OR] 10.17, 95% CI 5.27–19.64, p<0.0001); MFS by BICR was significantly improved, with HR 0.80, 95% CI 0.67–0.96, p=0.0169 and five-year MFS rate of 78.2% vs. 73.5%; median not reached [NR]. Investigator-assessed MFS favored APA + ADT, with HR 0.74, 95% CI 0.62–0.87; nominal p=0.0004. EFS, TTST1, and TTDM were all significantly improved with APA + ADT (Table 1), as was RCB/MRD: MRD 30.6% vs. 11.7%, OR 3.36, 95% CI 2.67–4.23, nominal p<0.0001. Grade 3/4 tx-emergent adverse events (TEAEs) for APA + ADT vs. PBO + ADT were 39.6% vs. 31.0%, with discontinuation due to TEAEs 7.4% vs. 2.7%, respectively.

Conclusions: APA + ADT significantly increased the curative success of RP in pts with HR LPC/LAPC, with a 10-fold higher odds of pCR/MRD and a clinically meaningful 20% reduction in risk of distant metastasis or death. Secondary endpoints all favored APA + ADT. These results support combined APA + ADT and RP as a new standard of care for pts with HR LPC/LAPC.

Funding: Supported by Johnson & Johnson.

Acknowledgements: This abstract is an encore presentation of the same abstract presented at ASCO 2026.

POD 3.7. Table 1

	HR (95% CI)	p[a]	APA + ADT n=1057 Median (mo)	PBO + ADT n=1052 Median (mo)
EFS	0.71 (0.630.80)	<0.0001	57.1	38.4
TTST1 (local, regional, or systemic, including ADT reinitiation)	0.65 (0.570.73)	<0.0001	74.2	41.5
TTDM (conventional or PSMA PET imaging)	0.68 (0.550.83)	0.0002	NR	NR

[a]Stratified by GS (7, ≥8), nodal status, and geographic region (North America, European Union, rest of world).