What Clinicians Want to Know: Addressing Community Oncologists' Questions About the Care of Patients with Prostate Cancer

Saturday, May 30, 2026

7:00 PM – 9:00 PM CT (8:00 PM – 10:00 PM ET)

Faculty

Wassim Abida, MD, PhD

Rahul Aggarwal, MD

Emmanuel S Antonarakis, MD

Karim Fizazi, MD, PhD

Moderator

Rana R McKay, MD, FASCO

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Faculty



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Karim Fizazi, MD, PhD
GETUG President
ESMO Public Policy Director
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Director, Centre Oscar Lambret
Lille, France



Moderator
Rana R McKay, MD, FASCO
Professor of Medicine, Urology, and Radiation Medicine and Applied Sciences
Associate Director, Clinical Research
Co-Lead, Genitourinary Program
Moores Cancer Center
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San Diego, California

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Faculty

Advisory Committees	AstraZeneca Pharmaceuticals LP, K36 Therapeutics, Nuvation Bio Inc, ORIC Pharmaceuticals
Consulting Agreements	AstraZeneca Pharmaceuticals LP, Boundless Bio, Duality Biologics, Endeavor BioMedicines, Tolmar
Contracted Research	AstraZeneca Pharmaceuticals LP, Ipsen Biopharmaceuticals Inc, K36 Therapeutics, Merus, MOMA Therapeutics, Nuvation Bio Inc, ORIC Pharmaceuticals, TransThera

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Dr Aggarwal — Disclosures
Faculty

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Nonrelevant Financial Relationships	Prostate Cancer Clinical Trials Consortium

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Dr Antonarakis — Disclosures
Faculty

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Contracted Research	Actinium Pharmaceuticals Inc, MacroGenics Inc, Merck, MorphoSys, Novartis, Orion Corporation, pharmaand GmbH, Seagen
Patents	Qiagen
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Prof Fizazi — Disclosures
Faculty

Honoraria, Former Institution	Advanced Accelerator Applications, Amgen Inc, Astellas, AstraZeneca Pharmaceuticals LP, Bayer HealthCare Pharmaceuticals, Daiichi Sankyo Inc, Janssen Biotech Inc, Merck, MSD, Novartis, Pfizer Inc

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Dr McKay — Disclosures
Moderator

Advisory Committees and Consulting Agreements	Ambrx, Arcus Biosciences, AstraZeneca Pharmaceuticals LP, Aveo Pharmaceuticals, Bayer HealthCare Pharmaceuticals, Blue Earth Diagnostics, Boundless Bio, Bristol Myers Squibb, Calithera Biosciences, Caris Life Sciences, Daiichi Sankyo Inc, Dendreon Pharmaceuticals Inc, Eisai Inc, Exelixis Inc, Janssen Biotech Inc, Lilly, Merck, Myovant Sciences, Neomorph, Nimbus Therapeutics, Novartis, Pfizer Inc, Sanofi, Seagen Inc, Sorrento Therapeutics, Telix Pharmaceuticals Limited, Tempus
Contracted Research	Artera, AstraZeneca Pharmaceuticals LP, Bristol Myers Squibb, Exelixis Inc, Incyte Corporation, Natera Inc, Oncternal Therapeutics

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Research To Practice President Neil Love, MD — Disclosures

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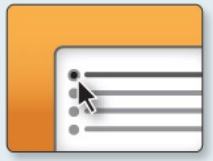
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Clinicians in the Meeting Room

Networked iPads are available.



Review Program Slides: Tap the Program Slides button to review speaker presentations and other program content.



Answer Survey Questions: Complete the pre- and postmeeting surveys. Survey questions will be discussed throughout the meeting.



Ask a Question: Tap Ask a Question to submit a challenging case or question for discussion. We will aim to address as many questions as possible during the program.

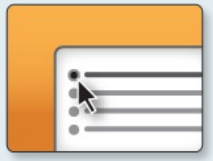
For assistance, please raise your hand. Devices will be collected at the conclusion of the activity.

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Clinicians Attending via Zoom



Review Program Slides: A link to the program slides will be posted in the chat room at the start of the program.



Answer Survey Questions: Complete the pre- and postmeeting surveys.



Ask a Question: Submit a challenging case or question for discussion using the Zoom chat room.



Get CME Credit: A credit link will be provided in the chat room at the conclusion of the program.

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About the Enduring Program



- The live meeting is being video and audio recorded.
- The proceedings from today will be edited and developed into an enduring web-based program. An email will be sent to all attendees when the activity is available.
- To learn more about our education programs, visit our website, **www.ResearchToPractice.com**

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Friday May 29	**Gastroesophageal Cancers** 11:30 AM – 1:00 PM CT (12:30 PM – 2:00 PM ET)
	Non-Small Cell Lung Cancer 6:30 PM – 8:30 PM CT (7:30 PM – 9:30 PM ET)
	Chronic Lymphocytic Leukemia 6:30 PM – 8:30 PM CT (7:30 PM – 9:30 PM ET)
	Colorectal Cancer 6:30 PM – 8:00 PM CT (7:30 PM – 9:00 PM ET)
Saturday May 30	**Ovarian Cancer** 7:00 PM – 9:00 PM CT (8:00 PM – 10:00 PM ET)
	Prostate Cancer 7:00 PM – 9:00 PM CT (8:00 PM – 10:00 PM ET)
	Small Cell Lung Cancer 7:00 PM – 9:00 PM CT (8:00 PM – 10:00 PM ET)
Sunday May 31	**Oral SERDs and Agents Targeting the PI3K/AKT/mTOR Pathway for Breast Cancer** 7:00 PM – 9:00 PM CT (8:00 PM – 10:00 PM ET)
	Endometrial Cancer 7:00 PM – 8:30 PM CT (8:00 PM – 9:30 PM ET)
	CAR T-Cell Therapy and Bispecific Antibodies for Non-Hodgkin Lymphoma 7:00 PM – 9:00 PM CT (8:00 PM – 10:00 PM ET)
Monday June 1	**ADCs for Breast Cancer** 7:00 PM – 9:00 PM CT (8:00 PM – 10:00 PM ET)
	Novel Therapies for Non-Hodgkin Lymphoma 7:00 PM – 9:00 PM CT (8:00 PM – 10:00 PM ET)
	Relapsed/Refractory Multiple Myeloma 7:00 PM – 9:00 PM CT (8:00 PM – 10:00 PM ET)
Tuesday June 2	**Myelofibrosis (Webinar)**

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Moderator

Rana R McKay, MD, FASCO

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What Clinicians Want to Know: Addressing Community Oncologists' Questions About the Care of Patients with Prostate Cancer

Survey of US-Based General Medical Oncologists
May 13-26, 2026

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Agenda

Module 1: Evolving Management of Nonmetastatic Hormone-Sensitive Prostate Cancer (HSPC) — Dr Antonarakis

Module 2: Current and Future Hormonal Treatment for Metastatic HSPC (mHSPC) — Dr Aggarwal

Module 3: Current and Future Role of Regimens Combining PARP Inhibitors and AR Pathway Inhibitors in Metastatic Prostate Cancer — Dr Abida

Module 4: Emerging Role of AKT Inhibition for Patients with mHSPC — Dr Fizazi

Module 5: Current and Future Use of Radiopharmaceuticals in Metastatic Prostate Cancer — Dr McKay

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Module 2: Current and Future Hormonal Treatment for Metastatic HSPC (mHSPC) — Dr Aggarwal

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Module 5: Current and Future Use of Radiopharmaceuticals in Metastatic Prostate Cancer — Dr McKay

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Research To Practice
May 30, 2026

Evolving Management of Nonmetastatic Hormone-Sensitive/*Androgen Pathway Modulation-Naïve* Prostate Cancer (nmHSPC/nmAPMN)

Emmanuel S. Antonarakis, M.D.
Clark Endowed Professor of Medicine
Division of Hematology/Oncology & Transplantation, University of Minnesota
Associate Director of Translation, Masonic Cancer Center

Itinerary

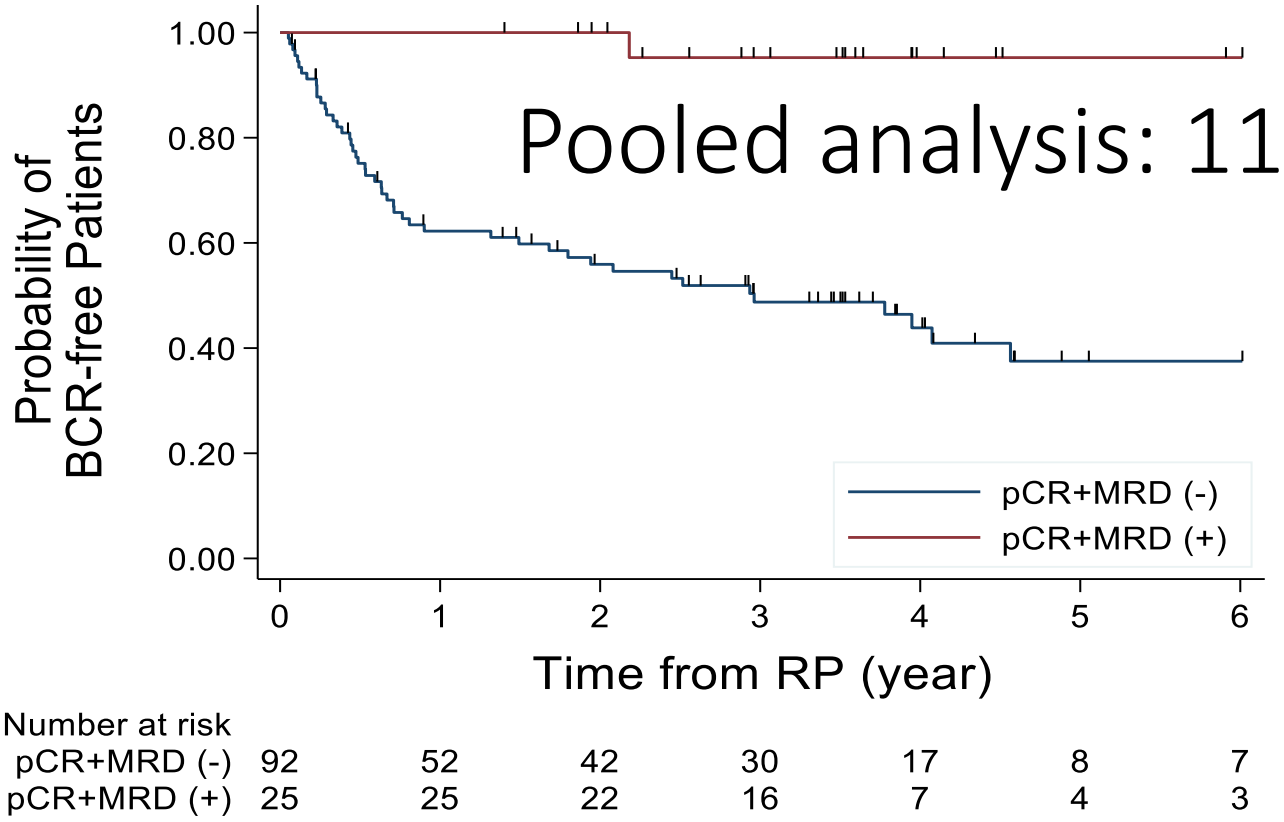
- Treatment intensification w/ ADT + ARPI in nmHSPC/nmAPMN
- The phase 3 PROTEUS study (**presented tomorrow at Plenary**)
- Updates on the phase 3 EMBARK study in high-risk BCR
- Updates on the phase 3 PRESTO study in high-risk BCR
- Ongoing phase 3 studies of ADT + ARPI in nmHSPC/nmAPMN

Rationale for PROTEUS: Peri-operative ADT +/- ARPI (plus RP) in high-risk localized PCa

- Series of phase 2 trials (DFCI x4, MDA x1, Eur x1) evaluated ADT + ARPI prior to radical prostatectomy (RP)
- pCR rate low: 5-10%
- Major pathologic response: 20-30%: either one dimension $\leq$ 5mm tumor or residual cancer burden (dimension, cellularity) < $0.25cm^3$
- Participants achieving a major pathologic response had high freedom-from-PSA relapse and improved MFS

McKay R, et al. *J Urol* 2021; 205: 1689.

Major pathologic response w/ peri-operative ADT +/- ARPI and long-term outcomes



3-year BCR-free rate:
Overall: 59.1%
(95% CI, 49.0 - 67.9)

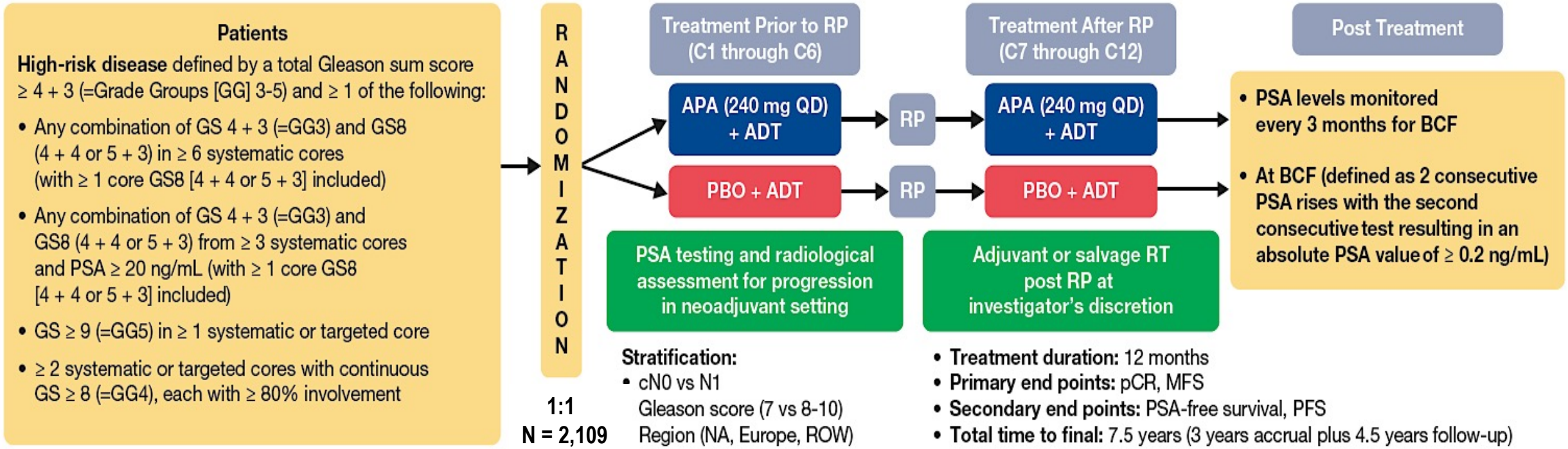
Exceptional Responder:
95.2% (95% CI, 70.7 - 99.3)

Non-Responder: 48.7%
(95% CI, 37.5 - 59.1)

McKay R, et al. *J Urol* 2021; 205: 1689.

PROTEUS: NCT03767244

Randomized, Double-blind, Placebo-Controlled, Phase 3 Study of ADT +/- Apalutamide in Subjects with High-risk Localized or Locally Advanced Prostate Cancer Who are Candidates for Radical Prostatectomy



Courtesy of Mary-Ellen Taplin (with permission)

PROTEUS: NCT03767244

- Completed accrual of 2,109 patients from 2019 to 6/2022
  - Accrual completed earlier than projected despite COVID pandemic
  - Last RP performed in 12/2022
- **Co-primary endpoints: pCR/MRD and MFS**
  - MFS endpoint incorporated Conventional or PSMA-PET imaging
- Completed accrual of Sub-study of RP vs Neoadj ADT/Apa in 9/2023
  - 3:1 randomization to RP vs Neoadj ADT/Apa [N = 400]
- **Secondary endpoints**:
  - Event-free survival (time to BCR, or local/metastatic recurrence, or death)
  - Time to next local or systemic treatment
  - NED at 4 years
  - Biochemical-free survival (with T >200 ng/dL)

Courtesy of Mary-Ellen Taplin

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 (HR LPC/LAPC): Final Analysis of the PROTEUS Phase 3 Study

Taplin M-E et al.

ASCO 2026;Abstract LBA1

Plenary Session

SUNDAY MAY 31, 2026

Hall B1

1:05 PM – 1:17 PM CDT

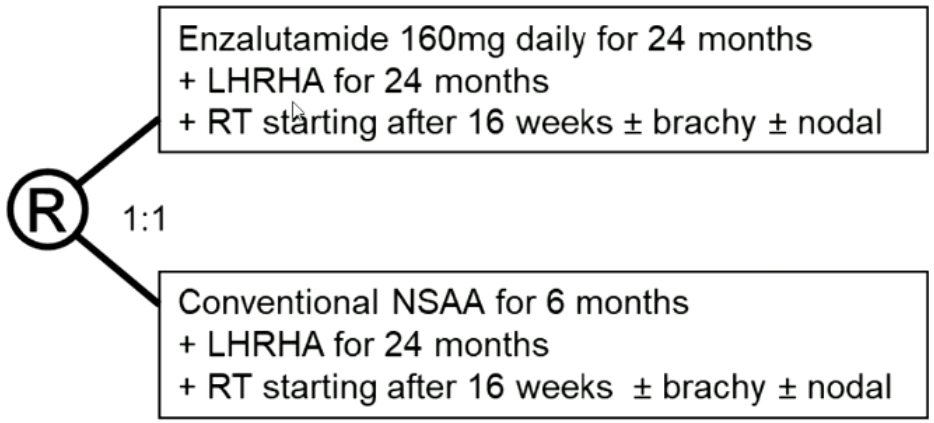
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Phase 3 ENZARAD Trial: Study Design

Median follow-up: 8 years

Eligibility
Localised prostate cancer
High risk of recurrence
Suitable for EBRT

Stratification
Gleason score 8-10
T3-4 disease
N1 disease
PSA ≥20 ng/mL
Brachytherapy boost
Pelvic nodal RT
Study Site



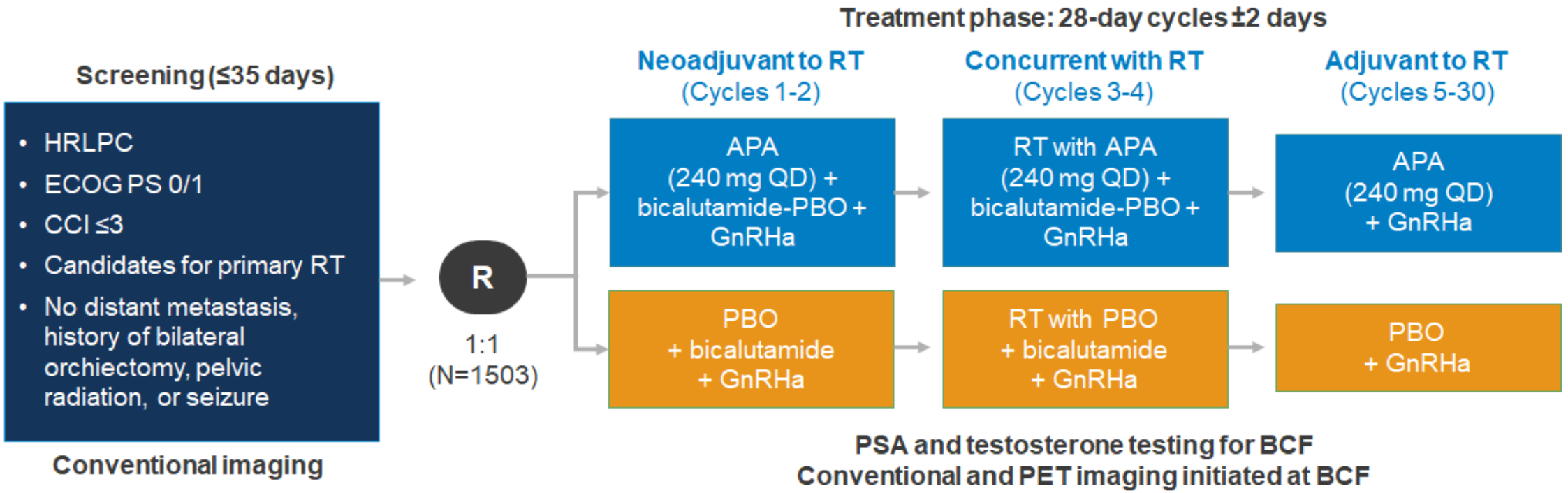
Endpoints
- Metastasis-free survival (primary)
- OS
- Cause-specific survival
- PSA PFS
- Clinical PFS
- Castration-resistance
- HRQoL
- AEs
- Incremental cost-effectiveness

N=800

Primary Endpoint:
MFS based on conventional imaging (CT or MRI or bone scan, per ICECAP)
Lesions on PSMA-PET alone insufficient
Event metastasis or death from any cause before metastasis

Nguyen PL, et al. *ESMO* 2025. Abstract LBA 86.

Phase 3 ATLAS Trial: Study Design



Long-term follow-up

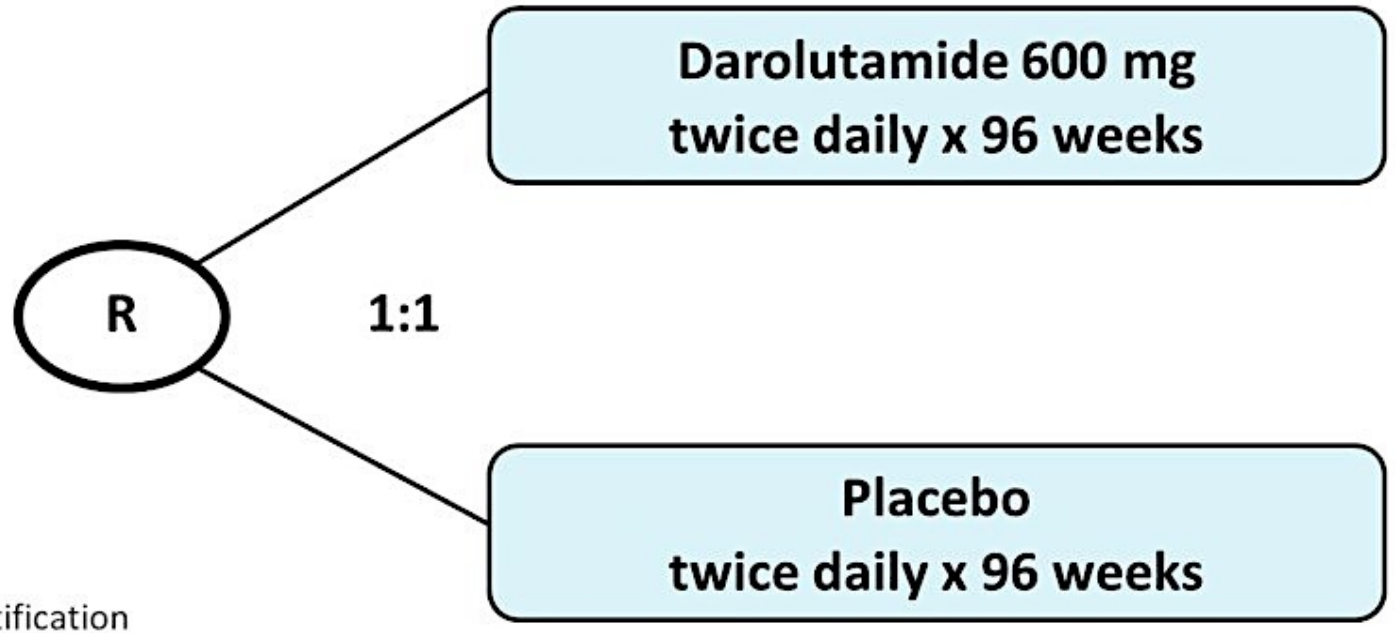
- PSA and testosterone levels monitored every 3 months until distant metastasis by BICR
- Conventional imaging every 6 months until distant metastasis by BICR or death
- PET imaging every 6 months until distant metastasis on PET or conventional imaging by BICR or death

ClinicalTrials.gov identifier: NCT02531516.

Phase 3 DASL-HiCaP trial: Study Design

Aim: To determine the effect on metastases free survival of adding darolutamide to ADT and radiation therapy, in either the primary definitive setting or very high-risk postoperative setting.

All participants are also treated concurrently with an LHRHA for 96 weeks post randomization, plus RT starting at week 8-24 post randomization.



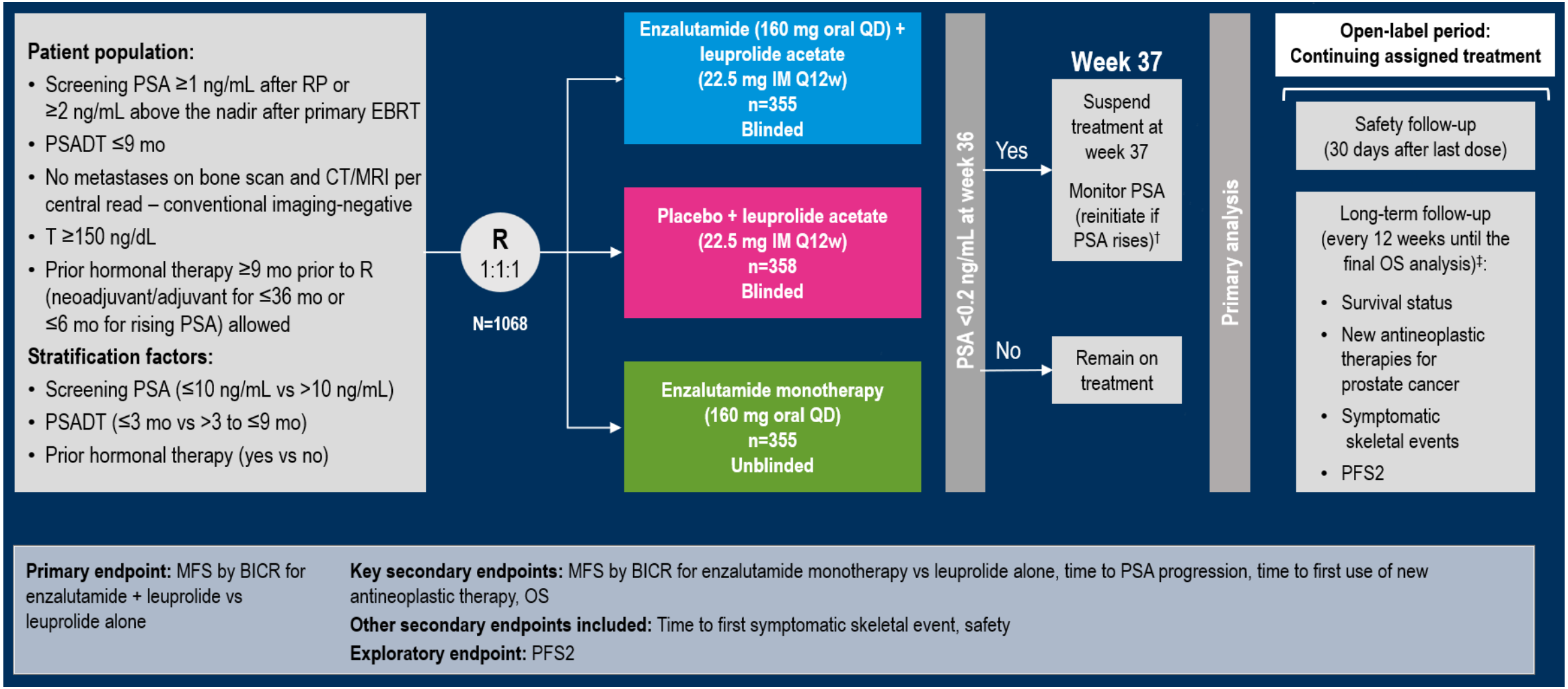
Patients planned for RT with very high-risk localized prostate cancer on conventional imaging or very high-risk features with PSA persistence or rise within 1 year following RP

Stratification

1. Previous radical prostatectomy (yes or no)
2. Planned docetaxel use (yes or no)
3. Clinical or pathological pelvic LN involvement (yes or no)

Niazi T, et al. *ASCO* 2022. Abstract TPS5103. ClinicalTrials.gov Identifier: NCT04136353.

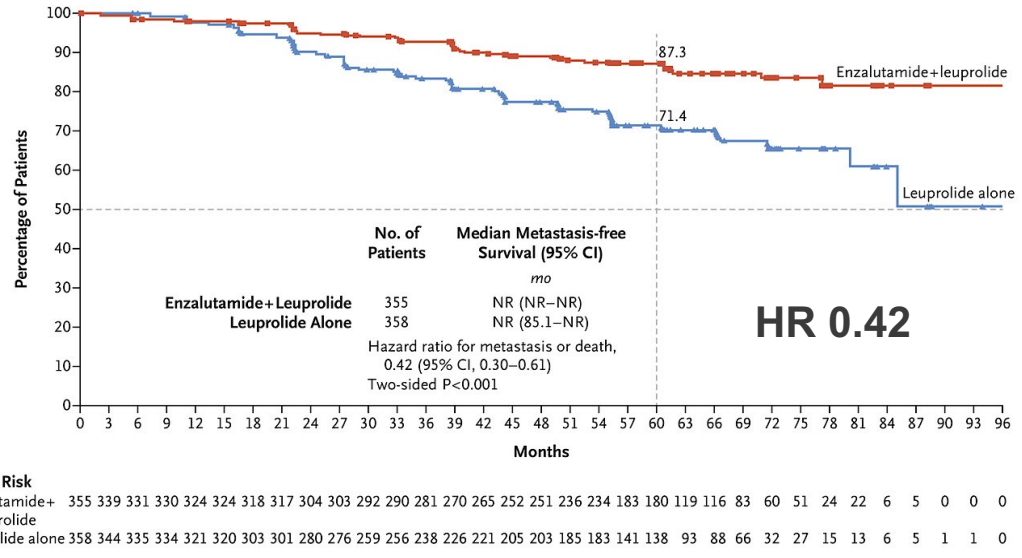
EMBARK: Study Design



Freedland, SJ. et al. *NEJM* 2023; 389: 1453. Shore, ND. et al. *NEJM.* 2026 Feb 5;394(6):563-575.
Shore, ND. et al. ESMO 2025;Abstract LBA87.

EMBARK study: MFS (primary)

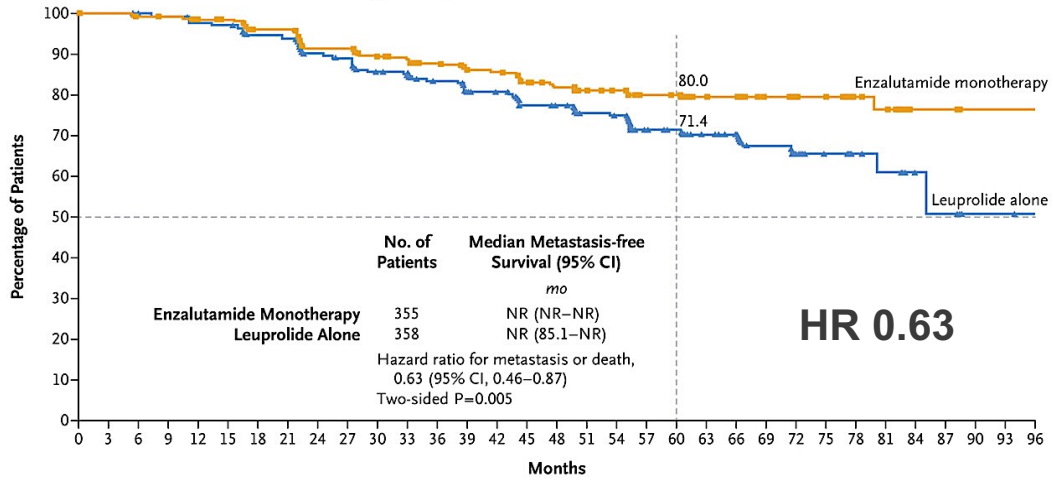
Enza Combo vs. Leuprolide Alone



No. at Risk

Enzalutamide+ leuprolide	355	339	331	330	324	324	318	317	304	303	292	290	281	270	265	252	251	236	234	183	180	119	116	83	60	51	24	22	6	5	0	0	0
Leuprolide alone	358	344	335	334	321	320	303	301	280	276	259	256	238	226	221	205	203	185	183	141	138	93	88	66	32	27	15	13	6	5	1	1	0

Enza Mono vs. Leuprolide Alone



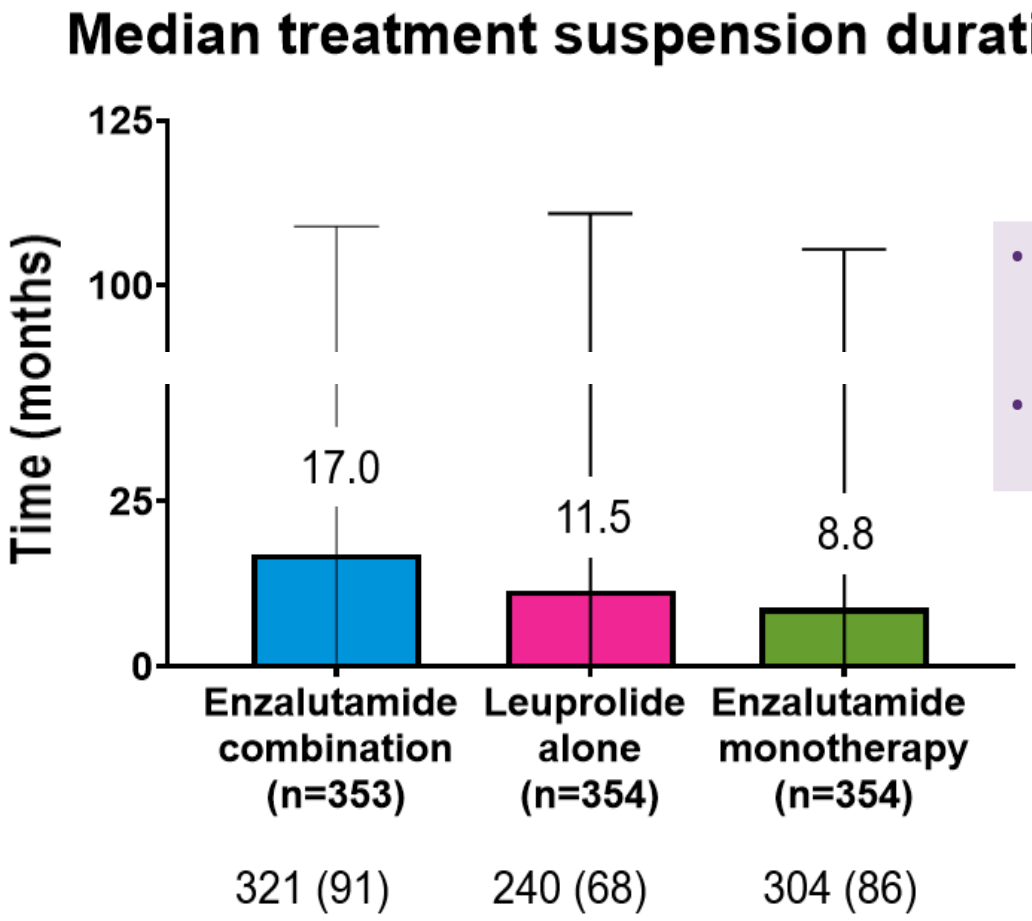
No. at Risk

Enzalutamide monotherapy	355	350	342	341	328	326	309	309	287	287	273	269	260	248	247	235	228	211	209	172	171	109	108	76	52	49	26	24	5	5	0	0	0
Leuprolide alone	358	344	335	334	321	320	303	301	280	276	259	256	238	226	221	205	203	185	183	141	138	93	88	66	32	27	15	13	6	5	1	1	0

Median follow-up: 5 years **Median PSADT: 4.9 months (at entry)** **Median PSA: 5.2**

Freedland, SJ. et al. *NEJM* 2023; 389: 1453.

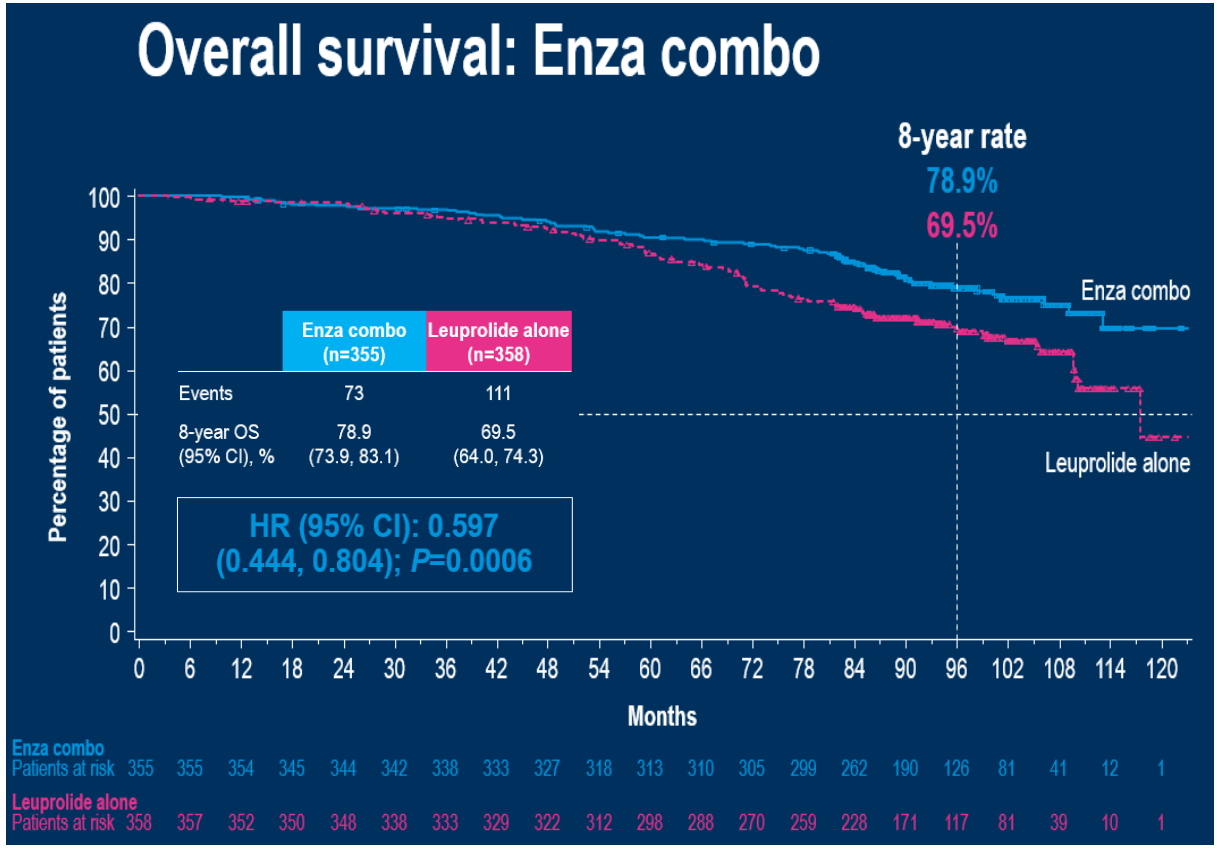
EMBARK: Treatment Suspension

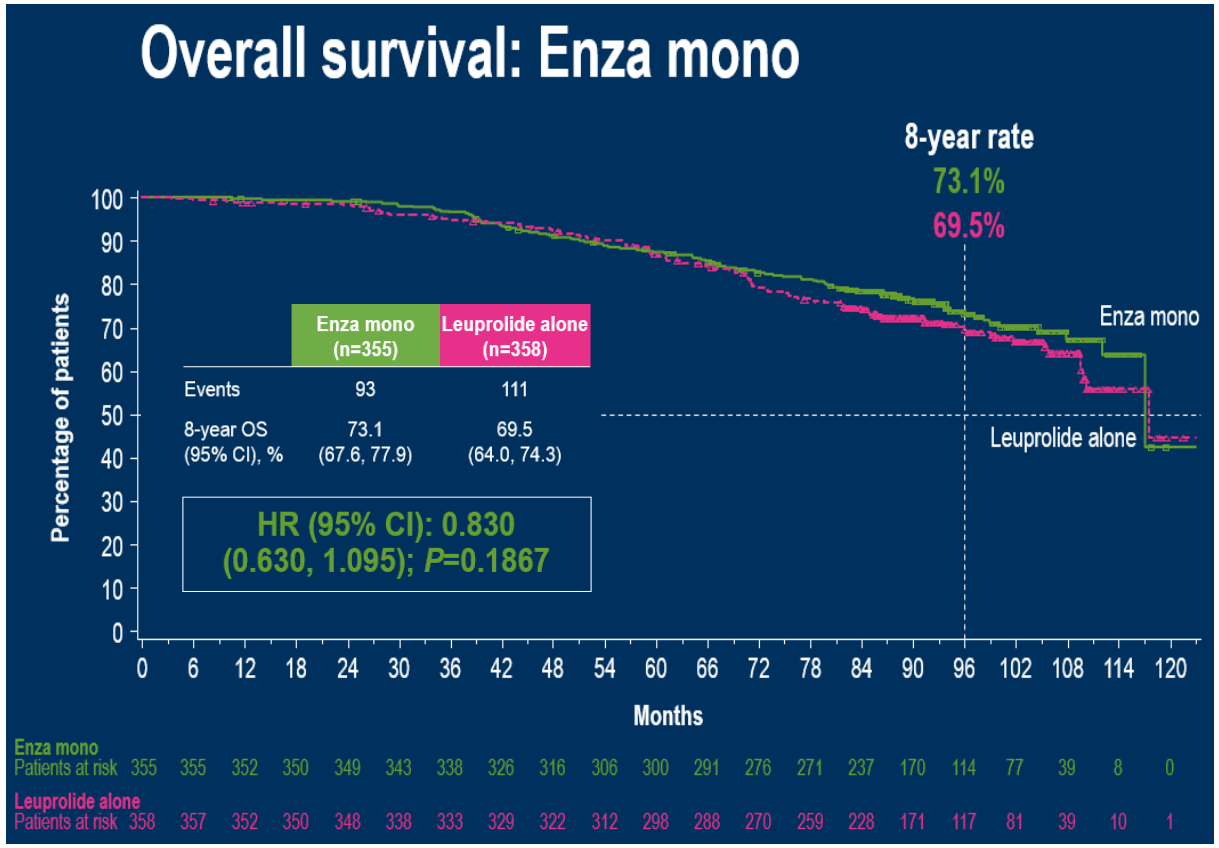


- Enzalutamide combination versus leuprolide alone: *P*<0.0001.†
- Enzalutamide monotherapy versus leuprolide alone: *P*=0.286.†

Shore, ND. et al. *NEJM.* 2026 Feb 5;394(6):563-575.

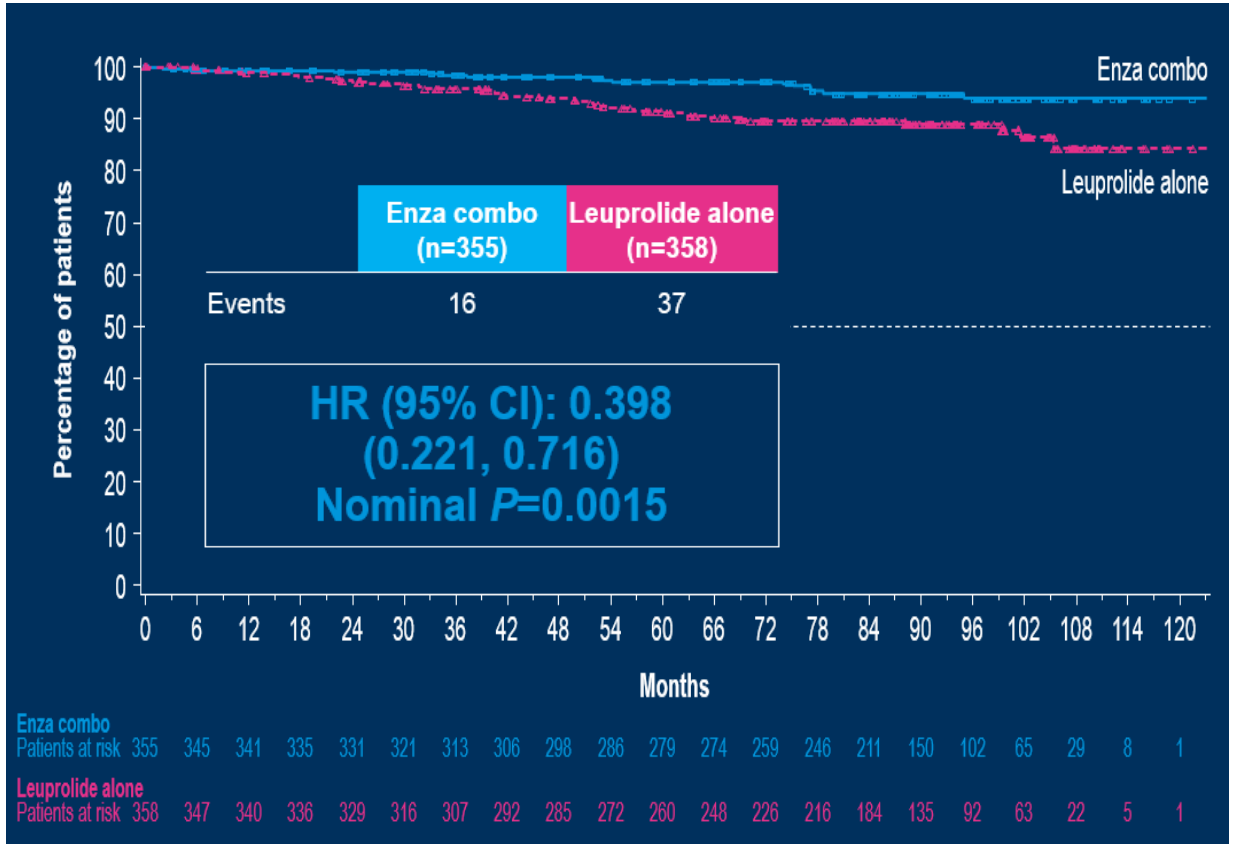
EMBARK: Overall Survival

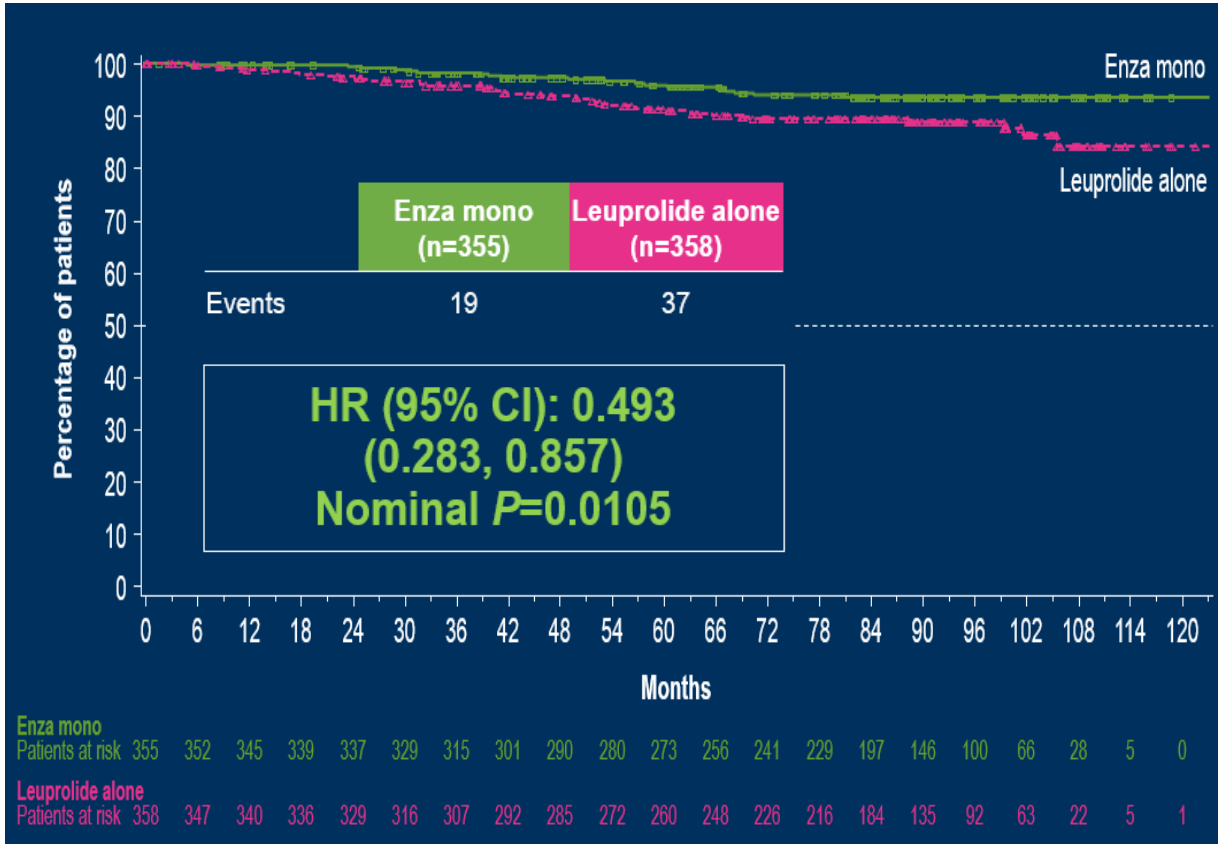




Shore, ND. et al. *NEJM.* 2026 Feb 5;394(6):563-575. Shore, ND. et al. ESMO 2025;Abstract LBA87

EMBARK: Time to First SSE





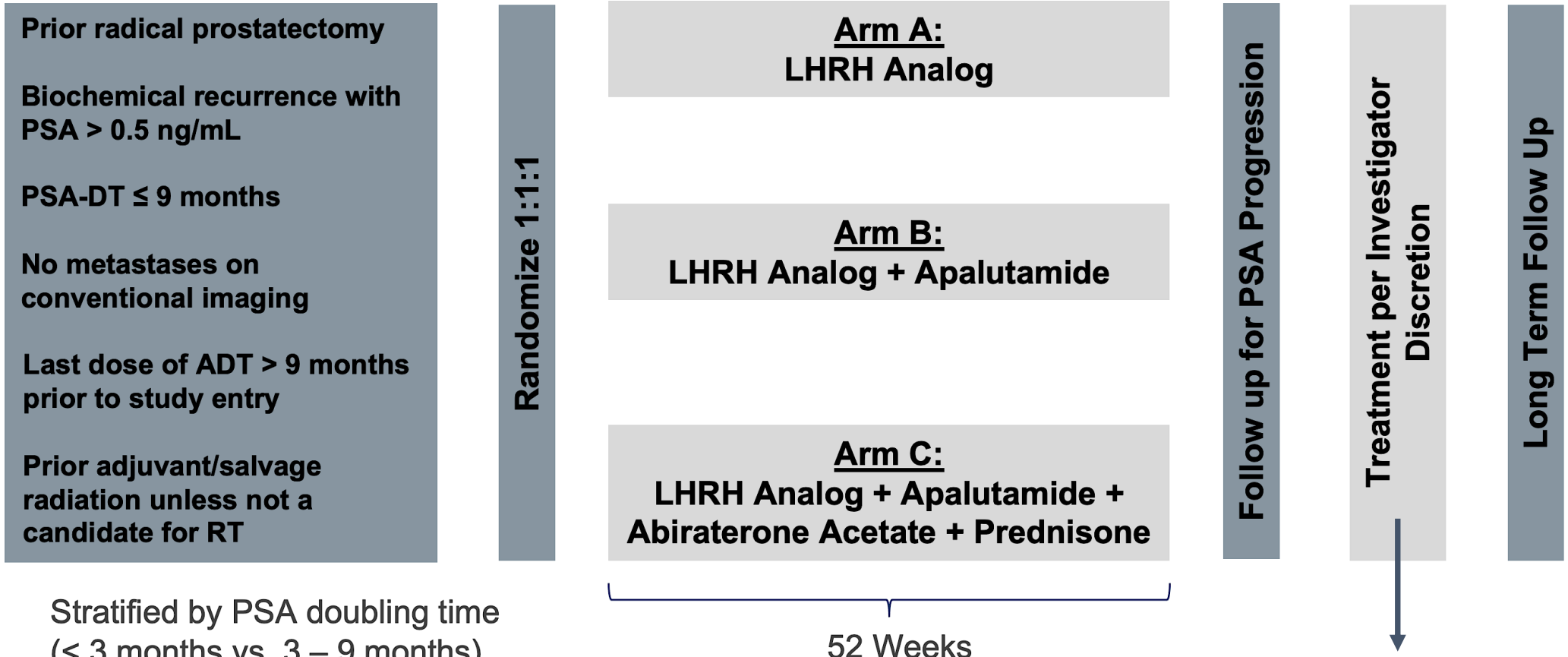
Shore, ND. et al. *NEJM.* 2026 Feb 5;394(6):563-575. Shore, ND. et al. ESMO 2025;Abstract LBA87

EMBARK: Adverse Events

Event, n (%)	Enza combo (N=353)	Leuprolide (N=354)	Enza mono (N=354)
Most common TEAEs (occurring in at least 15%)†			
Hot flash	246 (69.7)‡	206 (58.2)‡	80 (22.6)
Fatigue	154 (43.6)‡	119 (33.6)‡	170 (48.0)‡
Arthralgia	104 (29.5)	75 (21.2)	89 (25.1)
Fall	104 (29.5)	60 (16.9)	71 (20.1)
Hypertension	92 (26.1)	75 (21.2)	76 (21.5)
Back pain	62 (17.6)	56 (15.8)	67 (18.9)
Diarrhea	55 (15.6)	31 (8.8)	47 (13.3)
Constipation	53 (15.0)	35 (9.9)	38 (10.7)
Haematuria	50 (14.2)	57 (16.1)	53 (15.0)
Nausea	43 (12.2)	31 (8.8)	57 (16.1)
Gynecomastia	31 (8.8)	32 (9.0)	163 (46.0)‡
Nipple pain	13 (3.7)	4 (1.1)	54 (15.3)

Shore, ND. et al. *NEJM.* 2026 Feb 5;394(6):563-575. Shore, ND. et al. ESMO 2025;Abstract LBA87

PRESTO: Study Schema



Aggarwal, Rahul et al. *ESMO* 2025

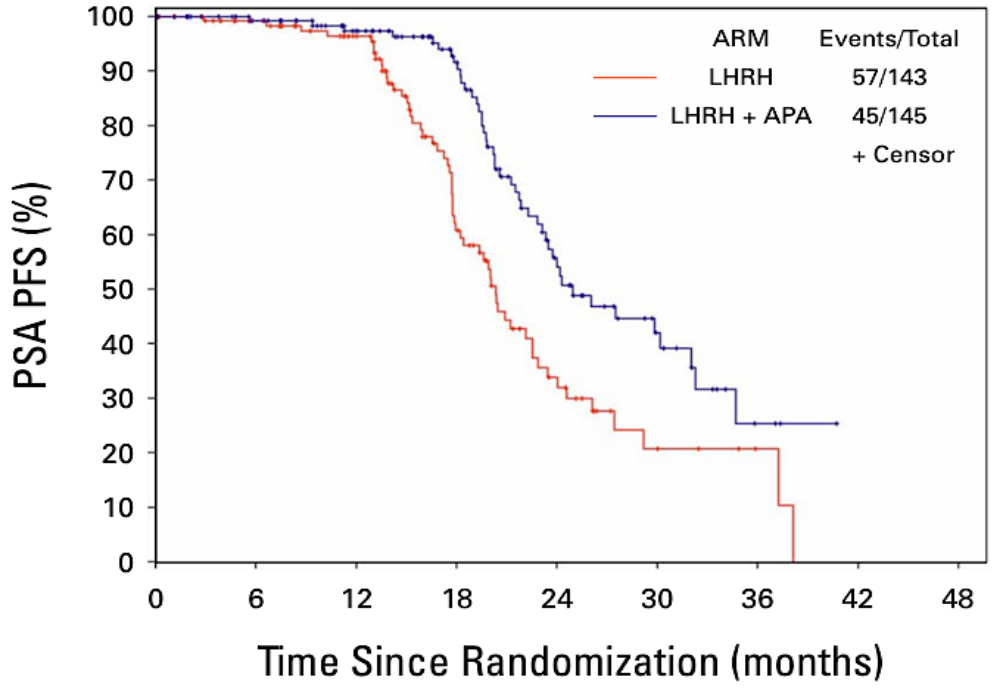
Baseline Characteristics

	Arm A: ADT (N = 166)	Arm B: ADT + Apalutamide (N = 168)	Arm C: ADT + Apalutamide + Abi/pred (N = 169)	Overall Study Cohort (N =503)
Median PSA at study entry, ng/mL (Q1, Q3)	1.7 (1.0, 3.2)	1.8 (1.0, 3.5)	1.8 (1.0, 4.2)	1.8 (1.0,3.6)
PSA doubling time strata (%)				
< 3 months	43 (25.9)	43 (25.6)	44 (26.0)	130 (25.8)
3 – 9 months	123 (74.1)	125 (74.4)	125 (74.0)	373 (74.2)
Gleason Grade at Diagnosis				
6-7	102 (61.4)	102 (60.7)	105 (62.1)	309 (61.4)
8	15 (9.0)	22 (13.1)	19 (11.2)	56 (11.1)
9-10	49 (29.5)	43 (25.6)	44 (26.0)	136 (27.0)
Unknown	0	1 (0.6)	1 (0.6)	2 (0.4)
Median time interval between radical prostatectomy and study entry, years (Q1, Q3)	4.6 (2.9, 7.3)	4.7 (2.8, 6.5)	4.0 (2.8, 6.9)	4.5 (2.8, 6.8)
Prior adjuvant or salvage radiation, n (%)	147 (88.6)	143 (85.1)	138 (81.7)	428 (85.1)
Prior androgen deprivation therapy, n (%)	71 (42.8)	75 (44.6)	67 (39.6)	213 (42.35)

Aggarwal, R. et al. *ESMO* 2025

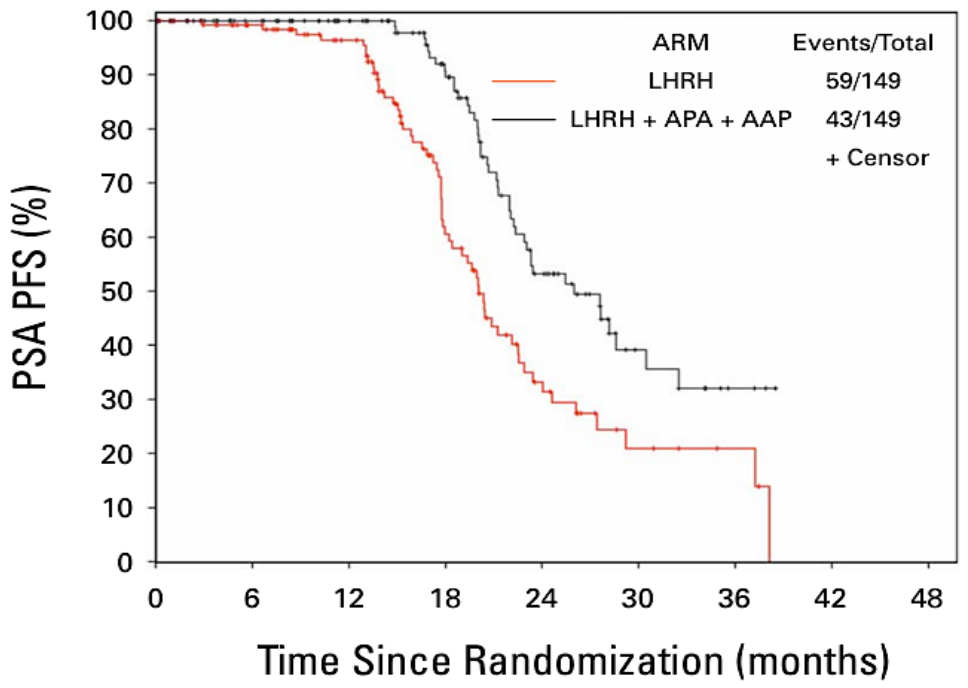
Primary Objective: PSA-based PFS

ADT + Apalutamide vs. ADT



No. at risk:					
LHRH	143	94	18	2	0
LHRH + APA	145	101	32	3	0

ADT + Apalutamide + AAP vs. ADT

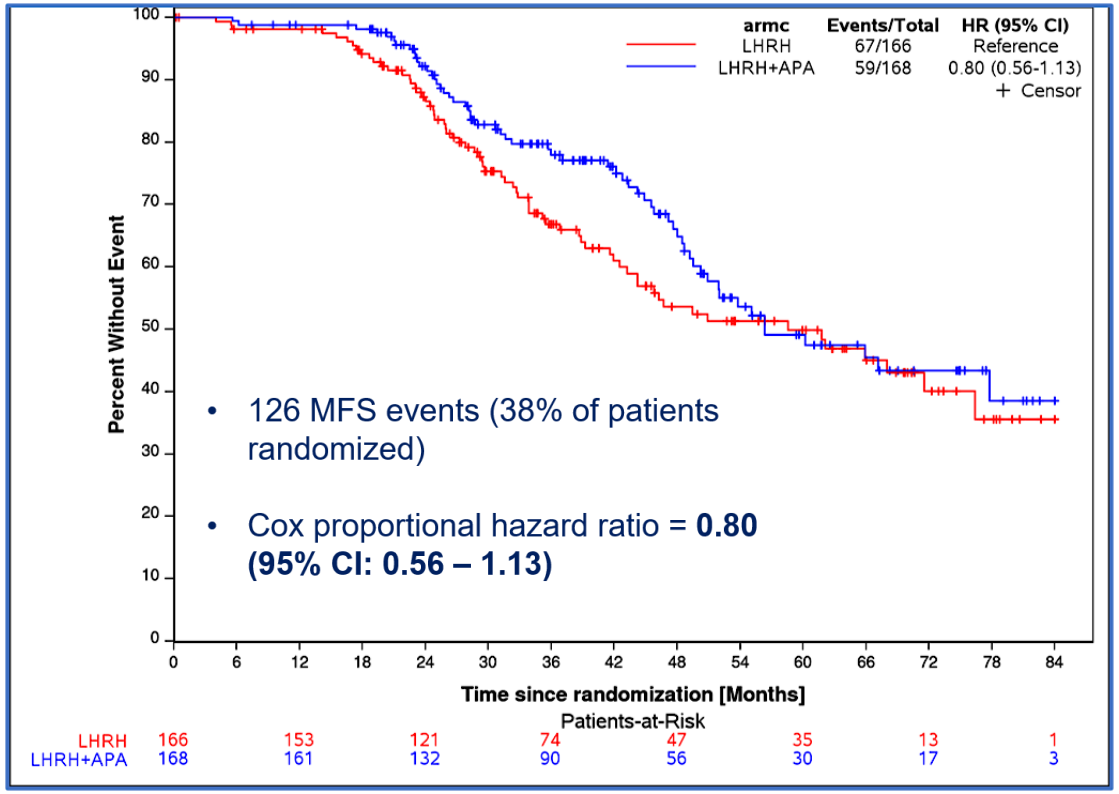


No. at risk:					
LHRH	149	97	18	3	0
LHRH + APA + AAP	149	103	35	3	0

Aggarwal, R. et al. *J Clin Oncol* 2024

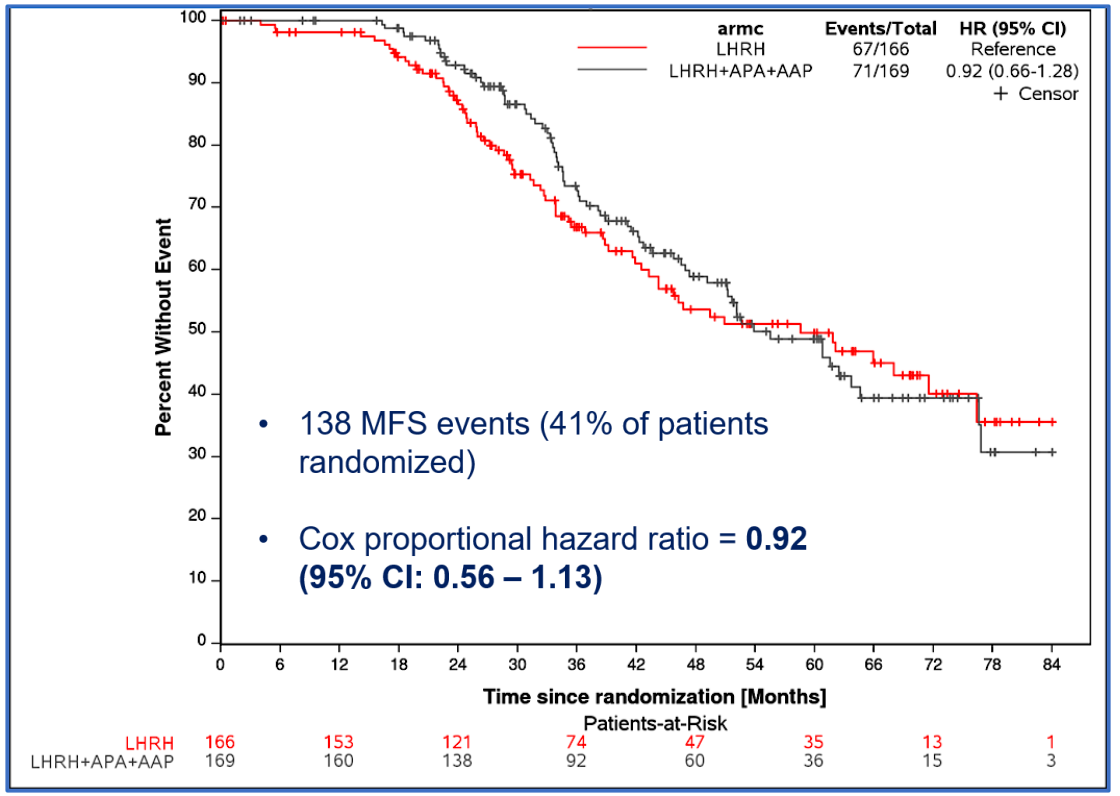
Key Secondary Objective: MFS

ADT + Apalutamide vs. ADT



- 126 MFS events (38% of patients randomized)
- Cox proportional hazard ratio = **0.80 (95% CI: 0.56 – 1.13)**

ADT + Apalutamide + AAP vs. ADT



- 138 MFS events (41% of patients randomized)
- Cox proportional hazard ratio = **0.92 (95% CI: 0.56 – 1.13)**

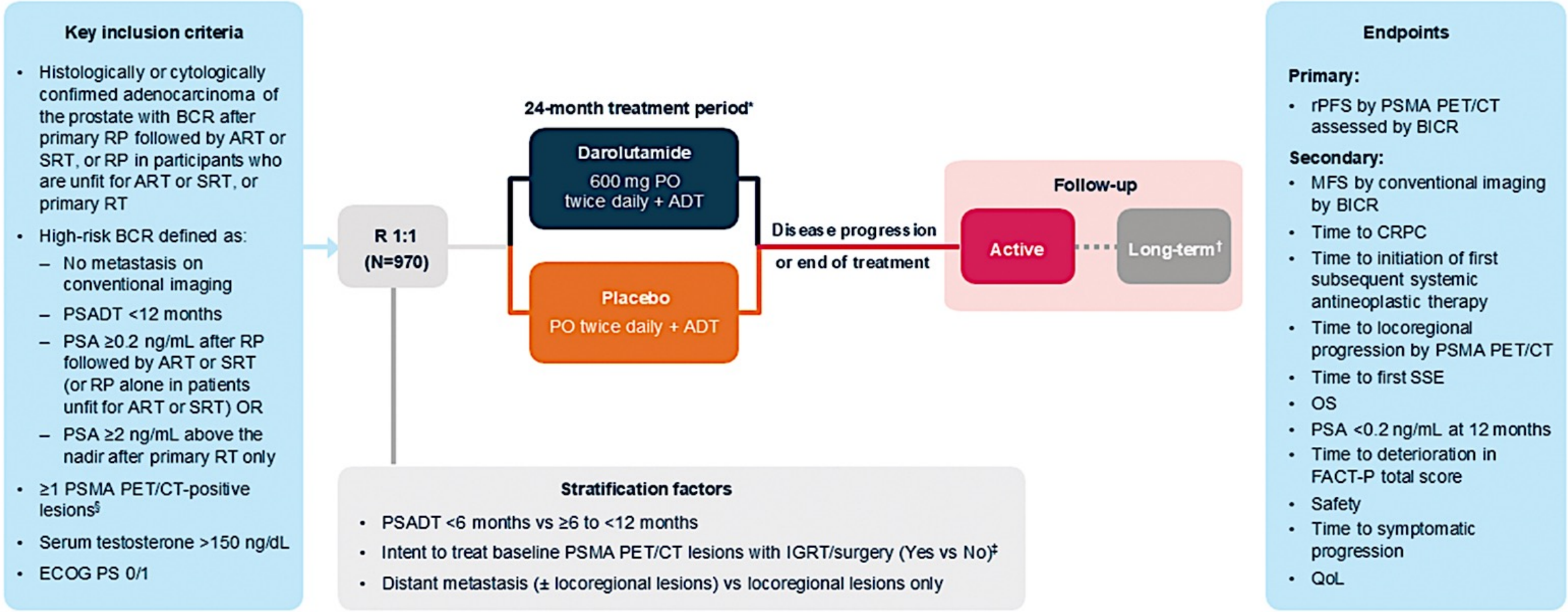
Aggarwal, R. et al. *ESMO* 2025

Adverse Events

	Arm A ADT (n = 160)		Arm B ADT + apalutamide (n = 167)		Arm C ADT + apalutamide + abi/pred (n = 164)	
Adverse Events (AE)	Grade 2	Grade ≥ 3	Grade 2	Grade ≥ 3	Grade 2	Grade ≥ 3
	n (%)		n (%)		n (%)	
Hypertension	19 (12)	12 (8)	30 (18)	13 (8)	20 (12)	34 (21)
Hot flashes	22 (14)	1 (1)	12 (7)	1 (1)	24 (15)	1 (1)
Fatigue	16 (10)	0	9 (5)	3 (2)	19 (12)	2 (1)
Injection site reaction	11 (7)	0	11 (7)	0	11 (7)	0
Insomnia	11 (7)	0	6 (4)	0	8 (5)	0
Hyperglycemia	1 (1)	3 (2)	7 (4)	2 (1)	6 (4)	6 (4)
Rash	2 (1)	1 (1)	8 (5)	3 (2)	3 (2)	5 (3)
Erectile dysfunction	9 (6)	1 (1)	6 (4)	1 (1)	3 (2)	0
Arthralgia	4 (3)	1 (1)	4 (2)	0	2 (1)	0
Fall	0	3 (2)	7 (4)	0	4 (2)	0

Aggarwal, R. et al. *ESMO* 2025

Phase 3 ARASTEP trial: Study Design



Morgan AK, et al. *Future Oncol*. 2025;21(27):3491-3498.

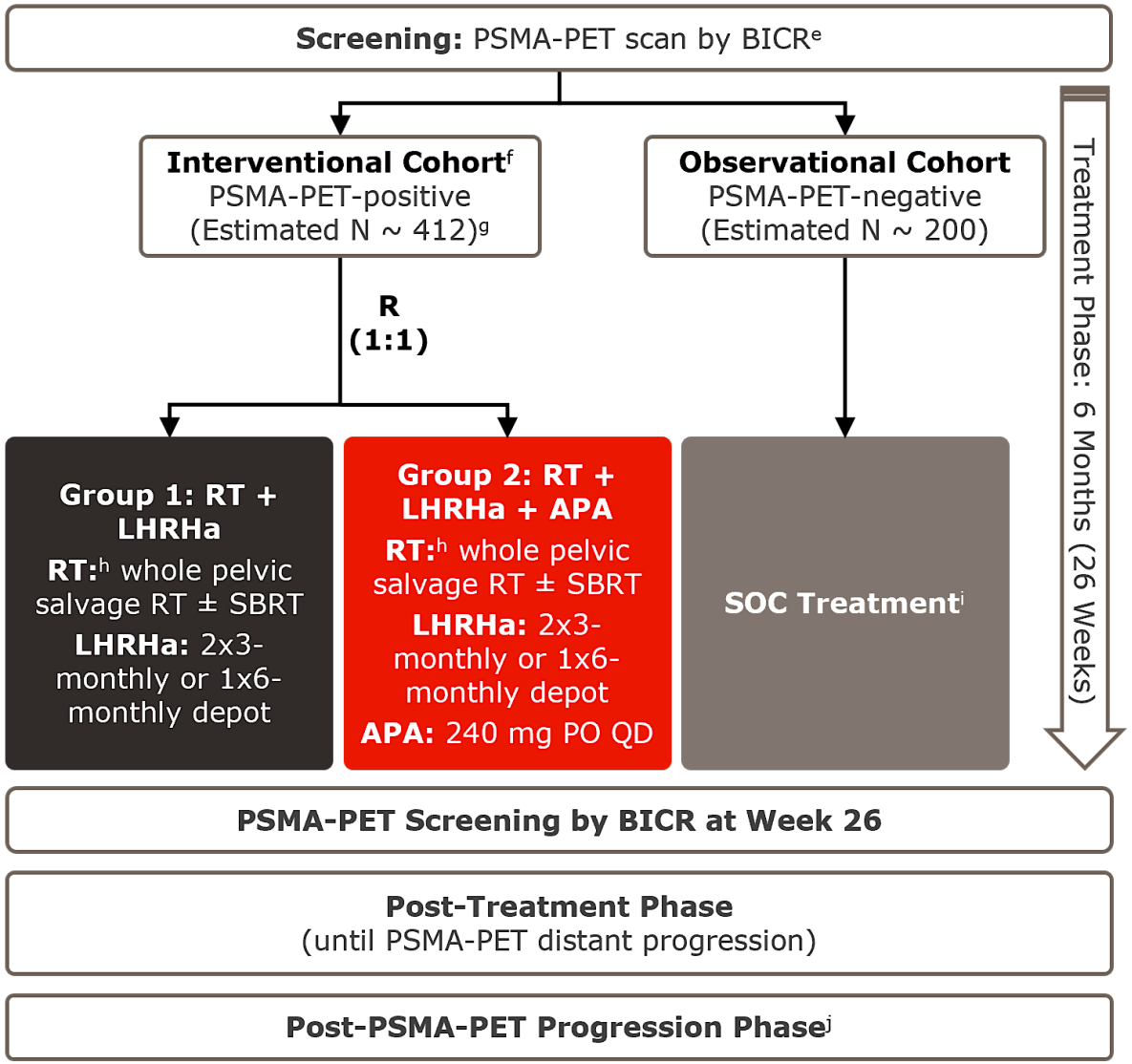
Phase 3 PRIMORDIUM Trial: Study Design

Select Inclusion Criteria

- Histologically confirmed adenocarcinoma of the prostate
- Previously underwent RP ± LND, with postoperative PSA levels of <0.1 ng/mL[a]
- BCR after RP[b] with high-risk for metastasis[c]
- No evidence of metastases on screening CT/MRI of the chest/abdomen/pelvis by ^{99}mTc whole-body bone scan
- ECOG PS 0-1

Primary Endpoint

- ppMPFS[k]



ClinicalTrials.gov: NCT04557059.

Discussion Questions

74 y/o M s/p primary radiation therapy, now with biochemically recurrent prostate cancer with PSA doubling time of 6 mo. Started EMBARK regimen of leuprolide and enzalutamide with good PSA response, planning to stop at 9 mo.

How would you manage this patient moving forward? How do you employ intermittent therapy, and will you discontinue and resume treatment more than once?

What are your criteria to treat a patient with M0 disease with PSA recurrence? What options do you offer and recommend?

In which situations, if any, will you use enzalutamide monotherapy, and how do you reduce the risk of gynecomastia?

RTP
RESEARCH
TO PRACTICE

Discussion Questions

64 y/o man with locally advanced prostate cancer, Gleason score 8 and PSA 45, treated with XRT, ADT and abiraterone/prednisone x 3 years, but 6 months after stopping developed rapidly doubling PSA. PSMA PET negative.

What would you do next for this patient?

What would you do if the PSMA PET showed widespread disease? What about oligometastatic disease?

What are your thoughts about the PROTEUS trial? Any predictions? If this trial is positive, will you use this approach in your practice? For whom?

RTP
RESEARCH
TO PRACTICE

Agenda

Module 1: Evolving Management of Nonmetastatic Hormone-Sensitive Prostate Cancer (HSPC) — Dr Antonarakis

Module 2: Current and Future Hormonal Treatment for Metastatic HSPC (mHSPC) — Dr Aggarwal

Module 3: Current and Future Role of Regimens Combining PARP Inhibitors and AR Pathway Inhibitors in Metastatic Prostate Cancer — Dr Abida

Module 4: Emerging Role of AKT Inhibition for Patients with mHSPC — Dr Fizazi

Module 5: Current and Future Use of Radiopharmaceuticals in Metastatic Prostate Cancer — Dr McKay

RTP RESEARCH TO PRACTICE

UCSF Helen Diller Family Comprehensive Cancer Center

Current and Future Approaches to Hormonal Therapy for Metastatic Hormone-Sensitive Prostate Cancer

Rahul Aggarwal MD
Professor of Medicine
University of California San Francisco





Outline

- Summary of the results of extended follow up from phase 3 doublet and triplet clinical trials
- Choosing between regimens: Decision factors and potential biomarkers
- Emerging hormonal treatment strategies for mHSPC

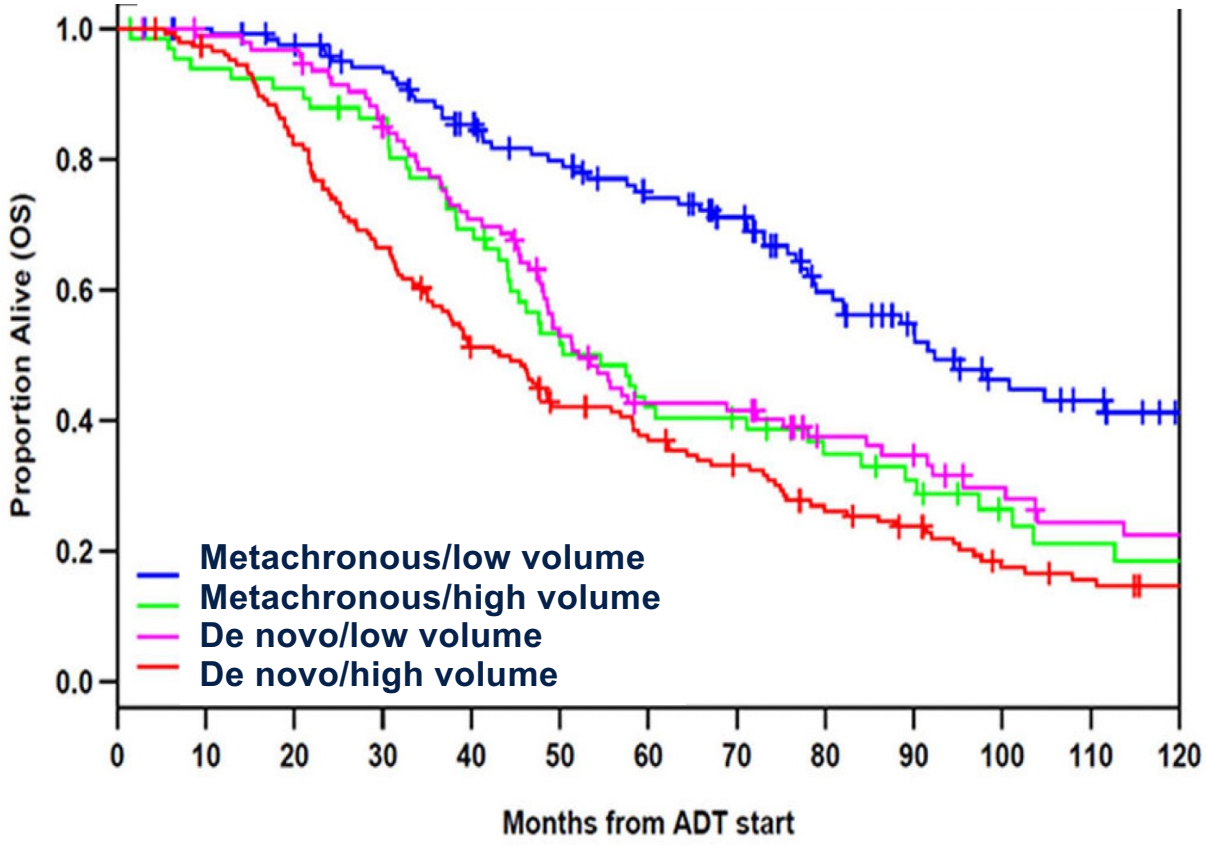
Definition of Low vs. High Volume Disease (by conventional imaging)

- CHAARTED definition
  - Visceral metastases and/or 4 or more bone metastases with at least one outside axial column

- LATITUDE:
  - Two or more: Visceral metastases, ≥ 3 bone metastases, Gleason ≥ 8

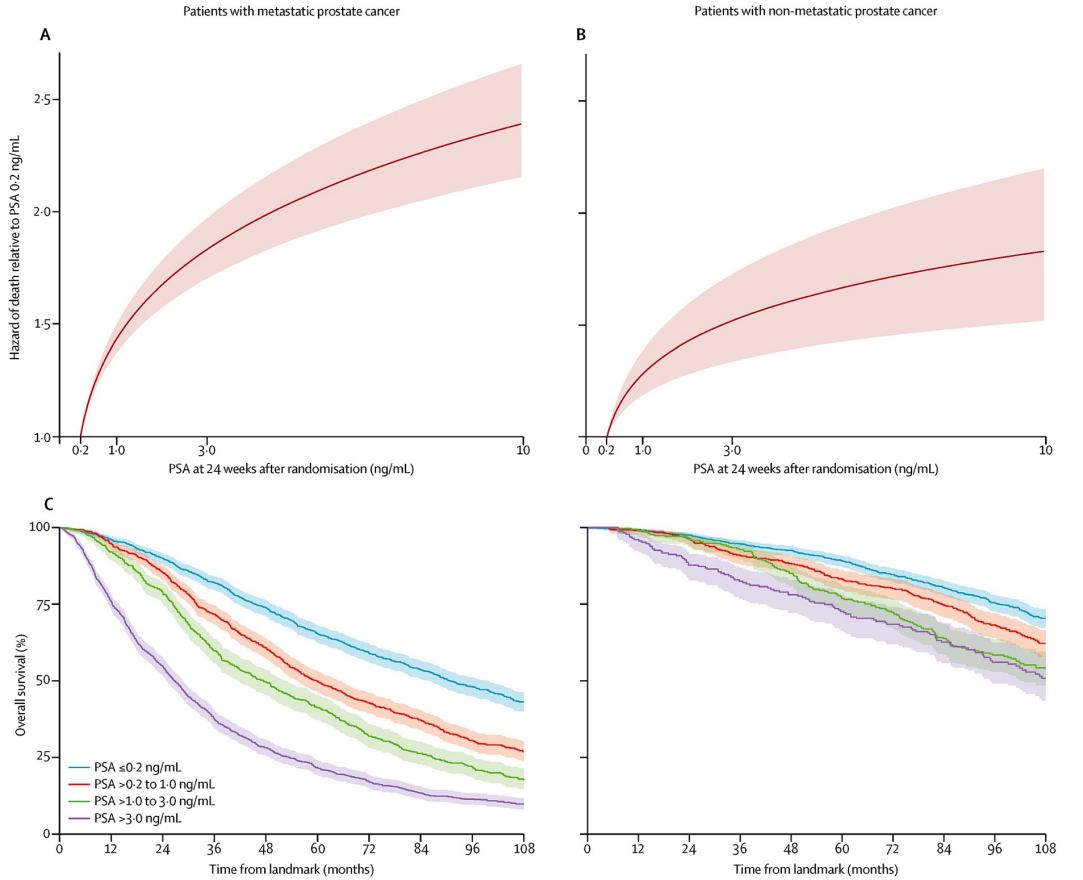
Prognosis of mHSPC According to Disease Volume and Timing of Metastases

Groups	N (% events)	mOS, years (95% CI)
Metachronous/ low volume	125 (50)	7.7 (6.7-10.6)
Metachronous/ high volume	67 (75)	4.6 (3.7-6.7)
De novo/ low volume	96 (70)	4.3 (4.0-6.5)
De novo/ high volume	148 (84)	3.6 (3.1-4.7)



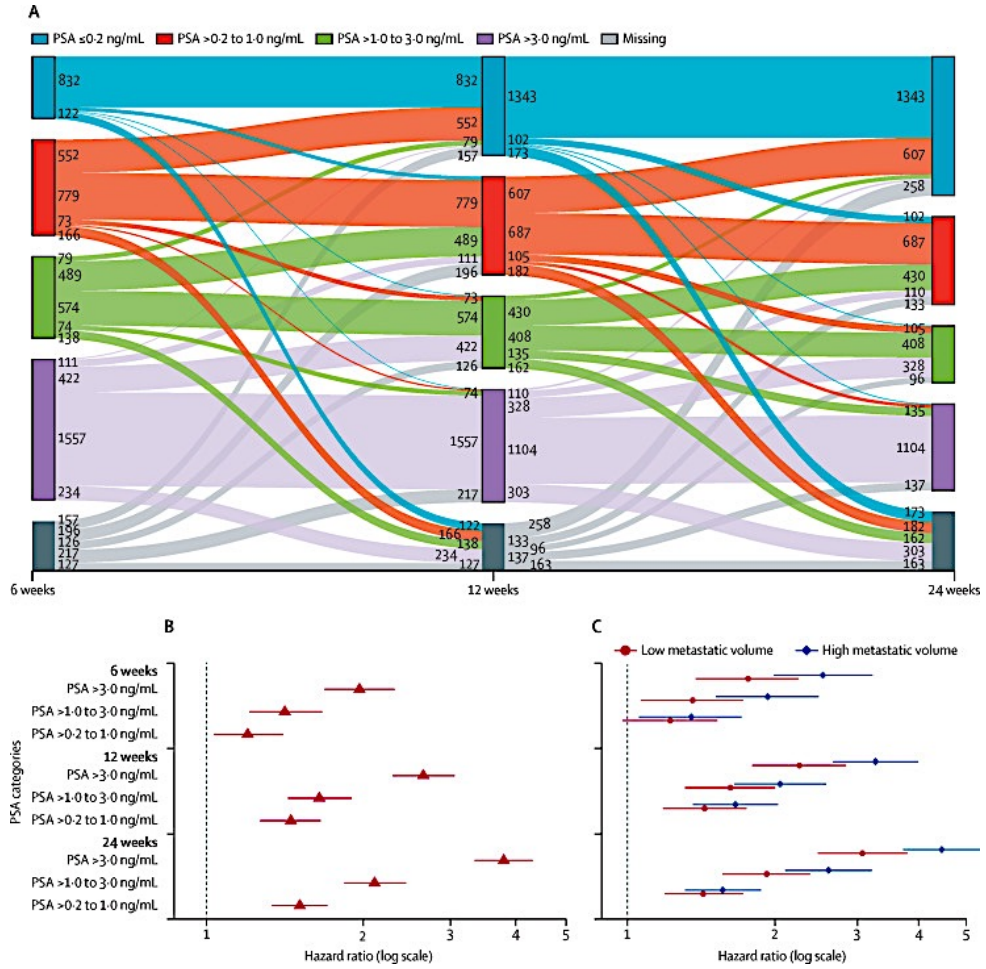
Francini E, et al. Prostate 2018

Depth of PSA Nadir and Disease Volume are Prognostic for Overall Survival



Number at risk (censored)

PSA ≤0·2 ng/mL	1164 (0)	1113 (7)	1041 (9)	945 (12)	837 (27)	726 (44)	635 (65)	560 (82)	400 (191)	247 (309)	1139 (0)	1122 (8)	1101 (10)	1066 (12)	1039 (16)	976 (42)	895 (74)	825 (100)	602 (276)	354 (492)
PSA >0·2 to 1·0 ng/mL	910 (0)	861 (2)	770 (8)	644 (11)	536 (21)	432 (30)	361 (41)	299 (57)	196 (112)	131 (156)	552 (0)	543 (4)	526 (6)	495 (7)	481 (7)	443 (18)	419 (25)	381 (36)	276 (109)	182 (182)
PSA >1·0 to 3·0 ng/mL	594 (0)	547 (0)	469 (1)	355 (1)	293 (3)	238 (9)	179 (15)	142 (21)	102 (39)	64 (59)	352 (0)	346 (2)	336 (3)	322 (5)	294 (6)	261 (11)	239 (18)	207 (22)	165 (47)	114 (88)
PSA >3·0 ng/mL	1206 (0)	910 (6)	656 (9)	447 (12)	333 (15)	251 (21)	193 (28)	145 (34)	102 (57)	71 (75)	229 (0)	220 (0)	200 (1)	188 (1)	177 (2)	159 (8)	144 (14)	128 (18)	102 (31)	72 (53)



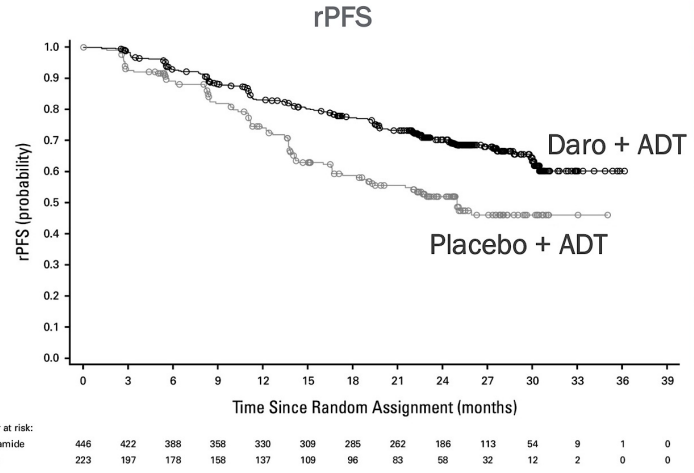
Kayani M, et al. Lancet Oncol 2026

Design Characteristics of the Phase 3 Doublet Studies

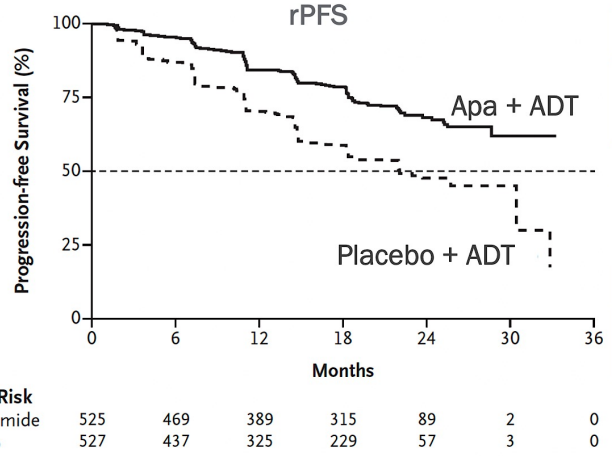
	ARANOTE[1]		TITAN[2]		ARCHES[3]		LATITUDE[4]	
	Daro + ADT	ADT	Apa + ADT	ADT	Enza + ADT	ADT	Abi + ADT	ADT
Patients, n	446	223	525	527	574	576	597	602
Key inclusion criteria	▪ ECOG PS 0-2 ▪ ADT started within 12 weeks before randomization		▪ ECOG PS 0-1 ▪ ADT started ≤6 months before randomization for mHSPC or ≤3 years for local disease		▪ ECOG PS 0-1 ▪ ADT started within 3 months before randomization (<6 months for Doce) without progression or rising PSA levels		▪ ECOG PS 0-2 ▪ High-risk disease (≥2 of following: Gleason score ≥8, ≥3 lesions on bone scan, measurable visceral lesion) ▪ No prior Doce or localized treatment	
Primary endpoint(s)	rPFS		OS, rPFS		rPFS		OS, rPFS	
Age, y	70	70	69	68	70	70	68	67
Prior docetaxel, %	0	0	11	10	18	18	0	0
High volume, %	71	70	62	64	62	65	82	78
Region, %	Asia (31); Latin America (29); Europe and rest of world (41) US sites: No		N. America and European Union (33); Other (67) US sites: Yes		Europe (59); Asia-Pacific (18); N. and S. America (21); Other (2) US sites: Yes		Europe, Asia–Pacific, Latin America, Canada (NA) US sites: No	
Enrollment period	Mar 2021 – Aug 2022		Dec 2015 – July 2017		Mar 2016 – January 2018		Feb 2013 – Dec 2014	

1. Saad F, et al. J Clin Oncol 2024; 2. Chi KN, et al. NEJM 2019; 3. Armstrong AJ, et al. J Clin Oncol 2019; 4. Fizazi K, et al. NEJM 2017

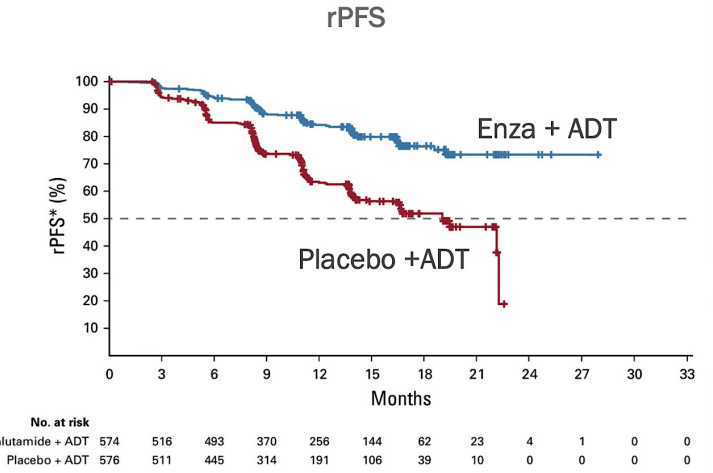
Radiographic Progression-Free Survival



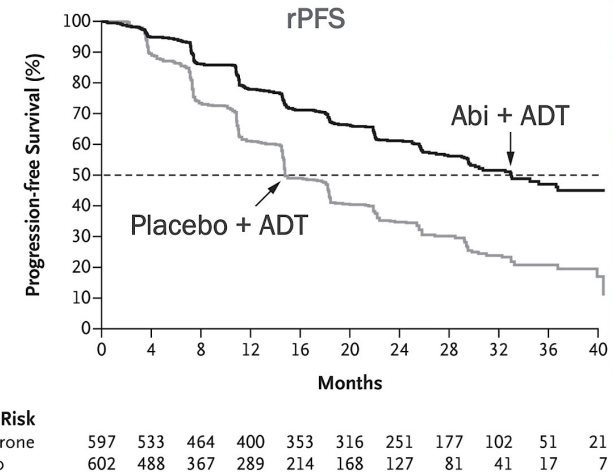
ARANOTE[1]	Daro + ADT	ADT
mPFS, mo	NR	25
HR (95% CI)	0.54 (0.41-0.71)	
mOS, mo	NR	NR
HR (95% CI)	0.78 (0.58-1.05)[a]	



TITAN[2]	Apa + ADT	ADT
mPFS, mo	NR	22.1
HR (95% CI)	0.48 (0.39-0.61)	
mOS, mo	NR	52.2
HR (95% CI)	0.65 (0.53-0.79)	



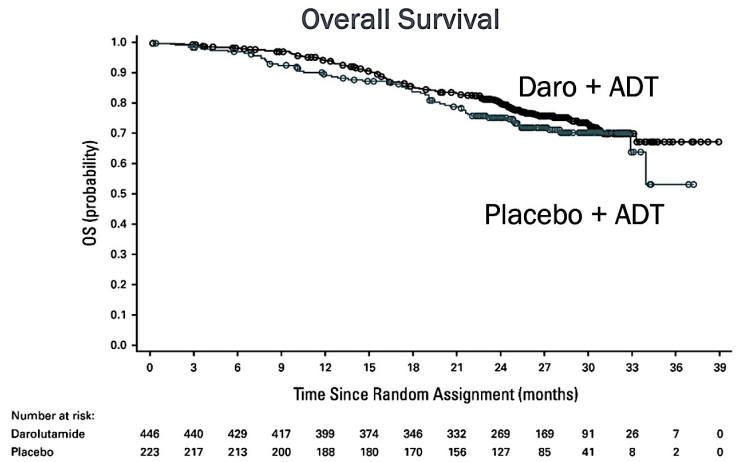
ARCHES[3]	Enza + ADT	ADT
mPFS, mo	NR	19
HR (95% CI)	0.39 (0.30-0.50)	
mOS, mo	NR	NR
HR (95% CI)	0.66 (0.53-0.81)	
mOS (5-yr)	NR	NR
HR (95% CI)	0.70 (0.58-0.85)	



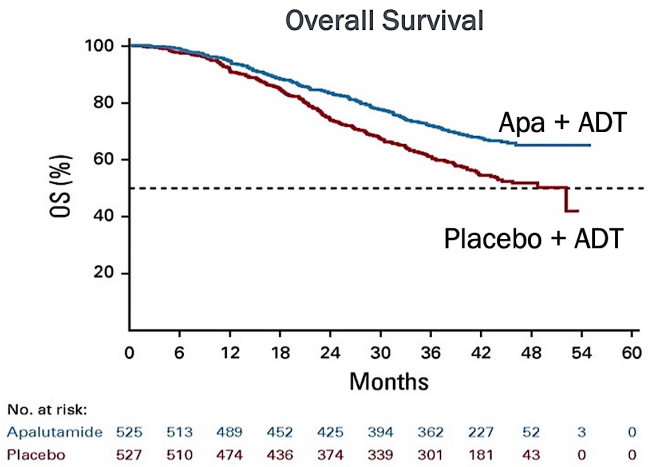
LATITUDE[4]	Abi + ADT	ADT
mPFS, mo	33	14.8
HR (95% CI)	0.47 (0.39-0.55)	
mOS, mo	53.3	36.5
HR (95% CI)	0.66 (0.56-0.78)	

1. Saad F, et al. J Clin Oncol 2024; 2. Chi KN, et al. J Clin Oncol 2021; 3. Armstrong AJ, et al. J Clin Oncol 2019; 4. Fizazi K, et al. Lancet Oncol 2019

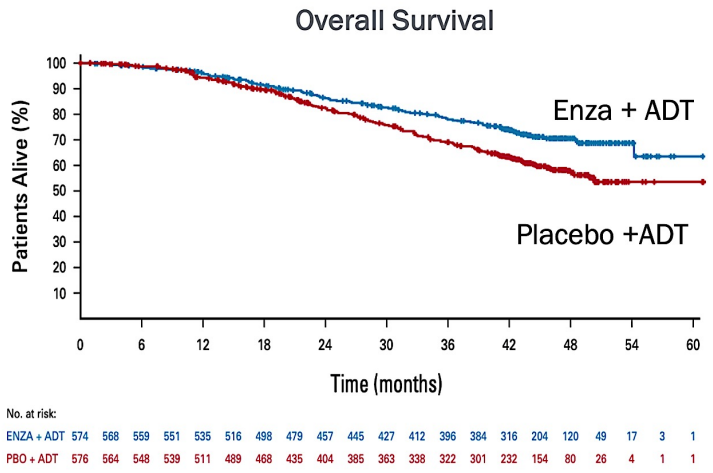
Overall Survival



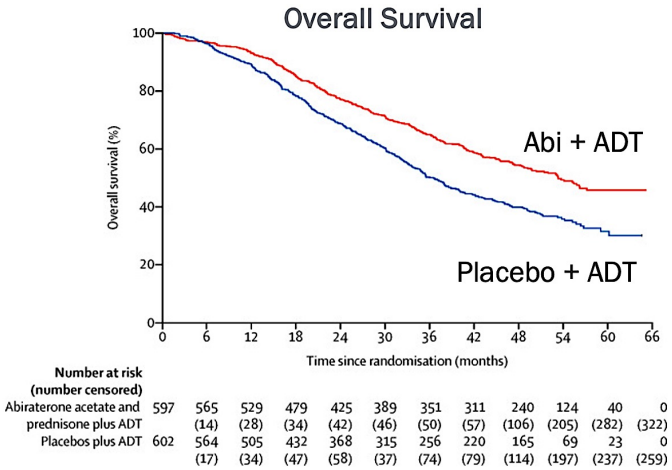
ARANOTE[1]	Daro + ADT	ADT
mPFS, mo	NR	25
HR (95% CI)	0.54 (0.41-0.71)	
mOS, mo	NR	NR
HR (95% CI)	0.78 (0.58-1.05)[a]	



TITAN[2]	Apa + ADT	ADT
mPFS, mo	NR	22.1
HR (95% CI)	0.48 (0.39-0.61)	
mOS, mo	NR	52.2
HR (95% CI)	0.65 (0.53-0.79)	



ARCHES[3]	Enza + ADT	ADT
mPFS, mo	NR	19
HR (95% CI)	0.39 (0.30-0.50)	
mOS, mo	NR	NR
HR (95% CI)	0.66 (0.53-0.81)	
mOS (5-yr)	NR	NR
HR (95% CI)	0.70 (0.58-0.85)	



LATITUDE[4]	Abi + ADT	ADT
mPFS, mo	33	14.8
HR (95% CI)	0.47 (0.39-0.55)	
mOS, mo	53.3	36.5
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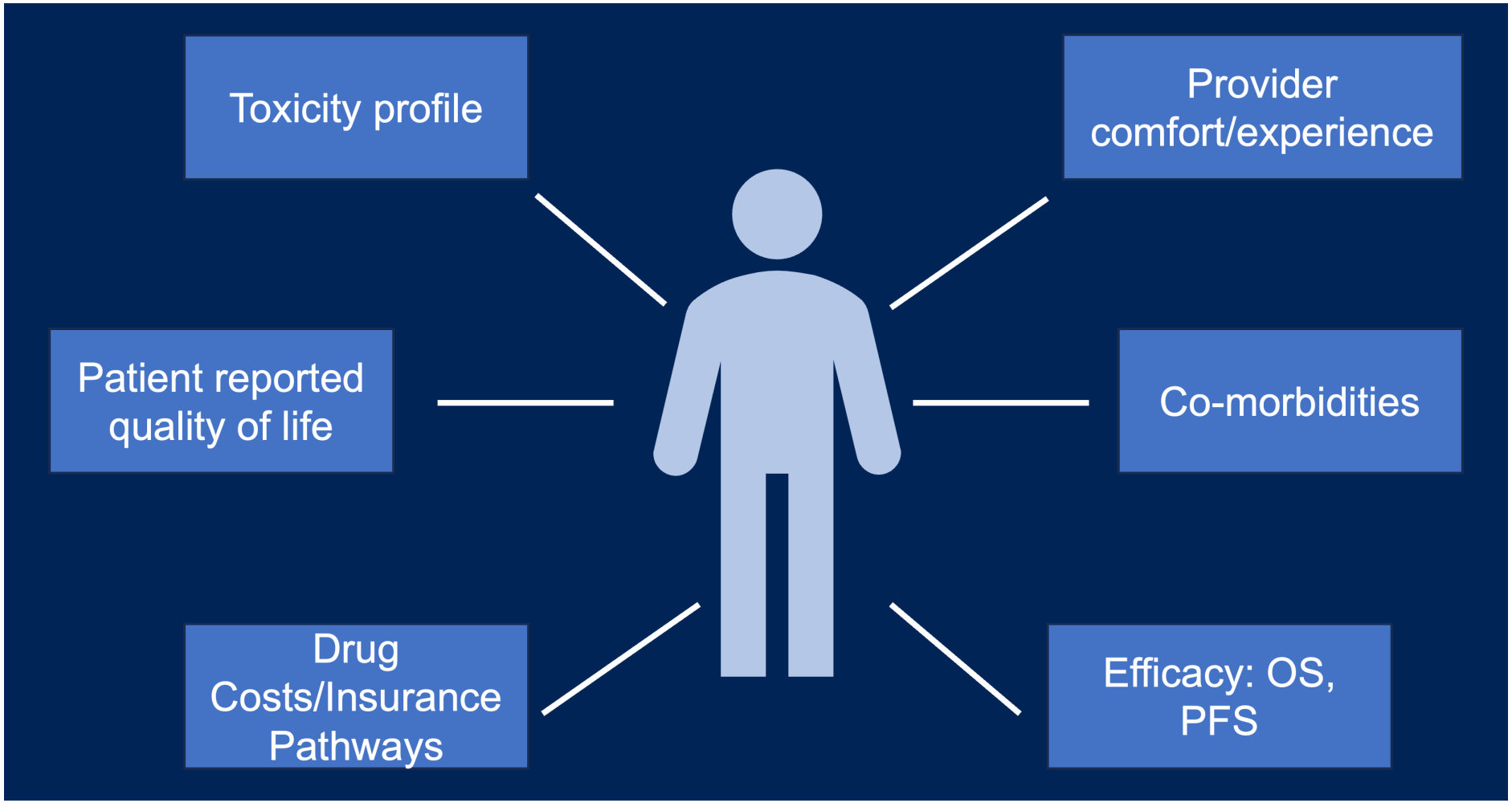
1. Saad F, et al. J Clin Oncol 2024; 2. Chi KN, et al. J Clin Oncol 2021; 3. Armstrong AJ, et al. Eur Urol 2026; 4. Fizazi K, et al. Lancet Oncol 2019

Safety

	ARANOTE[1]		TITAN[2]		ARCHES[3]		LATITUDE[4,5]	
	Daro + ADT	ADT	Apa + ADT	ADT	Enza + ADT	ADT	Abi + ADT	ADT
Median treatment duration, months	24.2	17.3	20.5	18.3	12.8	11.6	24	14
Any grade AE, % Grade ≥3, %	91 36	90 36	97 42	97 41	85 24	86 26	93 63	93 48
AEs leading to discontinuation, %	6	9	8	5	7	5	12	10
Grade ≥3 AE, % active arm vs control	Hypertension, 4.3 vs 3.6 Anemia, 3.1 vs 3.6 Increased AST, 2.2 vs 0.5		Hypertension, 8.4 vs 9.1 Rash, generalized, 2.7 vs 0.4		Hypertension, 3.3 vs 1.7 Fatigue, 0.9 vs 1.0 Back pain, 0.9 vs 0.5		Hypertension, 20 vs 10 Hypokalemia, 10 vs 1 ALT increased, 6 vs 1	
Any grade AESIs, % active arm vs control	Hypertension, 9.4 vs 9.5 Vasodilation/flushing, 9.2 vs 7.2 DM/hyperglycemia, 9.0 vs 9.5 Cardiac arrhythmias, 8.8 vs 6.8 Fatigue, 5.6 vs 8.1 Rash, 4.3 vs 3.6 Fracture, 4.0 vs 2.3		Rash, 27.1 vs 8.5 Falls, 7.4 vs 7.0 Fracture, 6.3 vs 4.6 Hypothyroidism, 6.5 vs 1.1 IHD, 4.4 vs 1.5		Musculoskeletal, 26.4 vs 27.7 Fatigue, 24.1 vs 19.5 Hypertension, 8.6 vs 6.3 Fractures, 6.5 vs 4.2 Cognitive impairment, 4.5 vs 2.1 Falls, 3.7 vs 2.6		Hypertension, 41 vs 24[a] Hepatotoxicity, 24 vs 18[a] Hypokalemia, 24 vs 4[a] Cardiac disorders, 16 vs 9[a] Fluid retention/edema, 14 vs 12[a]	

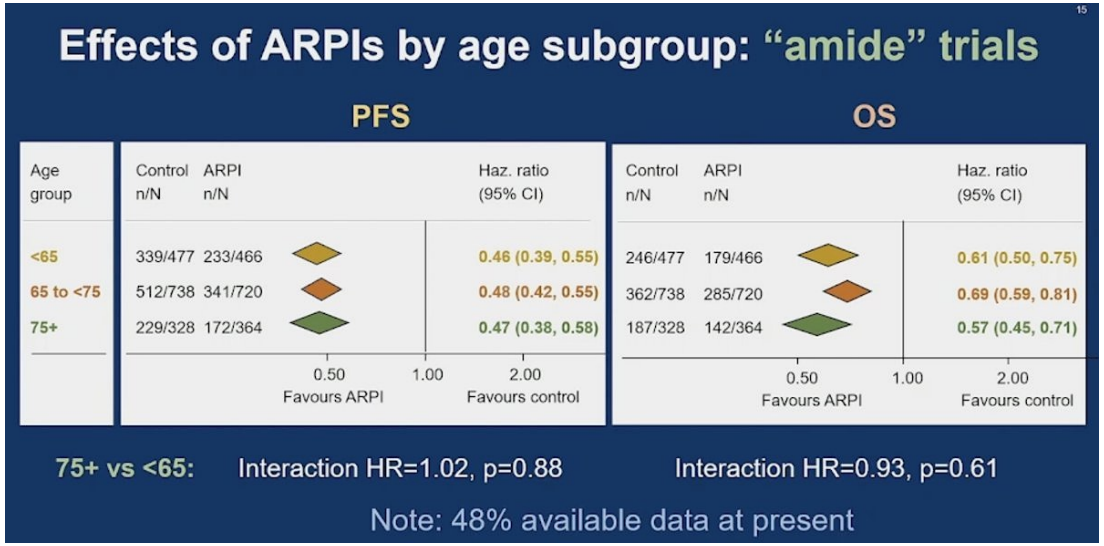
1. Saad F, et al. J Clin Oncol 2024; 2. Chi KN, et al. NEJM 2019; 3. Armstrong AJ, et al. J Clin Oncol 2019; 4. Fizazi K, et al. NEJM 2017; 5. Fizazi K, et al. Lancet Oncol 2019

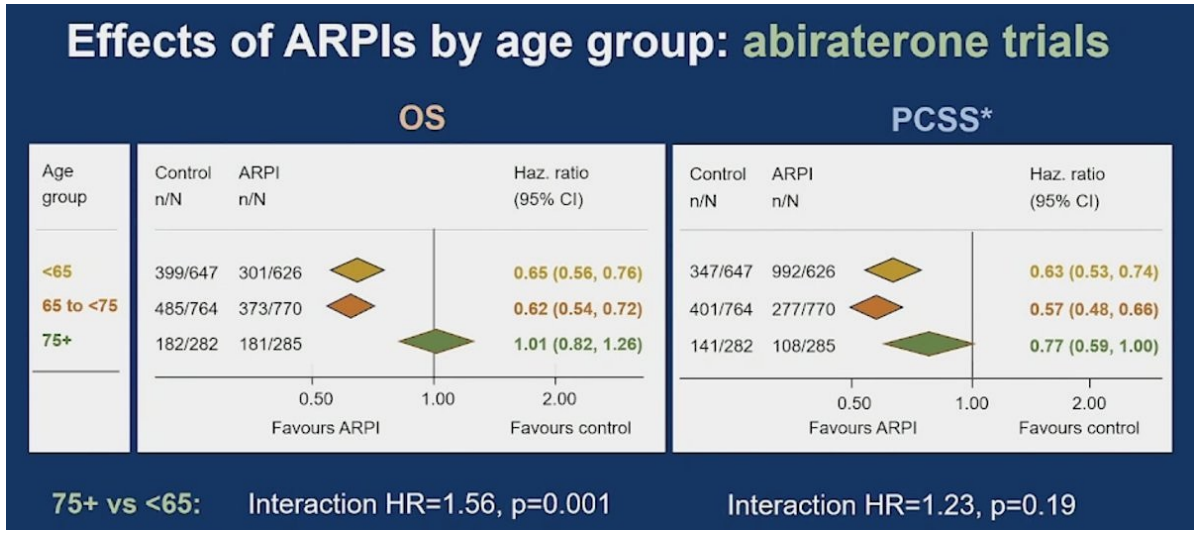
Choosing Between Androgen Receptor Pathway Inhibitors

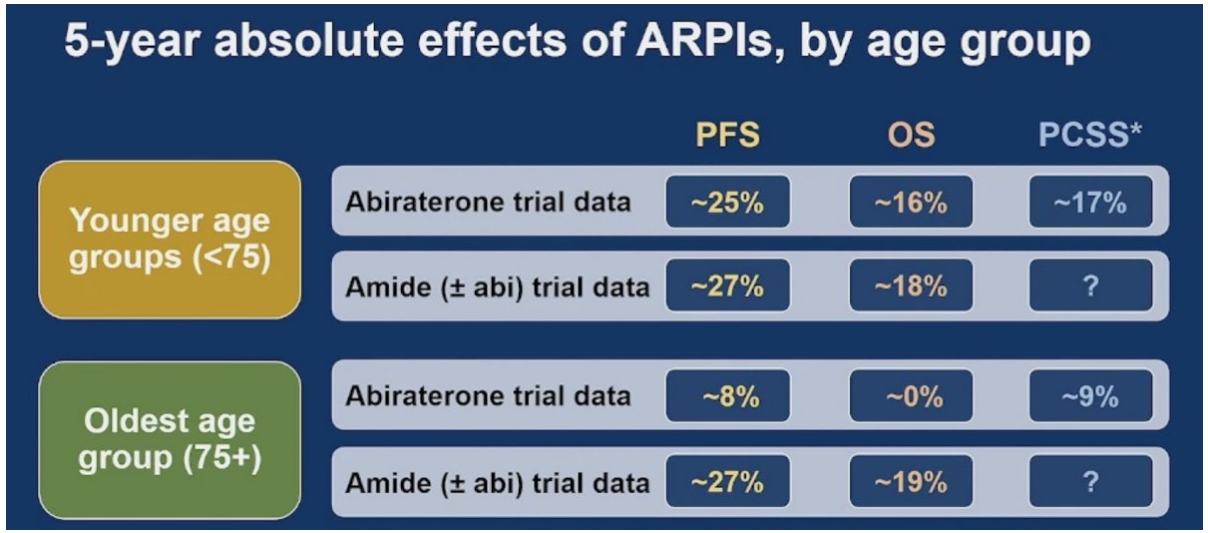


STOPCAP Meta-Analysis of mHSPC Phase 3 Trial Data

Trial	No. of participants	Follow-up (years)	Survival (years)
STAMPEDE (abi)	1,003	8.0	3.8
LATITUDE (abi)	1,199	4.3	3.0
PEACE-1 (abi)	1,172	6.0	4.6
ENZAMET (enz)	1,125	5.7	6.1
TITAN (apa)	1,052	3.7	4.4
STAMPEDE (abi + enza)	916	6.0	4.3
SWOG 1216 (ort)	1,311	6.9	6.3







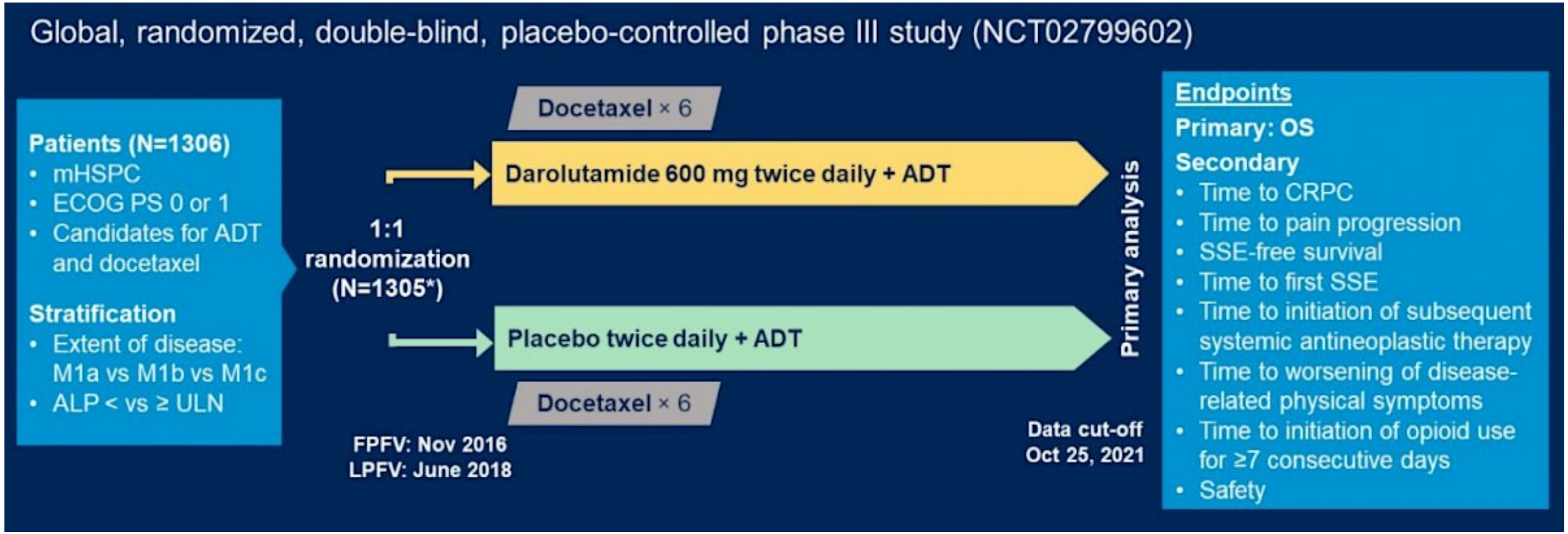
Fisher D, et al. ASCO GU 2025

NCCN Treatment Guidelines for mHSPC

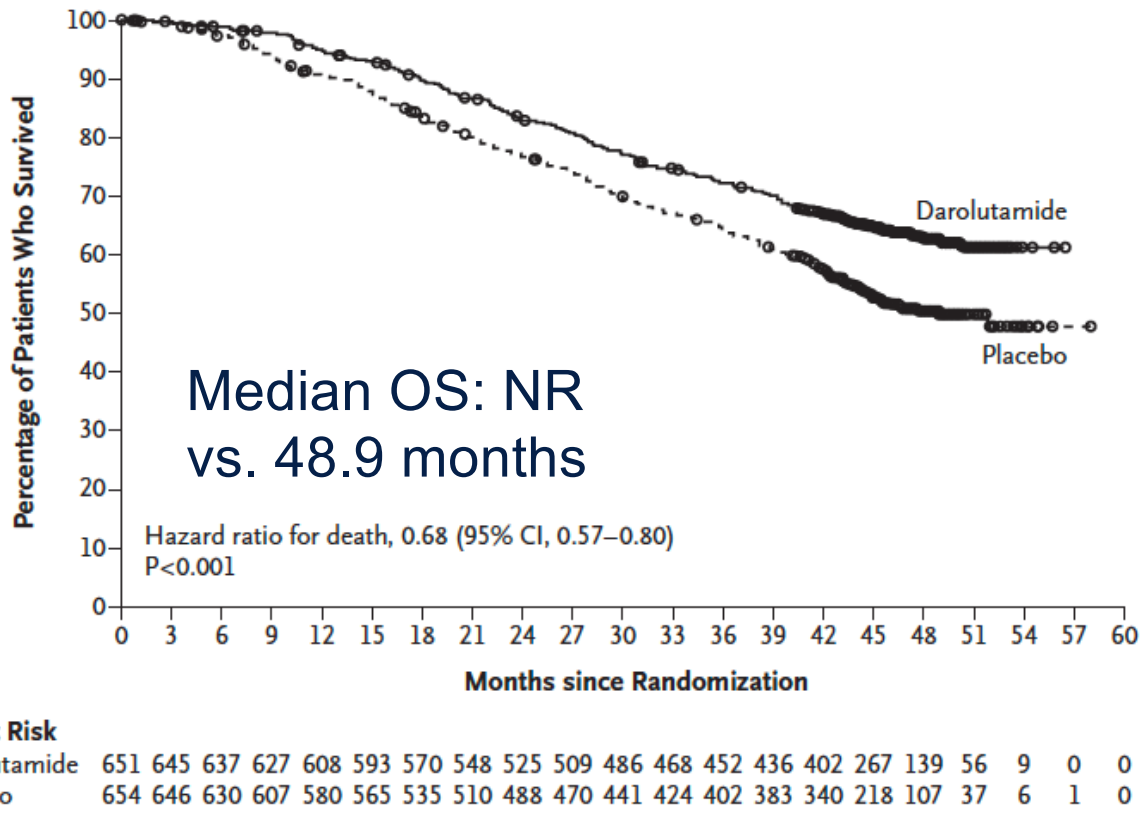
OLIGOMETASTATIC		LOW-VOLUME M1	HIGH-VOLUME M1[c]
Metachronous	Synchronous	Metachronous	Synchronous or Metachronous
MDT ± ADT (CAT 2A)	ADT[d] + 1 of the following: Preferred ▪ Abiraterone ▪ Apalutamide ▪ Enzalutamide Other recommended regimens ▪ Darolutamide (CAT 2B) *BRCA*2mut only ▪ Niraparib/abiraterone[e] (CAT 2B)	ADT[d] + 1 of the following: Preferred ▪ Abiraterone ▪ Apalutamide ▪ Enzalutamide Other recommended regimens ▪ Darolutamide (CAT 2B) *BRCA*2mut only ▪ Niraparib/abiraterone[e] (CAT 2B)	ADT + Docetaxel + 1 of the following: Preferred ▪ Abiraterone ▪ Darolutamide Other recommended regimens ▪ Apalutamide (CAT 2B) ▪ Enzalutamide (CAT 2B)
	ADT[d] + Docetaxel + 1 of the following (low-volume disease only): ▪ Abiraterone (CAT 2B) ▪ Apalutamide (CAT 2B) ▪ Darolutamide (CAT 2B) ▪ Enzalutamide (CAT 2B)		ADT + 1 of the following: Preferred ▪ Abiraterone ▪ Apalutamide ▪ Enzalutamide Other recommended regimens ▪ Darolutamide (CAT 2A) *BRCA*2mut only ▪ Niraparib/abiraterone[e] (CAT 2A)
	ADT[d] + EBRT to the primary tumor[f] alone or with 1 of the following: ▪ Abiraterone ▪ Apalutamide (CAT 2B) ▪ Docetaxel (CAT 2B) ▪ Enzalutamide (CAT 2B)		

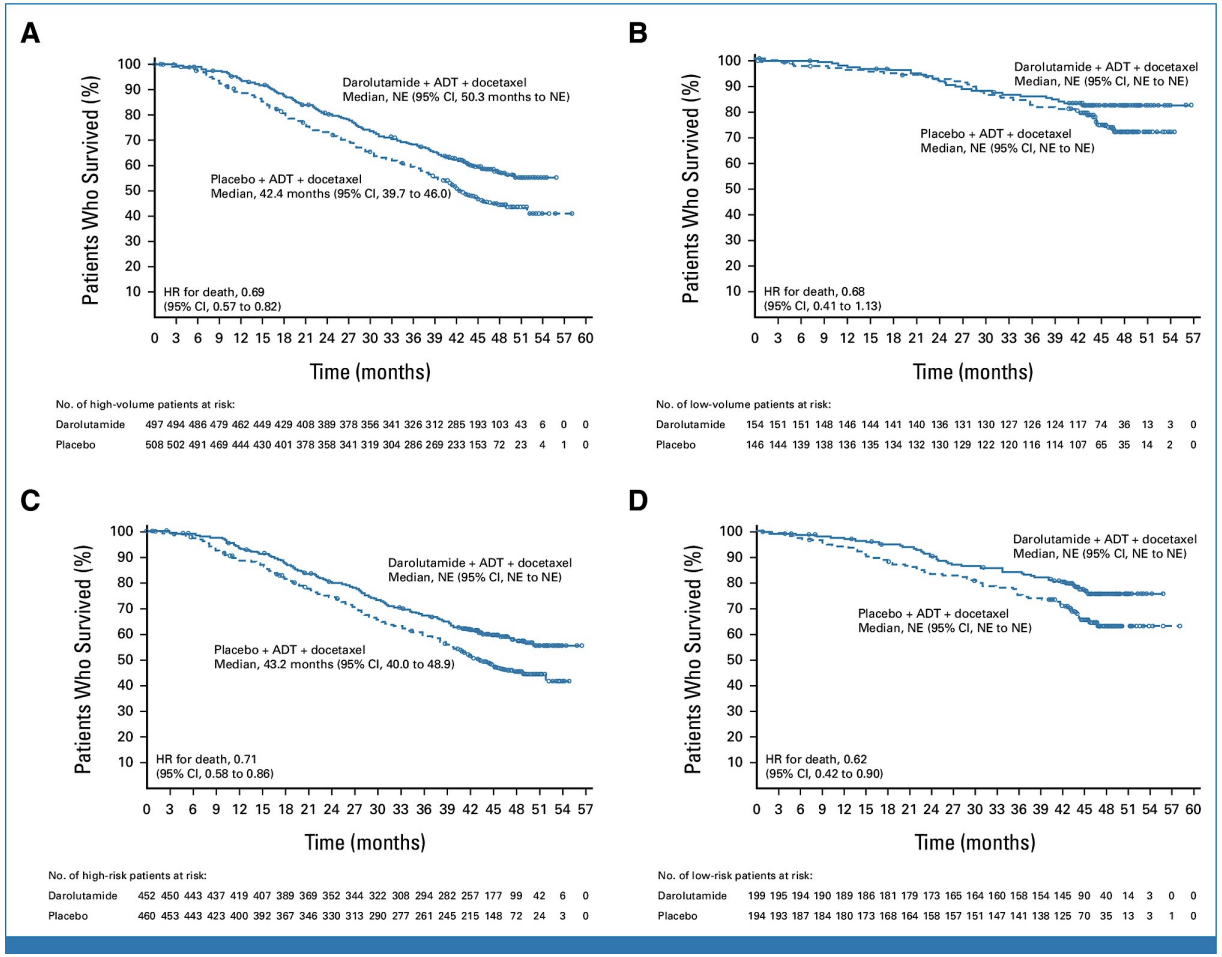
NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines®) for Prostate Cancer. V5.2026. Accessed March 3, 2026.

ARASENS Phase 3 Study



ARASENS Primary and Subgroup Results





Smith MR, et al. NEJM 2022

Hussain M, et al. J Clin Oncol 2023

ARASENS Safety Results

Event	Darolutamide–ADT–Docetaxel (N=652)†	Placebo–ADT–Docetaxel (N=650)†
	number of patients (percent)	
Any adverse event	649 (99.5)	643 (98.9)
Worst grade		
Grade 1	28 (4.3)	35 (5.4)
Grade 2	162 (24.8)	169 (26.0)
Grade 3	248 (38.0)	232 (35.7)
Grade 4	183 (28.1)	181 (27.8)
Grade 5	27 (4.1)	26 (4.0)
Serious adverse event	292 (44.8)	275 (42.3)
Adverse event leading to permanent discontinuation of trial agent		
Darolutamide or placebo	88 (13.5)	69 (10.6)
Docetaxel	52 (8.0)	67 (10.3)
Selected grade 3 or 4 adverse events‡		
Neutropenia§	220 (33.7)	222 (34.2)
Febrile neutropenia	51 (7.8)	48 (7.4)
Hypertension	42 (6.4)	21 (3.2)
Anemia	31 (4.8)	33 (5.1)
Pneumonia	21 (3.2)	20 (3.1)
Hyperglycemia	18 (2.8)	24 (3.7)
Increased ALT level	18 (2.8)	11 (1.7)
Increased AST level	17 (2.6)	7 (1.1)
Increased weight	14 (2.1)	8 (1.2)
Urinary tract infection	13 (2.0)	12 (1.8)

Smith MR, et al. NEJM 2022

PEACE-1

Key Eligibility Criteria

De novo mCSPC

Distant metastatic disease by ≥ 1 lesion on bone scan and/or CT scan

ECOG PS 0 -2

On-Study Requirement

Continuous ADT

Permitted

ADT ≤ 3 months

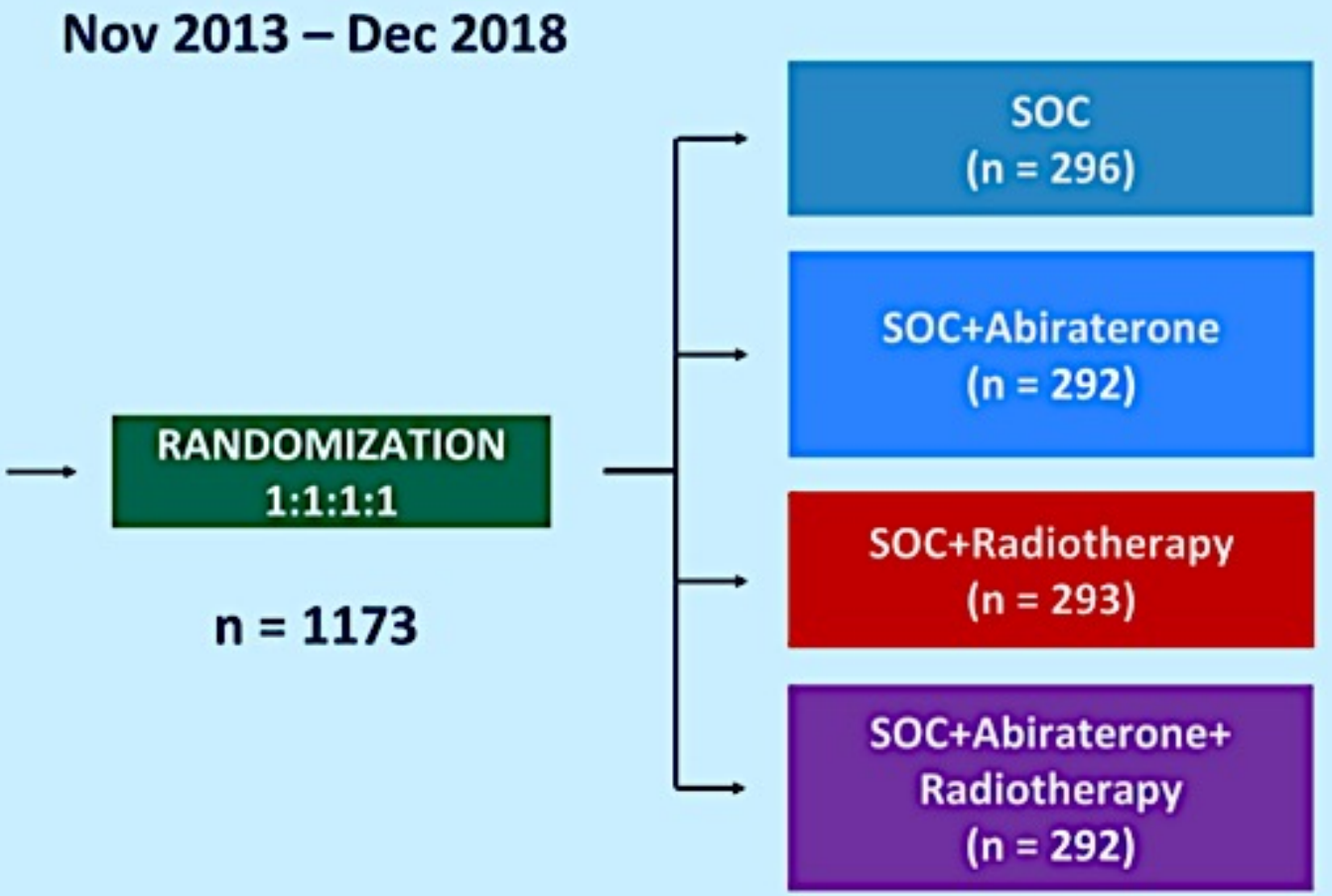
Stratification

ECOG PS (0 vs 1-2)

Metastatic sites (LN vs bone vs visceral)

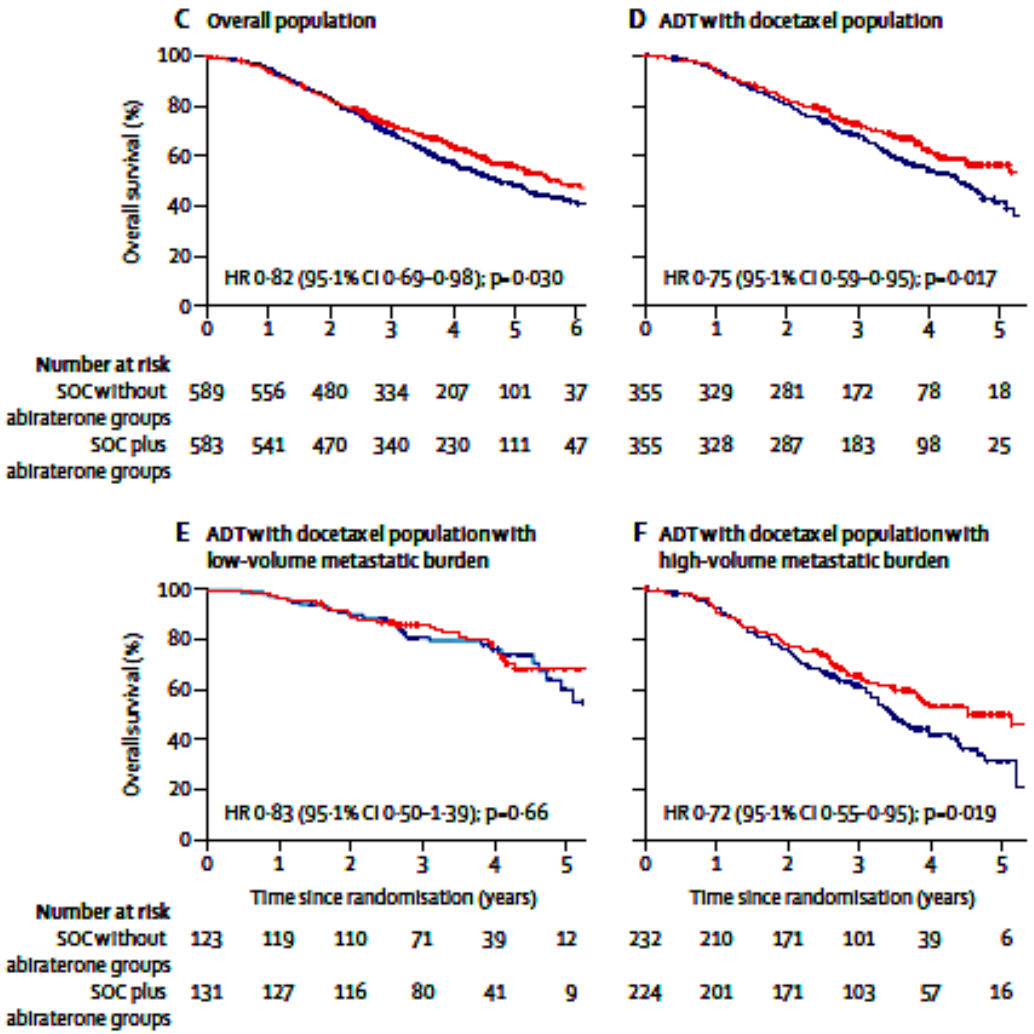
Type of castration (orchidectomy vs LHRH agonist vs LHRH antagonist)

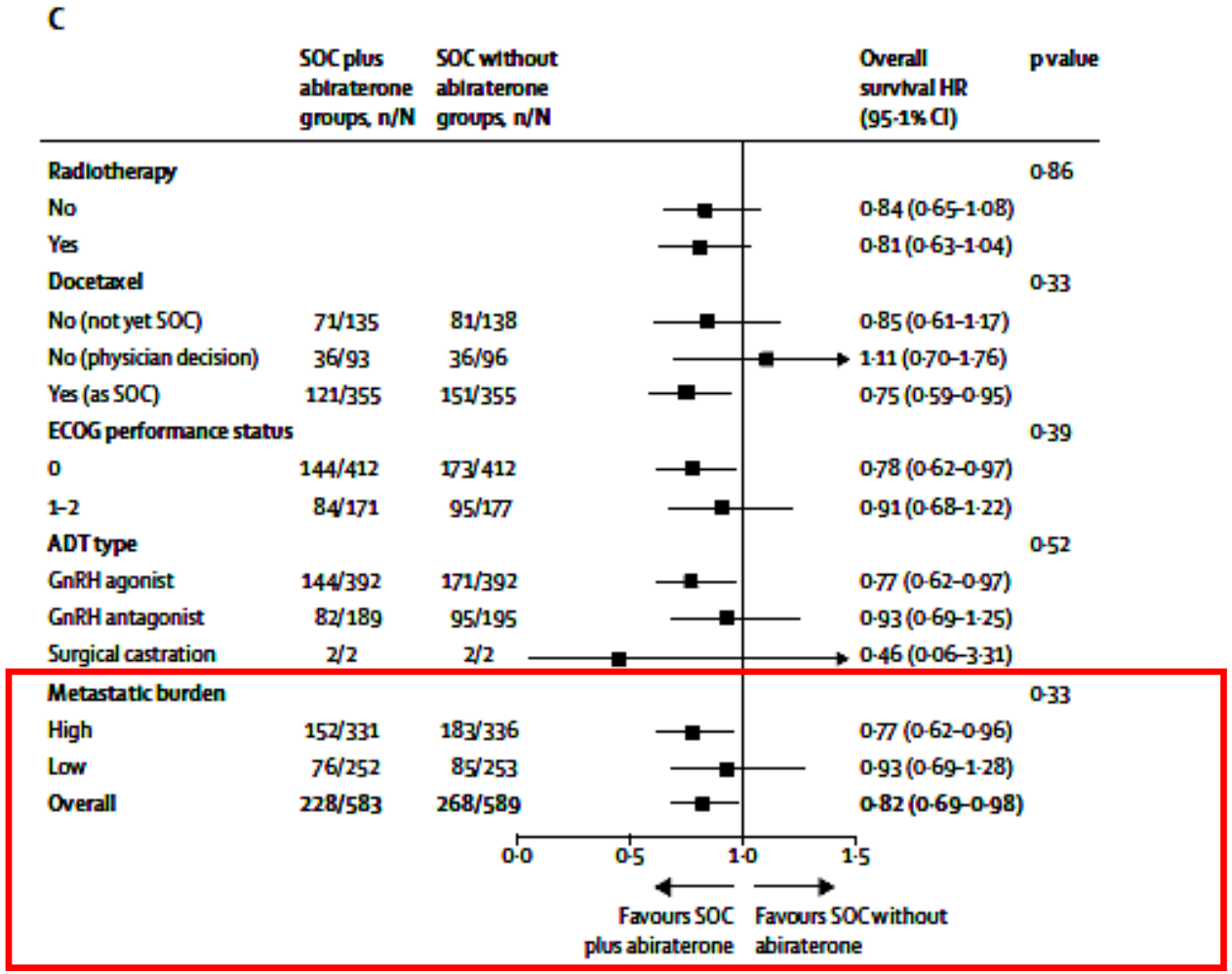
Docetaxel (yes vs no)



ECOG PS, Eastern Cooperative Oncology Group performance status

PEACE-1



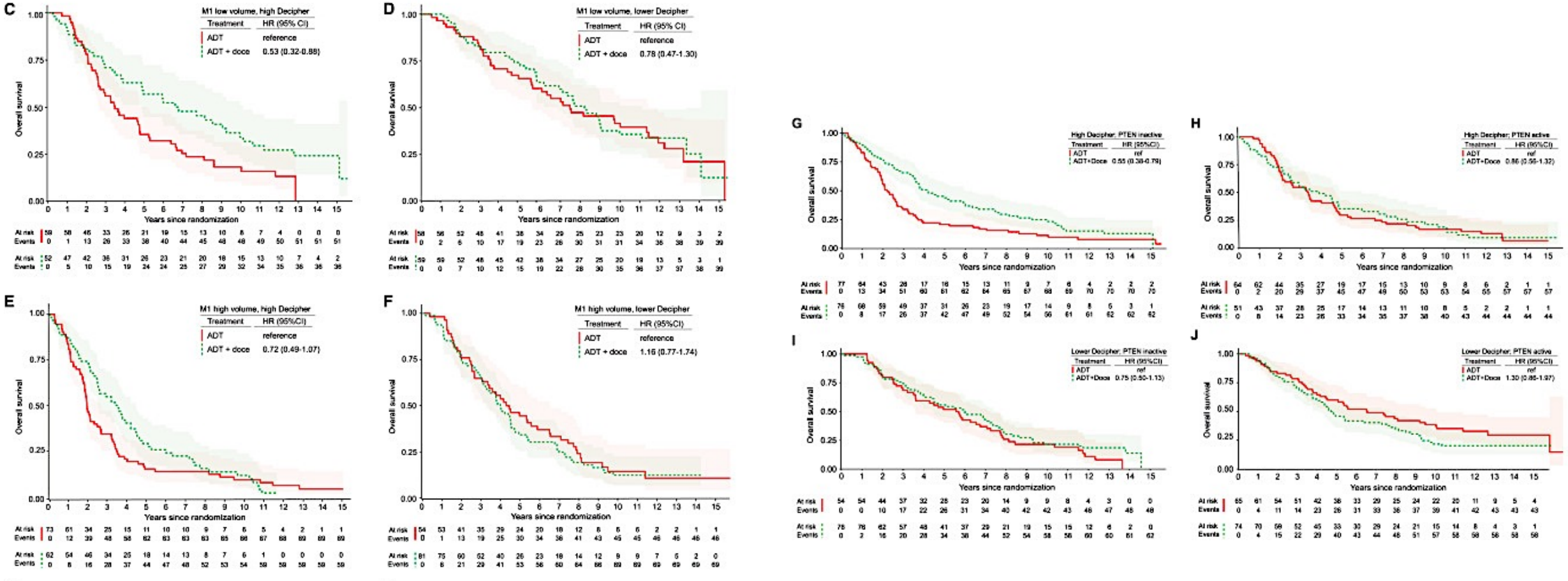


Fizazi K, et al. Lancet 2022

Who should receive ADT + ARPI + Docetaxel?

- Prospective phase 3 survival data ADT + ARSI +/- taxane not yet available (NCTN Phase 3 ASPIRE study)
- De novo high-volume fit patients ideal candidates for triplet therapy
  - Meta-analyses suggest potential benefit in high-volume patients
  - AR signaling inhibitor sensitivity can be heterogeneous in high grade tumors
  - Many patients don't ever receive taxane chemotherapy in the mCRPC setting
- Emerging predictive biomarkers but need prospective validation
  - *PTEN* status
  - Decipher score

PTEN Deficiency and Decipher score may predict benefit from docetaxel in mHSPC



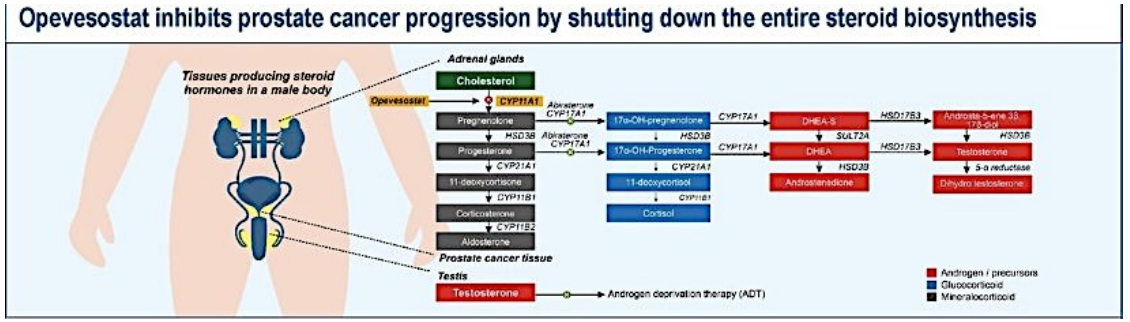
Grist E, et al. Cell 2025

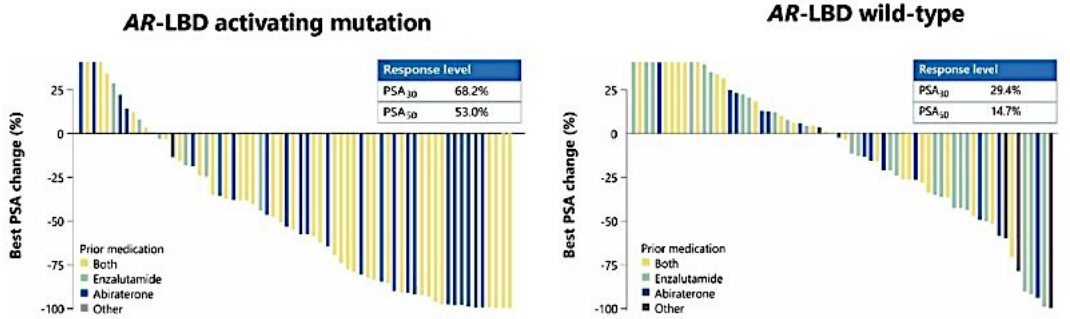
Novel Hormonal Approaches: CYP11 inhibition

PATIENT DEMOGRAPHIC CHARACTERISTICS:

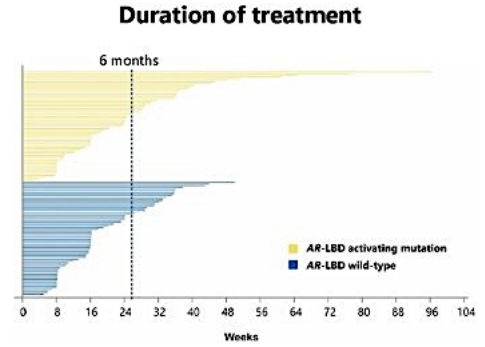
Variable		*AR*-LBD mut (n=66)	*AR*-LBD wt (n=68)
Baseline characteristics			
Age	Median	69.5	67.5
ECOG performance status	0	16 (24.2%)	25 (36.8%)
	1	49 (74.2%)	42 (61.8%)
	2	1 (1.5%)	1 (1.5%)
PSA (ug/L)	Median	262.5	49.9
Testosterone (ng/dL)	Median	3.1	4.1
Prior treatments			
Abiraterone		58 (87.9%)	41 (60.3%)
Enzalutamide		42 (63.6%)	48 (70.6%)
Both Abi and Enza		35 (53.0%)	25 (36.8%)
Docetaxel		65 (98.5%)	68 (100%)
Cabazitaxel		46 (69.7%)	44 (64.7%)
Both Doce and caba		46 (69.7%)	44 (64.7%)

Adverse events in CYPIDES Phase 2 (N=134)			
	n (%)		
Patients reporting an adverse event (all)	132 (98.5%)		
Grade ≥ 3 AE's	92 (68.7%)		
Most commonly reported SERIOUS adverse events (>1 event total)			
Preferred Term	**n (%)**	**Preferred Term**	**n (%)**
Adrenal insufficiency	7 (5.2)	Acute coronary syndrome	2 (1.5)
Prostate cancer	7 (5.2)	Anaemia	
Pulmonary embolism	6 (4.5)	Asthenia	
Dyspnoea	4 (3.0)	Chest pain	
Sepsis	4 (3.0)	Fall	
Back Pain	3 (2.2)	Haematuria	
COVID-19	3 (2.2)	Hyperkalaemia	
Confusional state	3 (2.2)	Hyponatraemia	
General physical health deterioration	3 (2.2)	Oxygen saturation decreased	
Pain	3 (2.2)	Pleural effusion	
		Pneumonia	
		Renal failure	
		Tumour pain	
		Urosepsis	





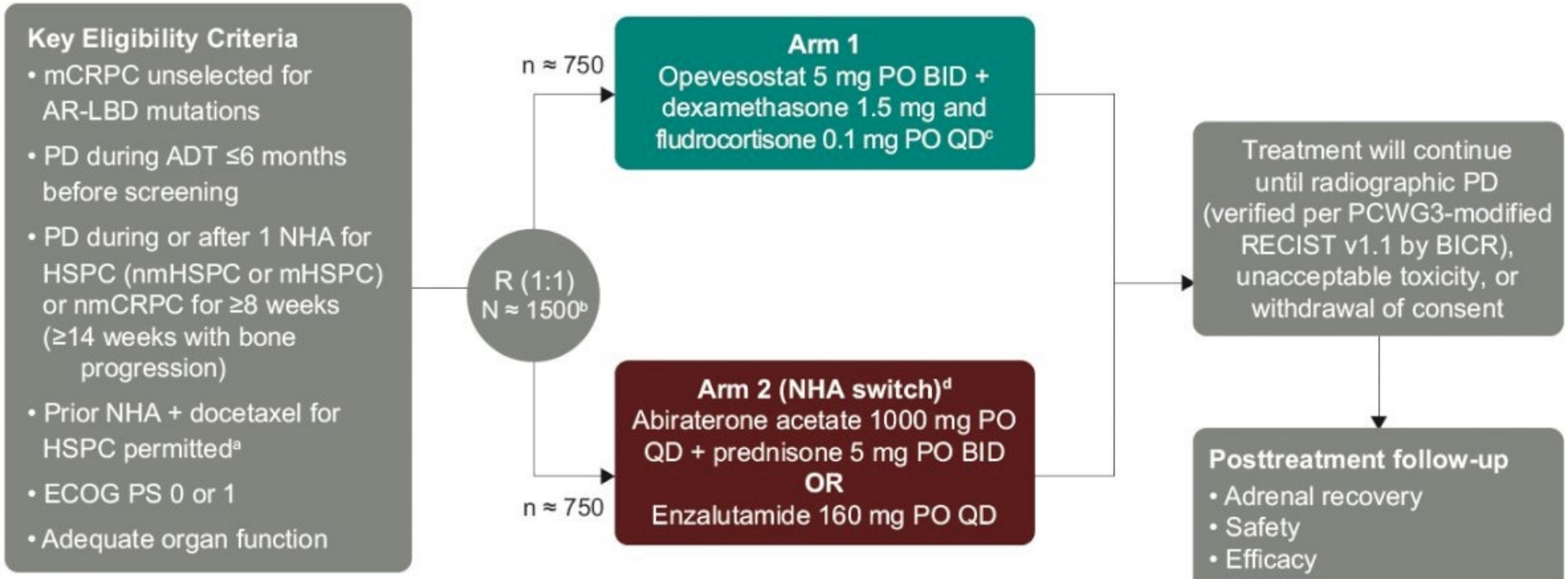
- At time of analysis, 2 *AR*-LBD mutated patients and 15 *AR*-LBDwt patients were ongoing on treatment.
- Durable responses have occurred in patients with and without *AR*-LBD mutation.



Fizazi K, et al. ESMO 2024

Novel Hormonal Approaches: CYP11 inhibition

Study design



Stratification

- Metastasis (bone only vs liver vs other)
- AR-LBD mutation status[e] (positive vs negative)
- Prior docetaxel for HSPC (yes vs no)

Novel Hormonal Approaches: EZH2 inhibition in Combination with ARPI

Methods: Study design

Patient population:
- mCRPC
- Prior abiraterone
- ≤1 regimen of prior chemotherapy in any setting
- Evidence of progression per modified PCWG3 criteria
- Ongoing ADT

R 1:1 N=81

- Mevrometostat 1250 mg orally BID empty stomach + Enzalutamide 160 mg orally QD n=41
- Enzalutamide 160 mg orally QD n=40

Primary endpoints:
- rPFS per investigator assessment
- Safety

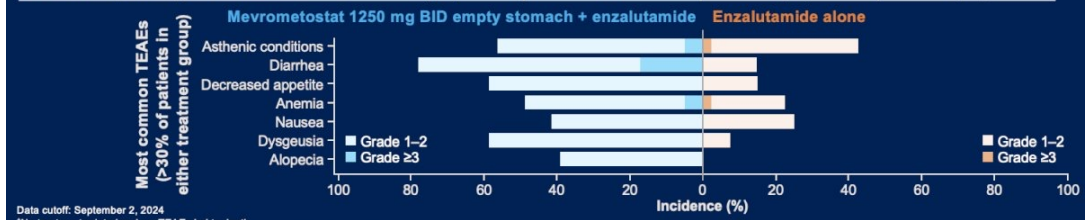
Secondary endpoints:
- OR†
- PSA_{50}
- Pharmacokinetics‡

Stratification factor:
- Prior chemotherapy

Safety summary

Mevrometostat 1250 mg BID empty stomach + enzalutamide has a manageable safety profile

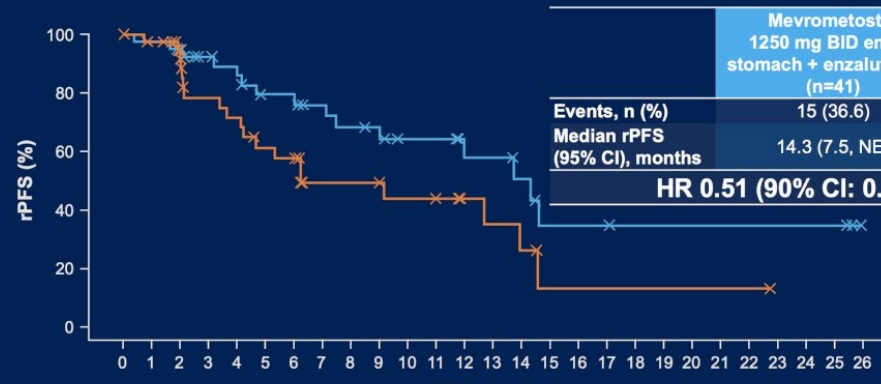
Event, n (%)	Mevrometostat 1250 mg BID empty stomach + enzalutamide (n=41)		Enzalutamide alone (n=40)	
	All grades	Grade ≥3	All grades	Grade ≥3
Any TEAE	40 (97.6)	22 (53.7)	37 (92.5)	17 (42.5)
Treatment-related TEAE	39 (95.1)	20 (48.8)	33 (82.5)	9 (22.5)
Serious AE	14 (34.1)	13 (31.7)	11 (27.5)	10 (25.0)
Treatment-related serious TEAE†	10 (24.4)	10 (24.4)	1 (2.5)	1 (2.5)
TEAE leading to dose reduction	15 (36.6)	7 (17.1)	3 (7.5)	0
TEAE leading to study discontinuation	1 (2.4)	0	2 (5.0)	1 (2.5)



Data cutoff: September 2, 2024
†No treatment-related serious TEAEs led to death
AE, adverse event; BID, twice daily; TEAE, treatment-emergent adverse event

Primary endpoint: rPFS by investigator

49% reduction in the risk of progression or death and ~8-month improvement in median rPFS



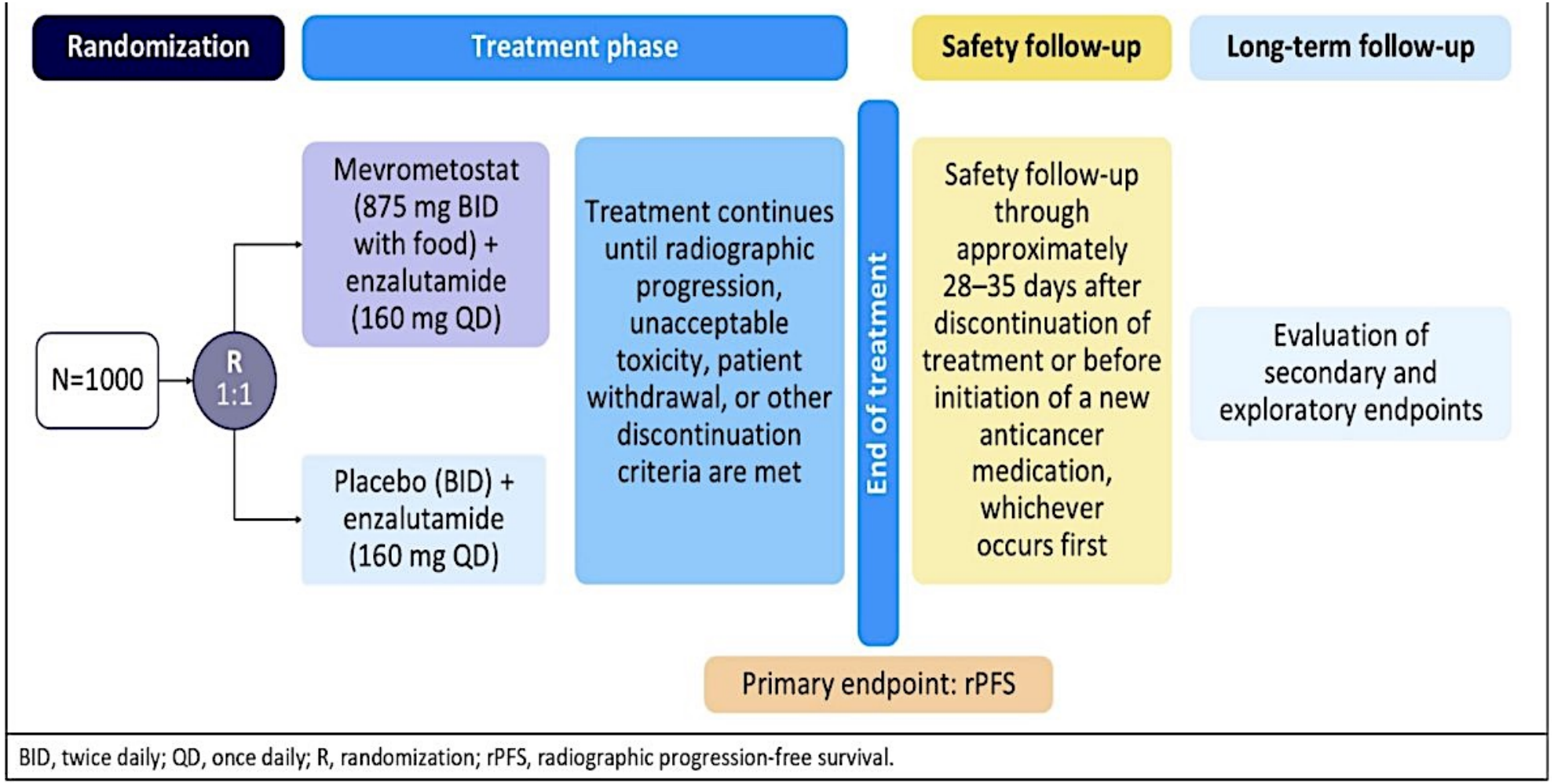
	Mevrometostat 1250 mg BID empty stomach + enzalutamide (n=41)	Enzalutamide alone (n=40)
Events, n (%)	15 (36.6)	19 (47.5)
Median rPFS (95% CI), months	14.3 (7.5, NE)	6.2 (4.1, 13.9)

HR 0.51 (90% CI: 0.28, 0.95)

Patients at risk																									
Mevrometostat 1250 mg BID empty stomach	41	39	36	30	28	23	23	20	18	17	14	14	9	9	7	4	4	4	3	3	3	3	3	3	3	0
Enzalutamide alone	40	37	31	23	21	17	16	10	10	10	8	7	5	4	3	1	1	1	1	1	1	1	0	0	0	0

Schweizer M, et al. ASCO GU 2025

Novel Hormonal Approaches: EZH2 inhibition in Combination with ARPI



BID, twice daily; QD, once daily; R, randomization; rPFS, radiographic progression-free survival.

Summary

- Unequivocal benefit of ARPIs in mHSPC
  - At least a doublet should be offered to nearly every patient regardless of disease volume or if de novo versus recurrent disease

- Triplet therapy has a role in higher risk mHSPC
  - Potential biomarkers pending prospective validation

- CYP11 inhibition and combination of ARPIs with epigenetic regulators including EZH2 inhibition may further improve outcomes in mHSPC pending results of ongoing and planned clinical trials

UCSF Helen Diller Family Comprehensive Cancer Center

Discussion Questions

61 yo man, presented with PSA over 4,000, extensive bony metastases. Treated with ADT + docetaxel + abiraterone. His PSA dropped to a nadir of 15.

When mHSPC patients achieve max response with PSA reaching nadir, do you consider consolidation radiation to residual disease or the prostate?

What do you consider the appropriate biomarker workup for a patient with hormone-sensitive metastatic disease?

How do you determine whether to add docetaxel to an ARPI/ADT for these patients? In which situations, if any, do you start with an ARPI/ADT and add in docetaxel later if suboptimal response?

RTP
RESEARCH
TO PRACTICE

Discussion Questions

67-year-old man: Progressive urinary frequency, nocturia, intermittent pelvic discomfort and fatigue. PSA level of 92 ng/mL, elevated alkaline phosphatase and mild anemia.
Imaging: Pelvic lymphadenopathy and two sclerotic bone lesions involving the pelvis and a thoracic vertebra.
Gleason 4 + 4 = 8 (Grade Group 4). Low volume (cT3aN1M1b).

Doublet vs triplet therapy?

Which ARPI would be preferred — abiraterone, enzalutamide, apalutamide or darolutamide?

I saw some real-world data suggesting that apalutamide may be superior to other agents. Any thoughts?

RTP
RESEARCH
TO PRACTICE

Discussion Questions (Continued)

67-year-old man: Progressive urinary frequency, nocturia, intermittent pelvic discomfort and fatigue. PSA level of 92 ng/mL, elevated alkaline phosphatase and mild anemia.
Imaging: Pelvic lymphadenopathy and two sclerotic bone lesions involving the pelvis and a thoracic vertebra.
Gleason 4 + 4 = 8 (Grade Group 4). Low volume (cT3aN1M1b).

Do you approach the patient differently if de novo mHSPC vs recurrent?

Which is your preferred type of ADT in this situation?

Would you offer a treatment break after good response? How would you execute it?

RTP
RESEARCH
TO PRACTICE

Discussion Questions

Patient is a 56-year-old with metastatic hormone-sensitive prostate cancer with transfusion-dependent anemia due to MDS. Currently on ADT and ARPI. NGS is unremarkable.

What would be the recommended second-line therapy?

RTP
RESEARCH
TO PRACTICE

Agenda

Module 1: Evolving Management of Nonmetastatic Hormone-Sensitive Prostate Cancer (HSPC) — Dr Antonarakis

Module 2: Current and Future Hormonal Treatment for Metastatic HSPC (mHSPC) — Dr Aggarwal

Module 3: Current and Future Role of Regimens Combining PARP Inhibitors and AR Pathway Inhibitors in Metastatic Prostate Cancer — Dr Abida

Module 4: Emerging Role of AKT Inhibition for Patients with mHSPC — Dr Fizazi

Module 5: Current and Future Use of Radiopharmaceuticals in Metastatic Prostate Cancer — Dr McKay

RTP
RESEARCH
TO PRACTICE

Memorial Sloan Kettering Cancer Center

Current and Future Role of Regimens Combining PARP Inhibitors and AR Pathway Inhibitors in mPC

Research To Practice
ASCO Prostate Cancer Symposium

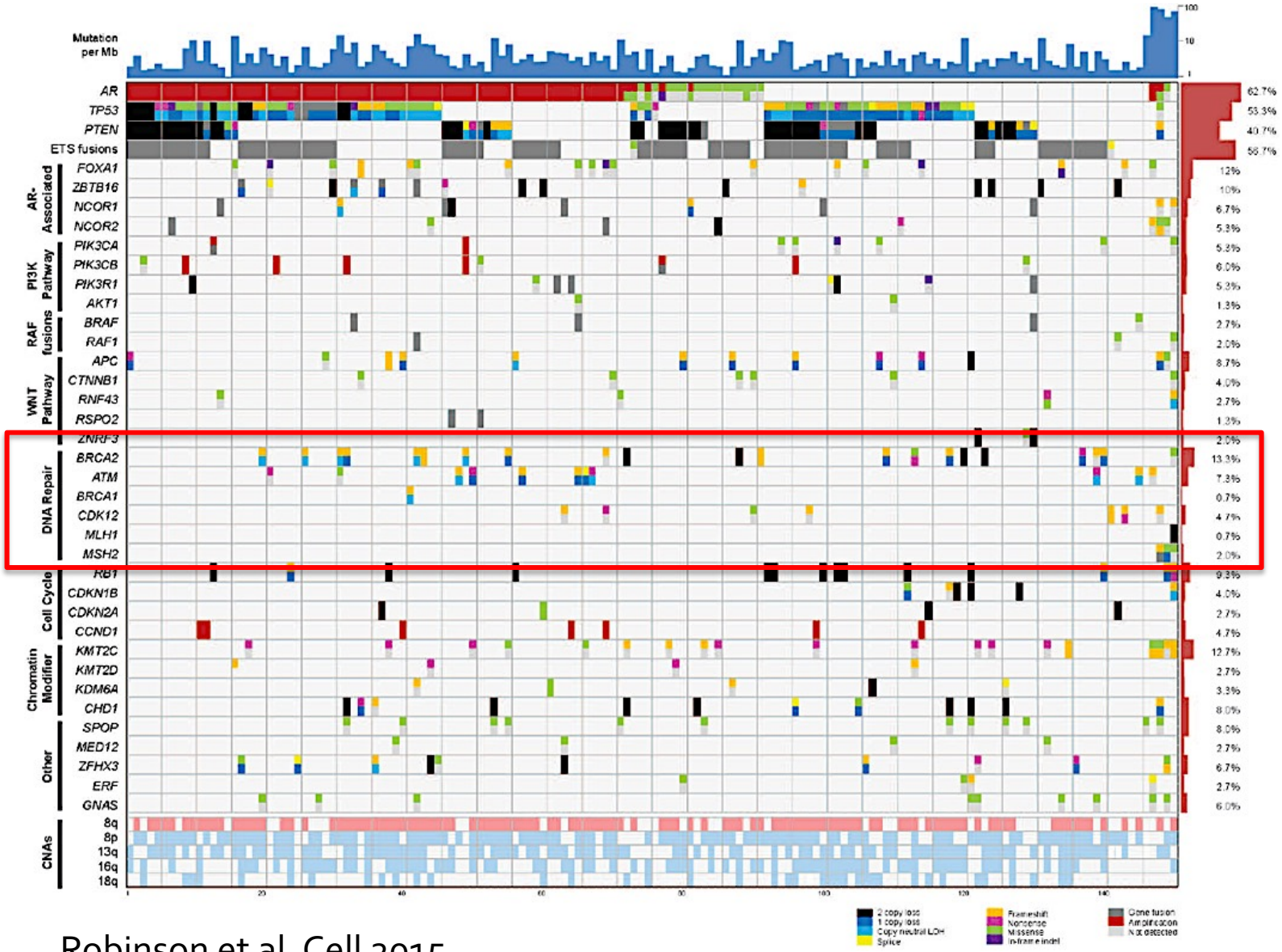
Wassim Abida MD, PhD

Director of Translational Research, Prostate Cancer
Associate Member
Genitourinary Oncology Service
Memorial Sloan Kettering Cancer Center
Associate Professor of Medicine, Weill Cornell Medical College

Discussion points

- Incidence of HRR gene alterations in metastatic prostate cancer
- Rationale for PARP inhibitor use in prostate cancer:
  - HRR-deficient prostate cancer
  - Combination with ARPI
- Key studies of PARP inhibitors in mCRPC (APMR metastatic prostate cancer)
  - PROFOUND and TRITON
  - PROPEL, MAGNITUDE and TALAPRO2 (1st line mCRPC)
- Key studies of PARP inhibitors in mCSPC (APMS metastatic prostate cancer): AMPLITUDE, TALAPRO3 and EVOPAR-01
- Current/future directions for PARP inhibitor use in prostate cancer

DNA damage repair (DDR) gene alterations in prostate cancer



Robinson et al. Cell 2015

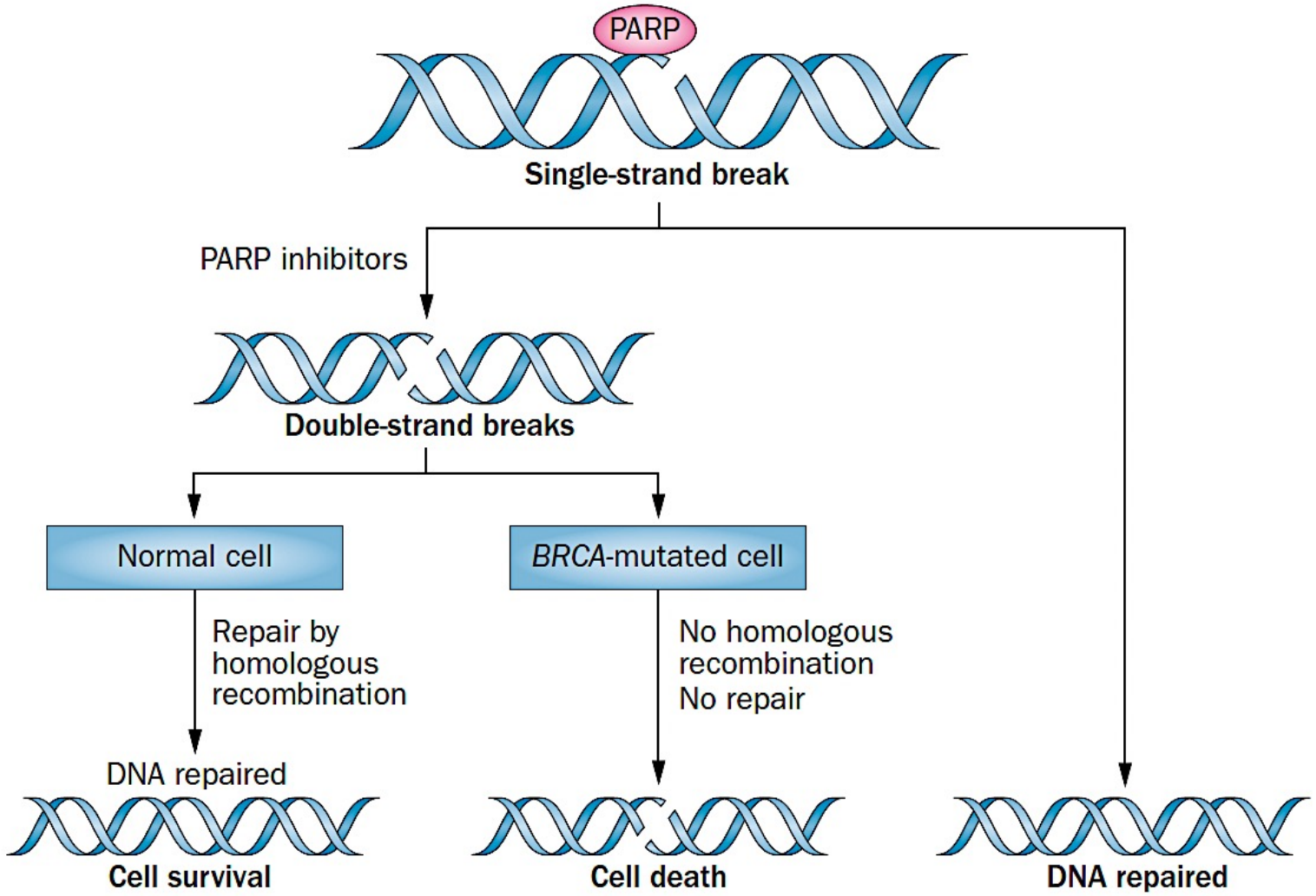
DNA damage repair gene alterations observed in ~20% of patients with mCRPC

HRR (homologous recombination repair) genes: BRCA2, BRCA1, PALB2, RAD51 homologs

Other DDR genes: ATM, CDK12, CHEK2

Memorial Sloan Kettering Cancer Center

PARP inhibitor mechanism of action



Sonnenblick et al, Nature Reviews Clinical Oncology 2015

Memorial Sloan Kettering Cancer Center

TRITON2 showed benefit of rucaparib in BRCA-mutated mCRPC

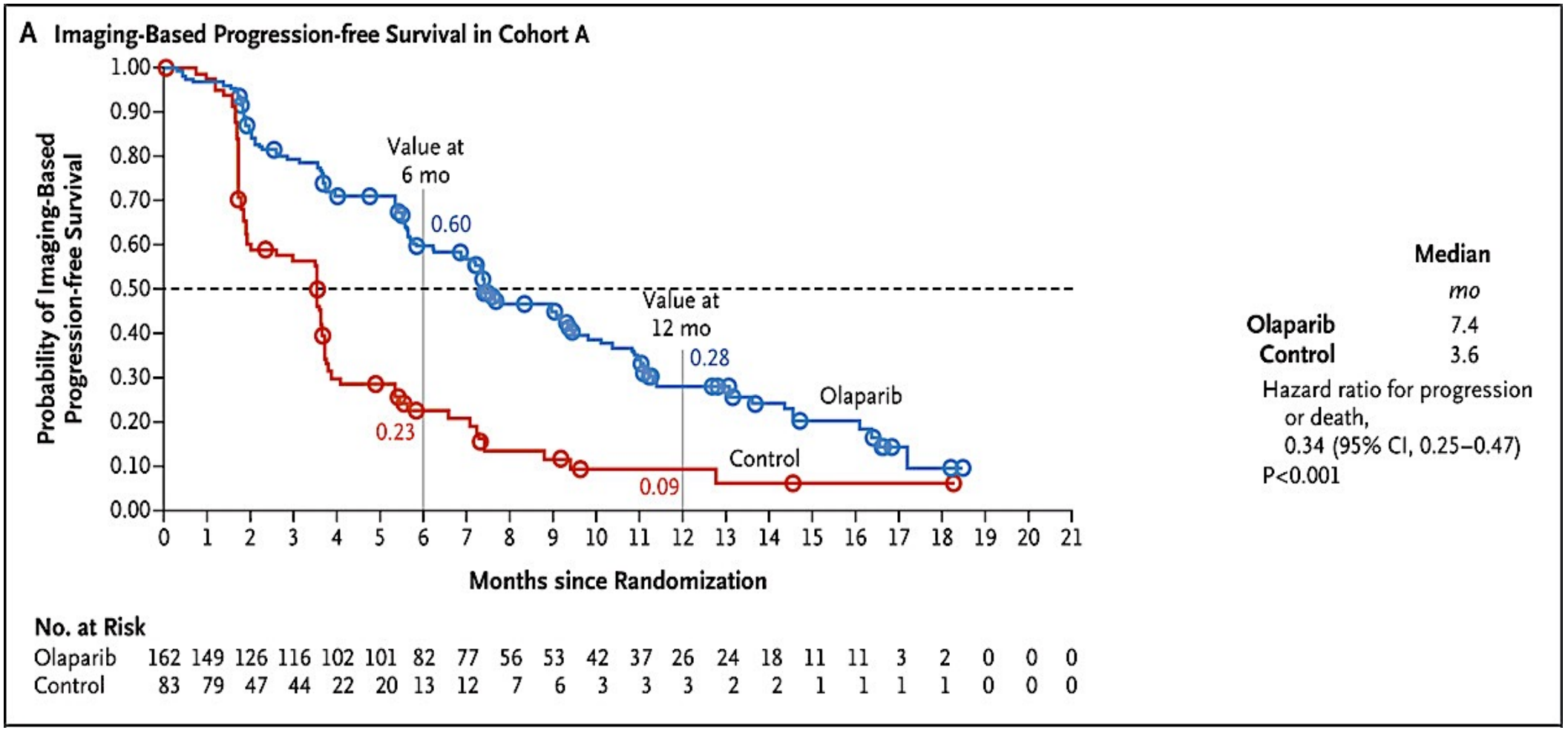
Response	Investigator-Evaluable Population (n = 65)	IRR-Evaluable Population (n = 62)
Confirmed ORR, No. (%; 95% CI)[a]	33 (50.8; 38.1 to 63.4)	27 (43.5; 31.0 to 56.7)
Complete response	4 (6.2)	7 (11.3)
Partial response	29 (44.6)	20 (32.3)
Stable disease	25 (38.5)	28 (45.2)
Progressive disease	6 (9.2)	6 (9.7)
Not evaluable	1 (1.5)	1 (1.6)

	Overall Efficacy Population (n = 115)
Confirmed PSA response rate, No. (5; 95% CI)	63 (54.8; 45.2 to 64.1)

Abida. J Clin Oncol. 2020;38:3763.

Memorial Sloan Kettering Cancer Center

PROFOUND showed benefit of olaparib in HRR-mutated mCRPC



De Bono. NEJM. 2020;382:2091.

Memorial Sloan Kettering Cancer Center

Rationale for PARP inhibitor + ARPI combinations

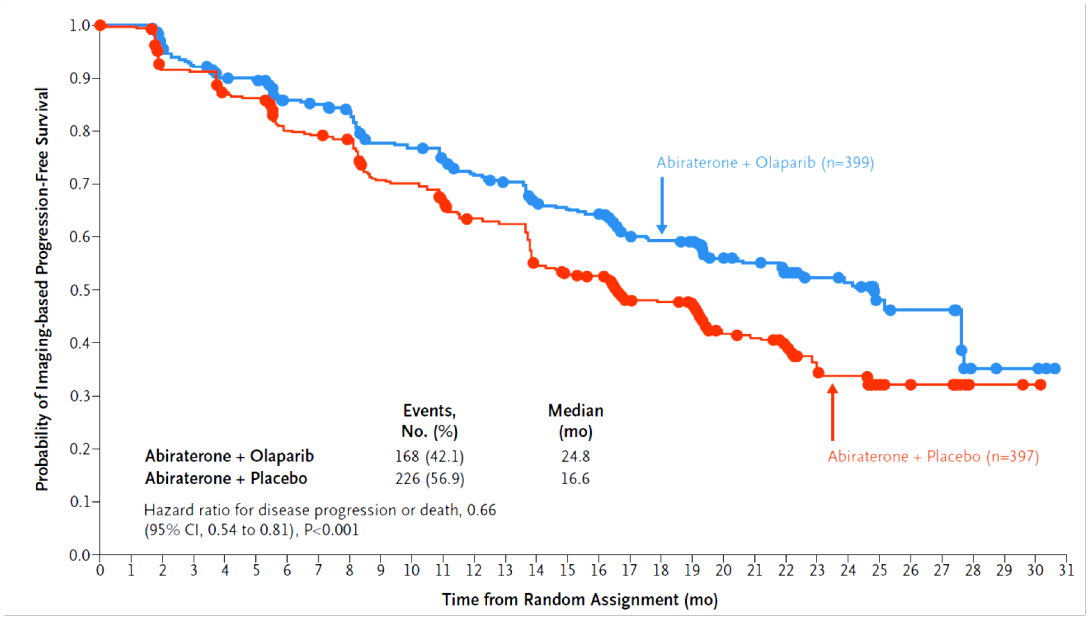
- There is likely to be at least an additive effect of PARP inhibitors and ARPIs where both agents are individually active (HRR-mutated prostate cancer)

- There may be synergistic effect of PARP inhibition and ARPI in non-HRR deficient contexts:
  - AR inhibition modulates expression of HRR genes in prostate cancer cell lines
  - The combination of ARPI and PARPi has synergistic effect in some 'HRR proficient' prostate cancer models

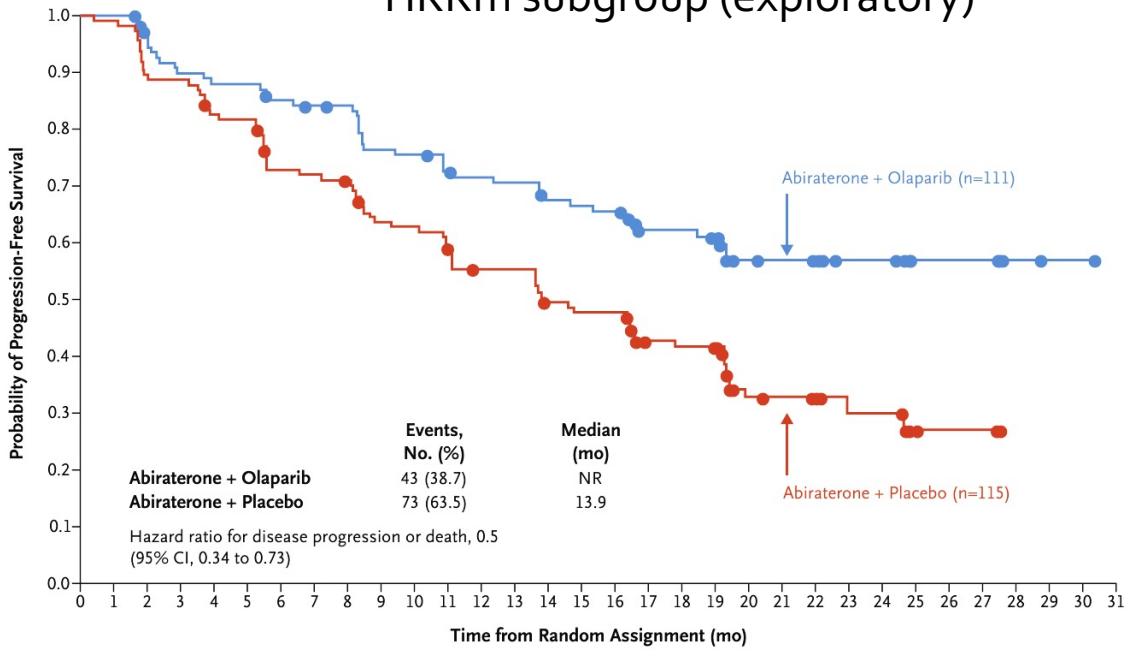
PROPEL: olaparib + abiraterone in 1st line mCRPC

- Phase 3 study, 1st line mCRPC: randomizing abiraterone alone vs abiraterone + olaparib
- Prior ARPI / docetaxel allowed for CSPC, not required (~1/4 had prior doce, nearly 0 had prior ARPI)
- Unselected study, exploratory DNA testing via plasma/tumor NGS for HRR mutation status
- Primary endpoint: rPFS

Overall population



HRRm subgroup (exploratory)



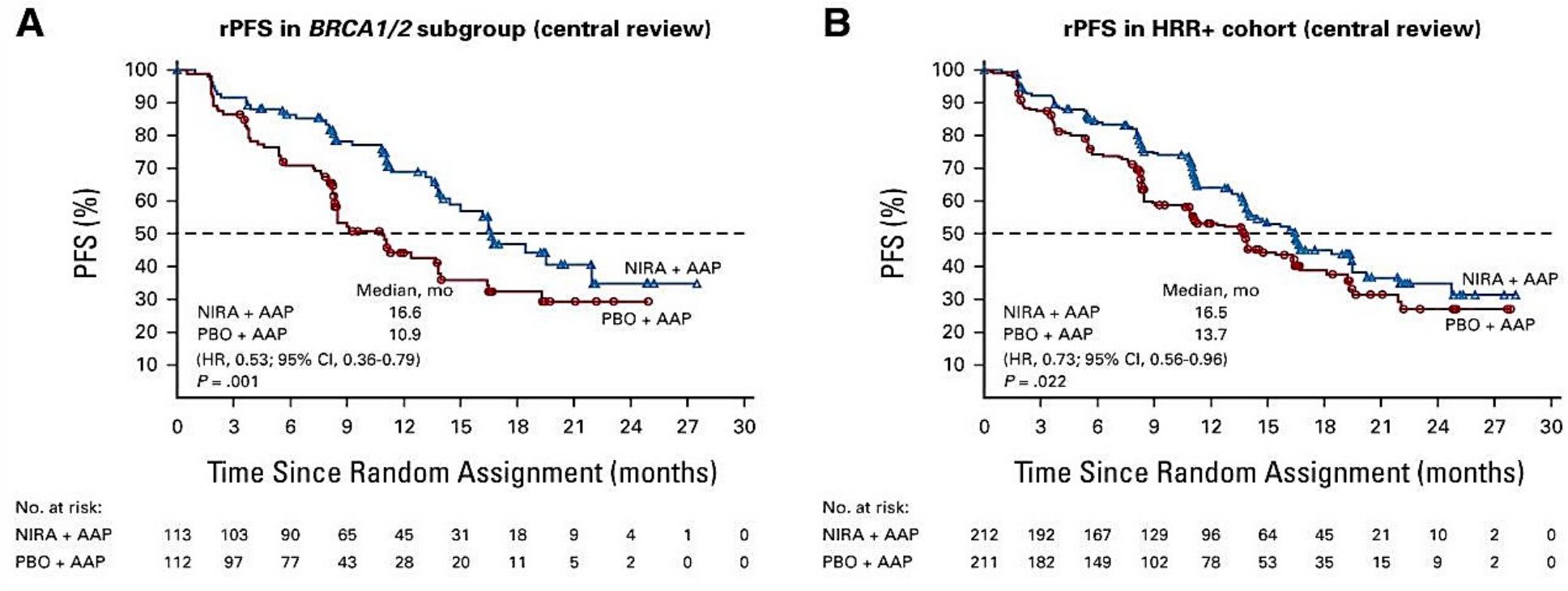
Clarke et al NEJM 2022

FDA approved combination for BRCA2/1 mutated first line mCRPC

Memorial Sloan Kettering Cancer Center

MAGNITUDE: niraparib + abiraterone in 1st line mCRPC

- Phase 3 study, 1st line mCRPC: randomizing abiraterone alone vs abiraterone + niraparib
- Prior ARPI / docetaxel allowed, not required (~1/5 had prior doce, ~3% had prior ARPI)
- 2 cohorts prospectively stratified by HRR status determined via NGS
- Primary endpoint: rPFS



Chi et al, JCO 2023

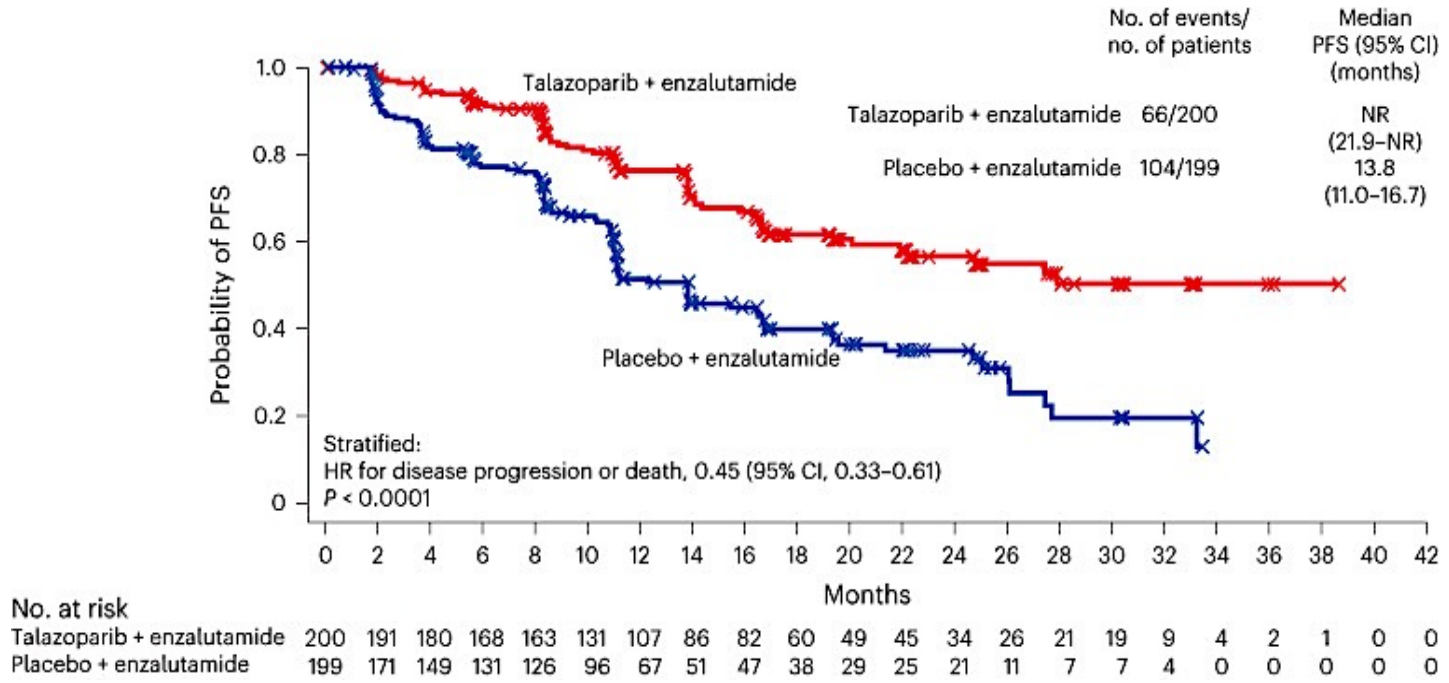
FDA approved combination for BRCA2/1 mutated first line mCRPC

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TALAPRO2: talazoparib + enzalutamide in 1st line mCRPC

- Phase 3 study, 1st line mCRPC: randomizing enzalutamide alone vs enzalutamide + talazoparib
- Prior ARPI / docetaxel allowed, not required (~1/4 had prior doce, ~5% had prior ARPI)
- Unselected study, prospective stratification for HRR status via NGS
- Primary endpoint: rPFS

rPFS in patients with any HRR gene alteration



Agarwal et al, Lancet 2023; Fizazi et al, Nature Medicine 2024

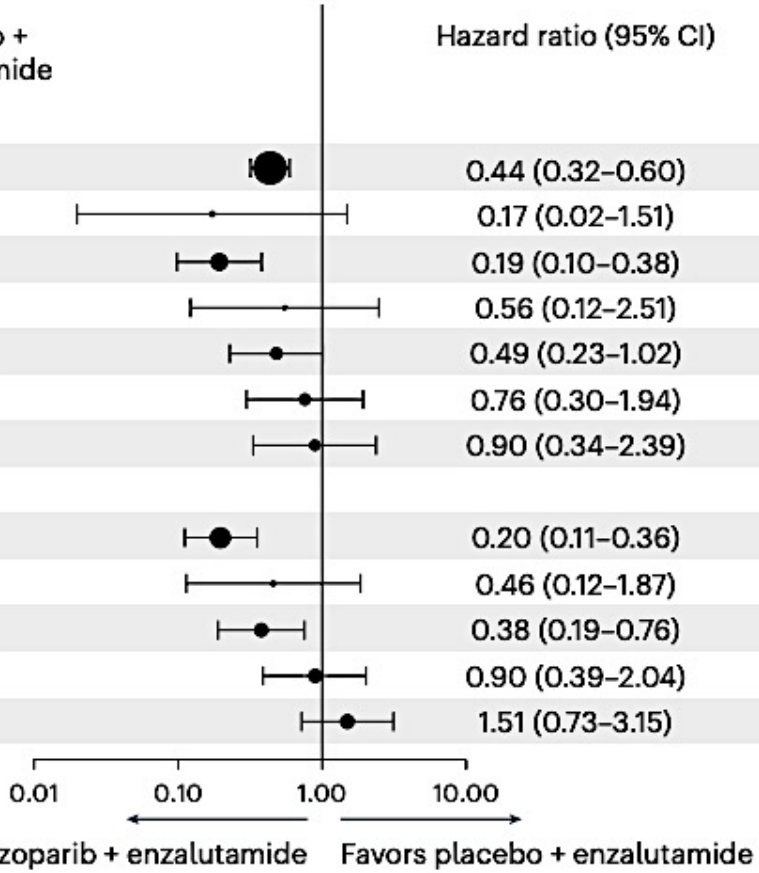
FDA approved combination for HRR gene- mutated first line mCRPC

Memorial Sloan Kettering Cancer Center

TALAPRO2: talazoparib + enzalutamide in 1st line mCRPC

Subgroup	Talazoparib + enzalutamide	Placebo + enzalutamide	Talazoparib + enzalutamide	Placebo + enzalutamide	Hazard ratio (95% CI)	Two-sided *P* value
	No. of events/no. of patients		Median (months)			
All HRR-deficient	65/198	104/197	NR	13.8	0.44 (0.32–0.60)	<0.0001
Only *BRCA1*	2/8	5/9	20.0	11.7	0.17 (0.02–1.51)	0.07
Only *BRCA2*	11/55	40/60	NR	11.0	0.19 (0.10–0.38)	<0.0001
Only *PALB2*	3/6	4/5	NR	8.6	0.56 (0.12–2.51)	0.44
Only *CDK12*	12/28	18/30	21.9	13.8	0.49 (0.23–1.02)	0.05
Only *ATM*	12/35	7/22	NR	27.7	0.76 (0.30–1.94)	0.58
Only *CHEK2*	8/24	8/24	22.1	NR	0.90 (0.34–2.39)	0.83
BRCA cluster	15/71	54/84	NR	11.0	0.20 (0.11–0.36)	<0.0001
PALB2 cluster	3/7	6/8	NR	8.3	0.46 (0.12–1.87)	0.27
CDK12 cluster	13/35	23/36	21.9	13.8	0.38 (0.19–0.76)	0.004
ATM cluster	16/43	9/29	27.9	27.7	0.90 (0.39–2.04)	0.80
Other gene cluster	18/42	12/40	22.1	NR	1.51 (0.73–3.15)	0.26

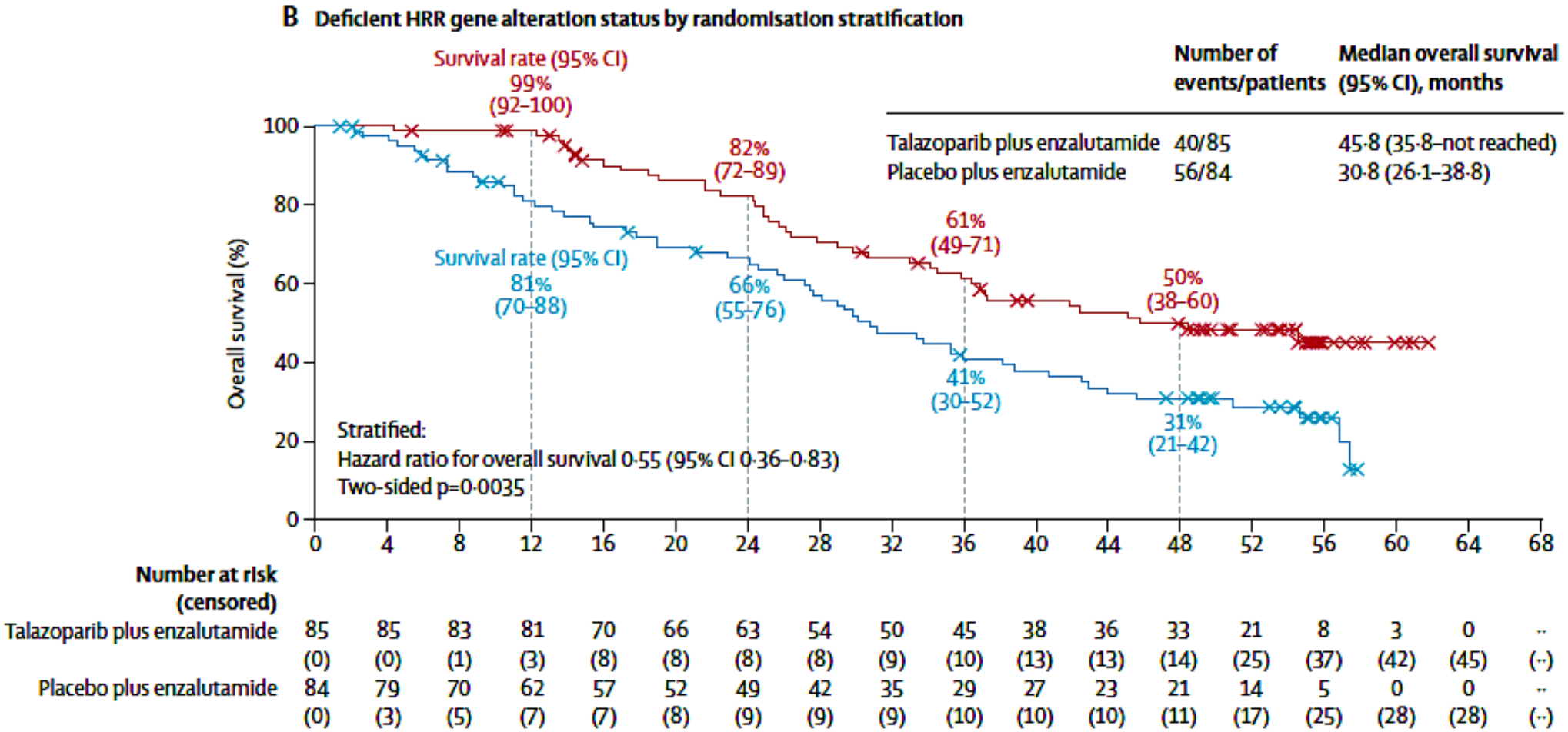
Hierarchy



Fizazi et al, Nature Medicine 2024

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TALAPRO2: talazoparib + enzalutamide in 1st line mCRPC



Agarwal et al, Lancet 2025

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Summary: PARPi monotherapy or combination in mCRPC

- The combination of PARPi and ARPI is appropriate for patients with HRR-mutant first-line mCRPC
  - Abiraterone/prednisone + olaparib (BRCA2/1-mutated first line mCRPC) (PROPEL)
  - Abiraterone/prednisone + niraparib (BRCA2/1-mutated first-line mCRPC) (MAGNITUDE)
  - Enzalutamide + talazoparib (HRR-mutated first-line mCRPC) TALAPRO2
  - Benefit for the combination is unclear in ARPI-treated patients
  - Few patients are ARPI-naïve when they develop castration resistance
- PARPi monotherapy remains the NCCN Category 1 / preferred option for mCRPC post-ARPI
  - Olaparib (HRR-mutated mCRPC post ARPI) (PROFOUND)
  - Rucaparib (BRCA2/1-mutated mCRPC post-ARPI) (TRITON2/3)

- Benefit is greatest in patients with mutations in *BRCA2 and BRCA-related genes (e.g. PALB2)*
  - No clear benefit for patients with ATM mutations although Olaparib and enza+talazoparib have approval in that context
  - Possible PFS benefit for enza+talazoparib in CDK12-mutant mCRPC

Toxicity of PARPi combinations

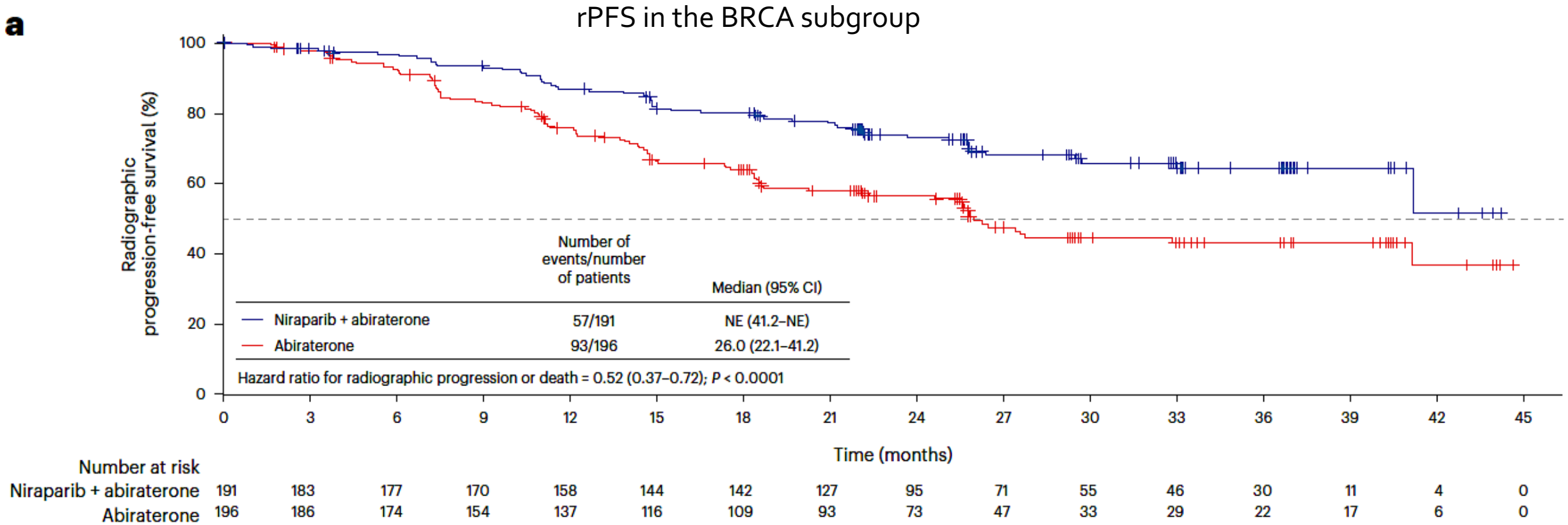
TEAE, %		PROpel: Olaparib + AAP (n = 398)		MAGNITUDE: Niraparib + AAP (n = 212)		TALAPRO-2: Talazoparib + Enzalutamide (n = 398)	
		All Grades	Grade ≥3	All Grades	Grade ≥3	All Grades	Grade ≥3
Hematologic	• Anemia • Thrombocytopenia • Neutropenia	49.7 6.8 10.1	16.1 0.8 5.0	52.4 24.1 16.0	30.7 8.5 6.6	68 26 38	49 7 19
Cardiovascular	• Hypertension • Arrythmia • Cardiac Failure	15.3 - 3.8	3.8 - 1.3	34.0 13.2 2.8	16.5 3.3 1.9	15 - -	6 - -
General	Fatigue	38.7	2.5	26.4	3.3	35	4
Gastrointestinal	• Constipation • Nausea • Diarrhea	18.6 30.7 41.5	0 0.3 1.3	30.7 23.6 -	-- 0.5 -	20 21 16	<1 <1 <1
Hepatotoxicity		3.8-4.0*	0.5*	11.8	1.9	-	-
Cerebrovascular	DVT/PE	2.5/7.3	0.5/2.3	-/1.9	-	1.5/3	-/2

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Saad F et al. Lancet 2023; Chi KN et al. Eur Urol Oncol 2025; Fizazi N et al. ASCO GU 2025;Abstract LBA18.

AMPLITUDE: niraparib+abiraterone in mCSPC

- Phase 3 study, newly diagnosed mCSPC: randomizing abiraterone alone vs abiraterone + niraparib
- Enrolled HRR-mutated mCSPC only
- Primary endpoint: rPFS. Secondary endpoint OS



FDA approved combination for BRCA2-mutated newly diagnosed mCSPC

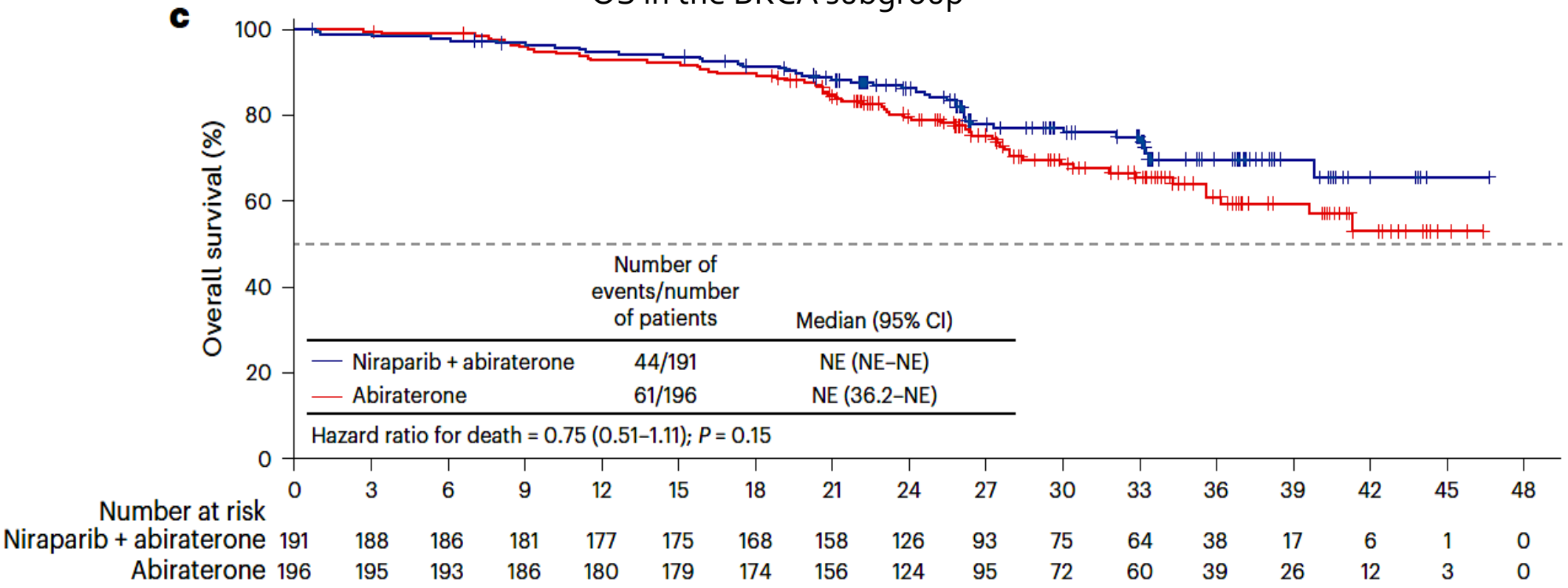
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Attard G et al. Nat Med. 2025

AMPLITUDE: niraparib+abiraterone in mCSPC

- Phase 3 study, newly diagnosed mCSPC: randomizing abiraterone alone vs abiraterone + niraparib
- Enrolled HRR-mutated mCSPC only
- Primary endpoint: rPFS. Secondary endpoint OS

OS in the BRCA subgroup



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Attard G et al. Nat Med. 2025

TALAPRO-3: talazoparib + enzalutamide in mCSPC

- Phase 3 study, newly diagnosed mCSPC: randomizing enzalutamide alone vs enzalutamide + talazoparib
- Enrolled HRR-mutated mCSPC only
- Primary endpoint: rPFS. Secondary endpoint OS

Talazoparib Plus Enzalutamide Significantly Improves Radiographic Progression-Free Survival in Metastatic Prostate Cancer

Thursday, March 19, 2026 - 06:45am | 17 min read

- *Primary endpoint met in Phase 3 TALAPRO-3 study demonstrating **a statistically significant and clinically meaningful reduction in risk of disease progression or death** in HRR gene-mutated metastatic hormone sensitive prostate cancer*
- *Consistent rPFS efficacy benefit was observed in patients whose tumors harbored BRCA and non-BRCA HRR gene alterations*
- *Interim analysis showed a strong trend toward improvement in overall survival*
- *These results will be discussed with global health authorities to potentially expand Talazoparib indication in this earlier stage disease*

PRESENTED TODAY IN ORAL ABSTRACT SESSION (LBA5007)

TALA + ENZA significantly improved rPFS vs PBO + ENZA (HR, 0.481; 95% CI, 0.357–0.647; 2-sided *P*<0.0001; median rPFS not reached vs 45.8 mo). The HR for rPFS was 0.368 (95% CI, 0.222–0.609) in the *BRCA*-mutated subgroup(n=207; 34.6%) and 0.567 (95% CI, 0.392–0.819) in the non-*BRCA*-mutated subgroup (n=392; 65.4%).Interim analysis of OS favored TALA + ENZA but has not reached statistical significance (74 deaths TALA +ENZA, 91 deaths PBO + ENZA; HR, 0.767; 95% CI, 0.564–1.044)

Aggarwal et al, ASCO Annual Meeting 2026

Memorial Sloan Kettering Cancer Center

Summary: PARPi in metastatic prostate cancer

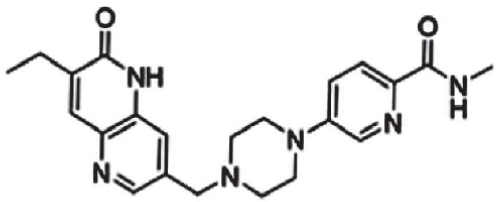
- PARP inhibitors are beneficial for patients with metastatic prostate cancer and selected HRR gene mutations:
  - Niraparib with abiraterone/prednisone is approved for patients with newly diagnosed mCSPC and a BRCA2 mutation
  - Several PARP inhibitors are approved either as monotherapy or in combination with ARPIs for men with HRR-mutated mCRPC
    - Choice of therapy depends on prior ARPI exposure, specific gene mutation (if not BRCA2/1), and patient's co-morbidities/drug interactions

- *BRCA2* alterations confer the highest benefit. Benefit is unclear for other HRR or DDR gene mutations

- Early genomic testing is key to identify patients who may benefit

Saruparib (AZD5305): PARP1-Selective Inhibition

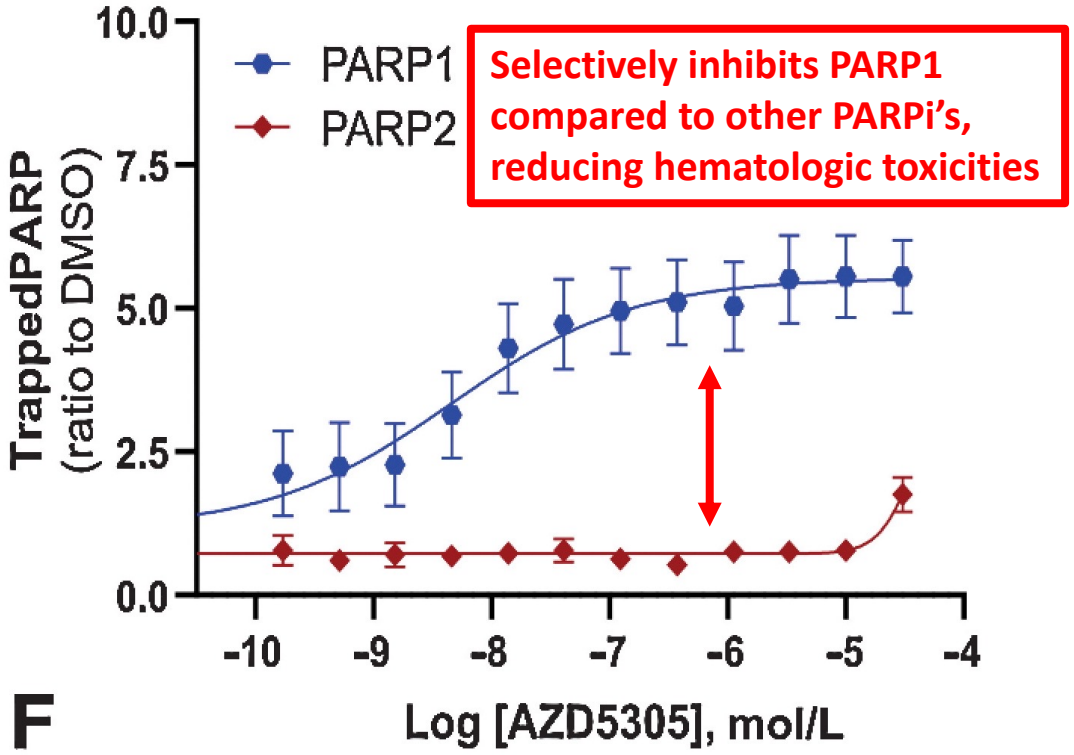
AZD5305



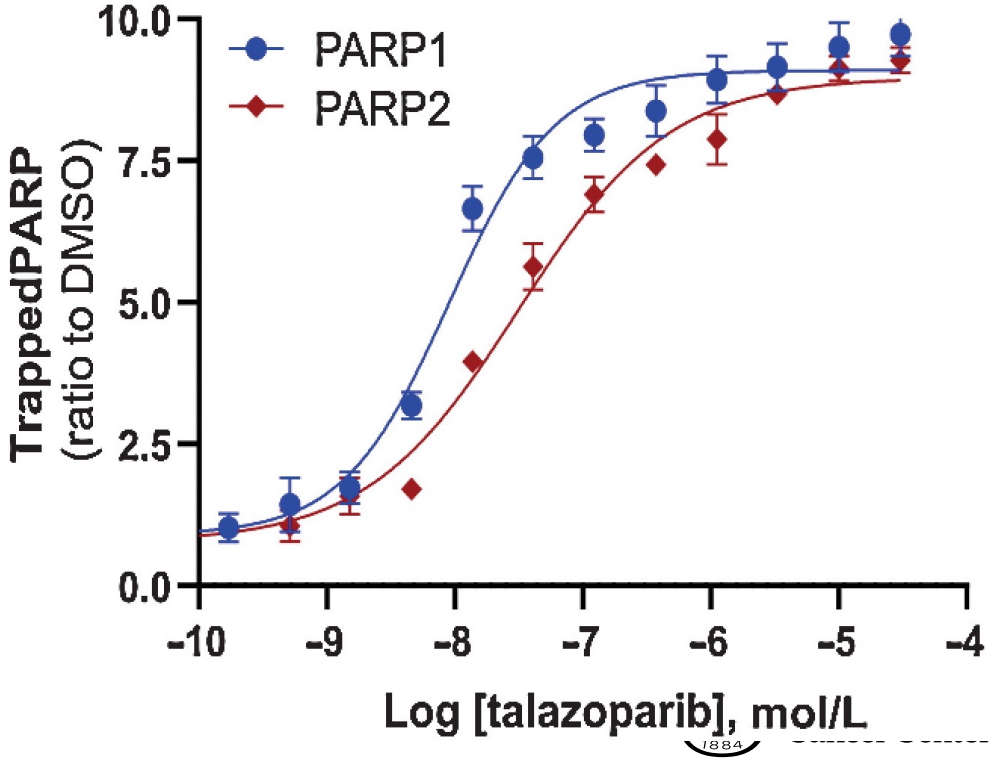
AZD5305	Biochemical assay*	Cellular assay
PARP1 IC_{50} (nmol/L)	3	1.5
PARP2 IC_{50} (nmol/L)	1,400	653
PARP1/2 fold selectivity	460	435

*Johannes et al., 2021





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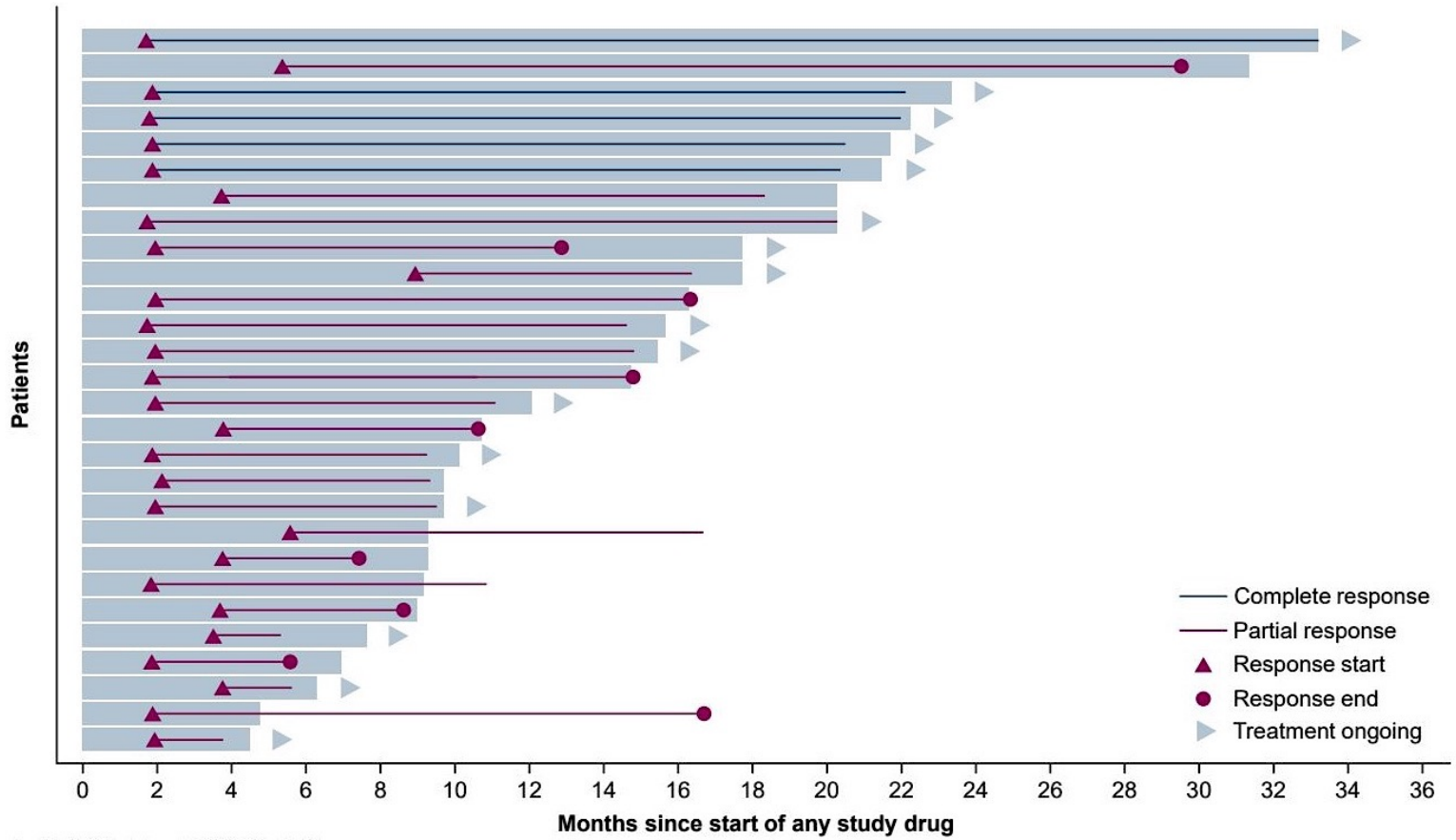


Illuzzi G et al. *Clin Cancer Res* 2022;28(21):4724-36.

Phase I/II PETRANHA: Responses

	All patients (N=34)*
ORR, % (80% CI)	82.4 (71.1–90.5)
Best overall response, n (%)	
Complete response	5 (14.7)
Partial response	23 (67.6)
Stable disease	4 (11.8)
Progressive disease	1 (2.9)
Not evaluable	1 (2.9)
Median DoR, months (80% CI)	24.2 (14.4–NC)

Per RECIST v1.1 and PCWG3 criteria. Data cutoff 10 June 2025.
*Response evaluable analysis set.
CI, confidence interval; DoR, duration of response; NC, not calculated; ORR, objective response rate; PCWG3, Prostate Cancer Working Group 3; RECIST v1.1, Response Evaluation Criteria in Solid Tumors version 1.1



Per RECIST v1.1 and PCWG3 criteria.
Response evaluable analysis set. Data cutoff 10 June 2025.
PCWG3, Prostate Cancer Working Group 3; RECIST v1.1, Response Evaluation Criteria in Solid Tumors version 1.1

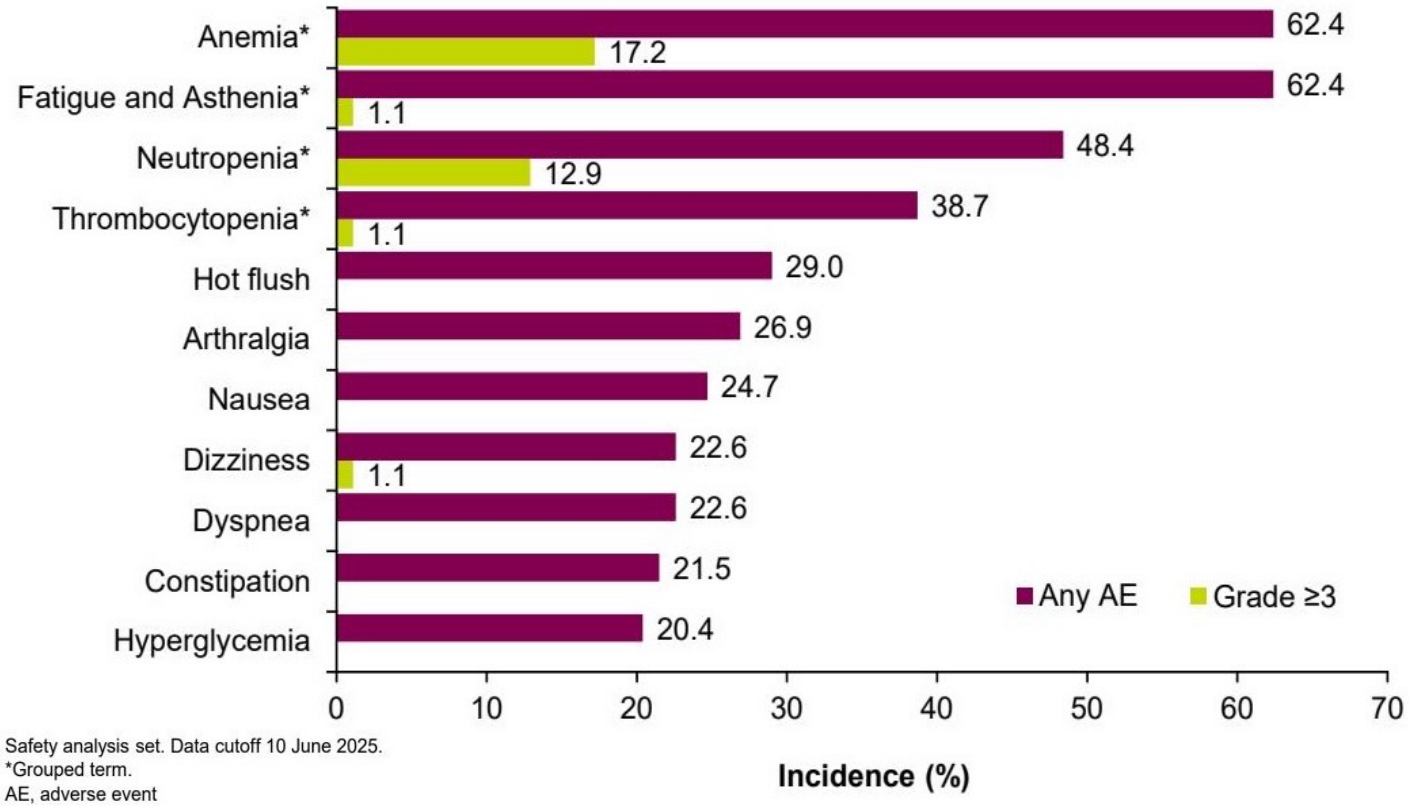
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Azad A et al. Genitourinary Cancers Symposium 2026;Abstract 177.

Phase I/II PETRANHA: Safety Data, Common AEs

Safety parameter, n (%)	mHSPC (N=93)
Any AE	92 (98.9)
Causally related to saruparib*	83 (89.2)
Any Grade ≥3 AE	45 (48.4)
Causally related to saruparib*	29 (31.2)
Any serious AE	24 (25.8)
Causally related to saruparib*	8 (8.6)
Saruparib / ARPI discontinuation due to AE†	7 (7.5) / 3 (3.2)
Saruparib / ARPI dose reduction due to AE†	24 (25.8) / 3 (3.2)
Saruparib / ARPI dose interruption due to AE†	52 (55.9) / 36 (38.7)

*Irrespective of relationship to ARPI. †Irrespective of the action taken on other drugs.
AE, adverse event; ARPI, androgen receptor pathway inhibitor; mHSPC, metastatic hormone-sensitive prostate cancer



Safety analysis set. Data cutoff 10 June 2025.
*Grouped term.
AE, adverse event

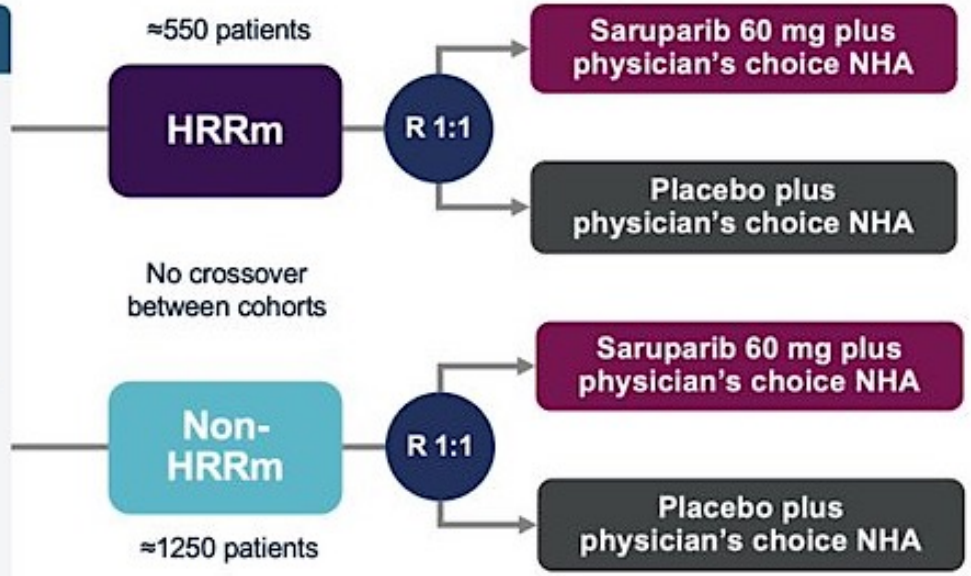
Memorial Sloan Kettering Cancer Center

Azad A et al. Genitourinary Cancers Symposium 2026;Abstract 177.

EvoPAR-Prostate01: Study Design

Eligibility criteria

- Aged ≥18 years
- Histologically confirmed mCSPC (*de novo* or recurrent low- or high-volume disease)
- ECOG PS 0–1
- Prospectively defined HRRm status*
- Must be receiving ADT throughout the study or have undergone bilateral orchiectomy, and must be suitable for treatment with NHAs
- No prior treatment with PARP inhibitors, CT, or NHAs in the metastatic setting†
- No suspected or prior history of myelodysplastic syndrome/acute myeloid leukemia



Select endpoints

HRRm cohort
- rPFS
- OS

Non-HRRm cohort
- rPFS
- OS

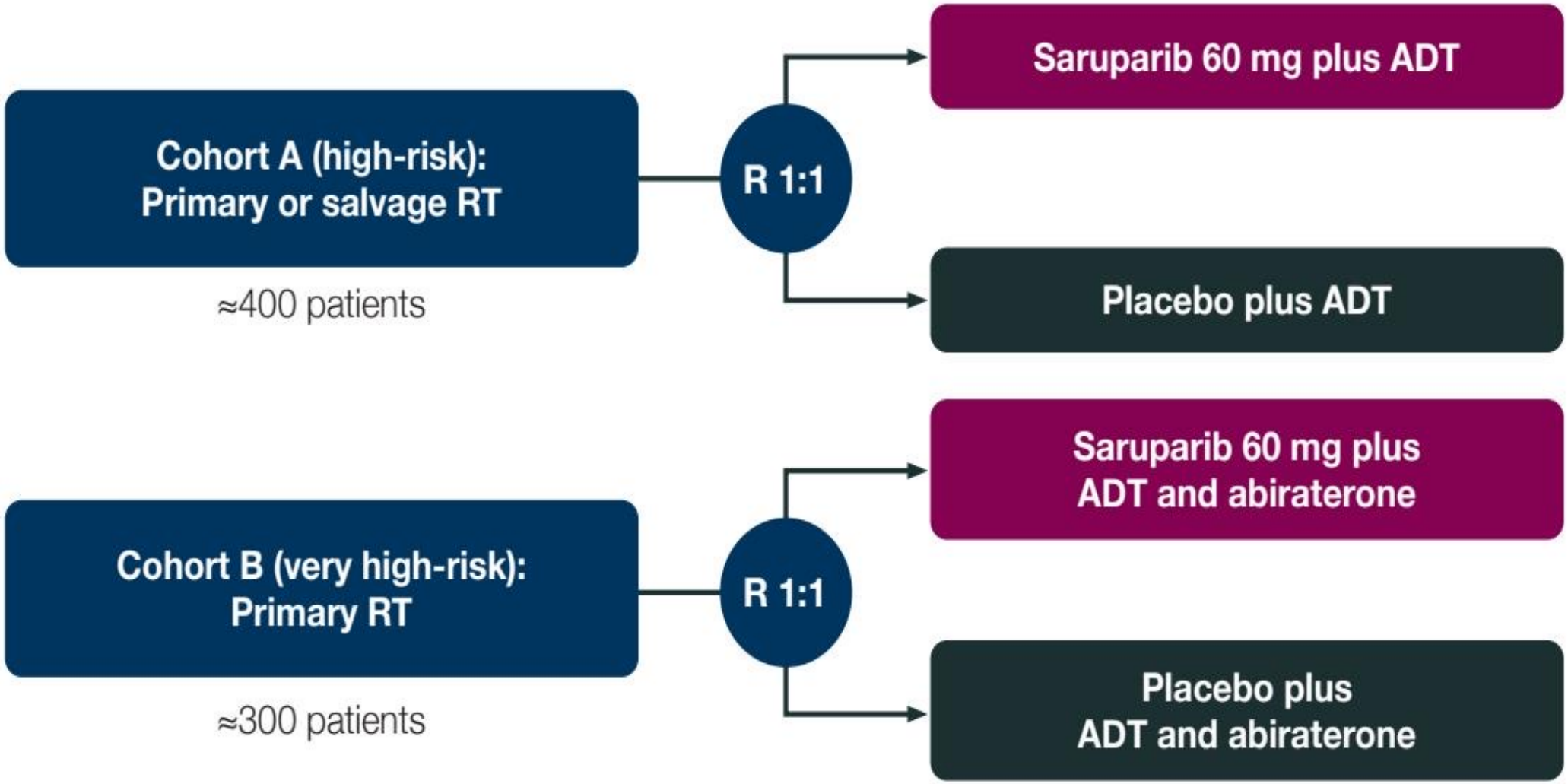
Statistical analyses

rPFS and OS will be tested for each cohort separately using a stratified log-rank test

Memorial Sloan Kettering Cancer Center

Agarwal N et al. AUA 2024

EvoPAR-Prostate02: Study Design



Memorial Sloan Kettering Cancer Center

McKay R et al. ASCO GU 2026

Current and future directions for PARPi in prostate cancer

- Earlier use of PARP inhibitors in HRR-mutant prostate cancer:
  - mCSPC
    - TALAPRO-3: rPFS benefit announced
    - EVOPAR-01
  - High-risk localized disease (EVOPAR02 saruparib adjuvant post-RT)

- PARPi combinations in non-HRR-mutated prostate cancer:
  - PARPi + ARPI: TALAPRO-3 and EVOPAR01 in mCSPC
  - PARPi + radioligand therapy
  - PARPi + other DNA repair inhibitors (phase 1)

Discussion Questions

74-year-old male: PSA 1,200; Gleason 8 prostate cancer with bone metastases.
Treated with apalutamide/leuprolide. PSA became undetectable.
2 years later PSA started increasing and now is 2. Bone scan, CT negative.
PSMA PET shows PSMA-avid bone lesions.
Germline BRCA2 mutation.

Should I change therapy now or once there is convincing radiological progression?

Would the experts use a PARP inhibitor second line or lutetium? If a PARP inhibitor, would you use monotherapy or combine with an alternative ARPI?

RTP
RESEARCH
TO PRACTICE

Discussion Questions (Continued)

74-year-old male: PSA 1,200; Gleason 8 prostate cancer with bone metastases.
Treated with apalutamide/leuprolide. PSA became undetectable.
2 years later PSA started increasing and now is 2. Bone scan, CT negative.
PSMA PET shows PSMA-avid bone lesions.
Germline BRCA2 mutation.

Are there substantial differences between different PARP inhibitors?

In which situations will you include a PARPi for hormone-sensitive metastatic disease? Which exact regimen — which PARPi and which endocrine partner?

RTP
RESEARCH
TO PRACTICE

Discussion Questions

65 y/o M with h/o CLL on surveillance and early-stage prostate cancer, s/p XRT but developed relapse in bone, abdominal LN and lung 4-5 years later. Germline PALB2.

How would you treat this patient?

What are your genomic criteria to use a PARPi? What are the experts' thoughts on using PARP inhibitors in patients who do not have HRR deficiency?

Do you use preemptive GI medications in patients receiving PARPi?

RTP
RESEARCH
TO PRACTICE

Discussion Questions

69-year-old man, castration-resistant metastatic disease. Germline BRCA2. Significant biochemical and radiographic response to olaparib but has required serial dose reductions because of fatigue, anemia, thrombocytopenia and intermittent neutropenia.

Is there evidence to support intermittent PARP inhibitor therapy or rechallenge strategies in mCRPC patients with durable responses?

How would you manage persistent cytopenias in a patient deriving ongoing benefit from PARPi?

Would you consider switching to an alternative agent?

For hormone-resistant metastatic disease, which PARPi and which endocrine partner do you prefer?

RTP
RESEARCH
TO PRACTICE

Discussion Questions (Continued)

69-year-old man, castration-resistant metastatic disease. Germline BRCA2. Significant biochemical and radiographic response to olaparib but has required serial dose reductions because of fatigue, anemia, thrombocytopenia and intermittent neutropenia.

How is saruparib different from other PARPi, and what are your thoughts about the ongoing trial in localized disease? Has PARP inhibition been looked at in that setting?

RTP
RESEARCH
TO PRACTICE

Agenda

Module 1: Evolving Management of Nonmetastatic Hormone-Sensitive Prostate Cancer (HSPC) — Dr Antonarakis

Module 2: Current and Future Hormonal Treatment for Metastatic HSPC (mHSPC) — Dr Aggarwal

Module 3: Current and Future Role of Regimens Combining PARP Inhibitors and AR Pathway Inhibitors in Metastatic Prostate Cancer — Dr Abida

Module 4: Emerging Role of AKT Inhibition for Patients with mHSPC — Dr Fizazi

Module 5: Current and Future Use of Radiopharmaceuticals in Metastatic Prostate Cancer — Dr McKay

RTP RESEARCH TO PRACTICE

Centre Oscar Lambret

unicancer HAUTS-DE-FRANCE

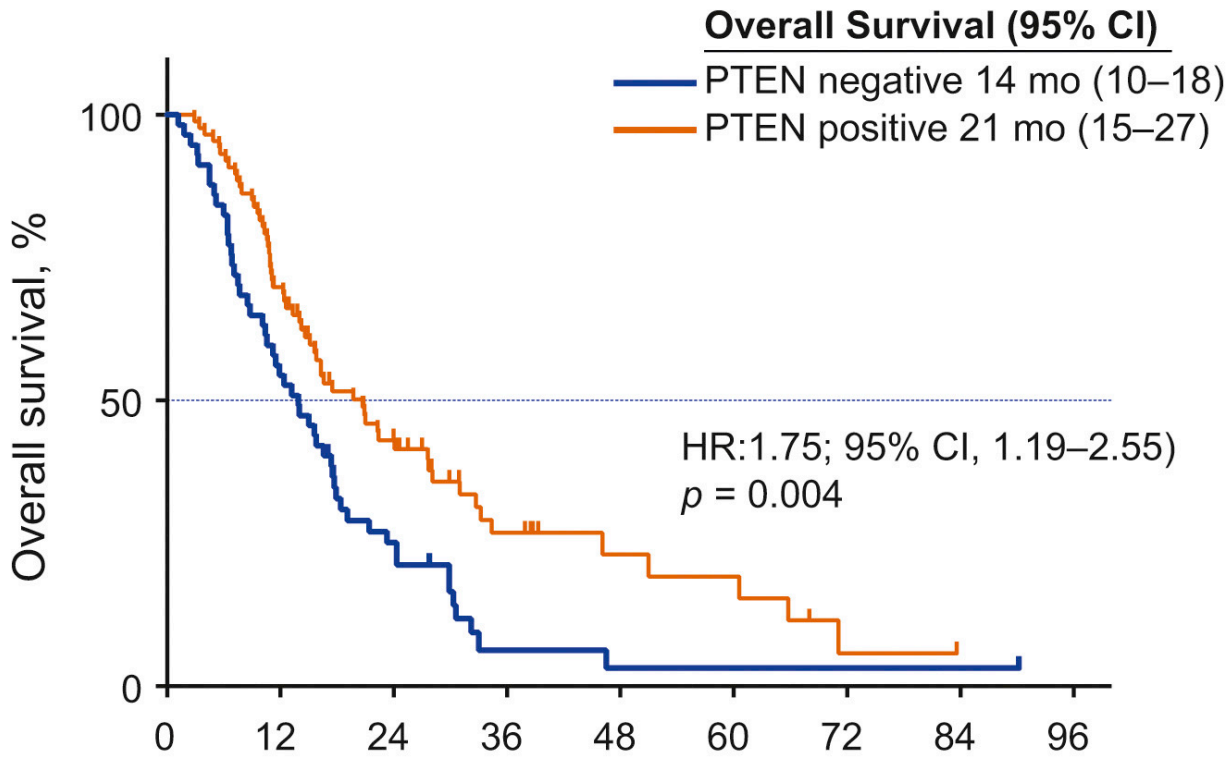
Targeting Akt in metastatic prostate cancer

Karim Fizazi, MD, PhD

France



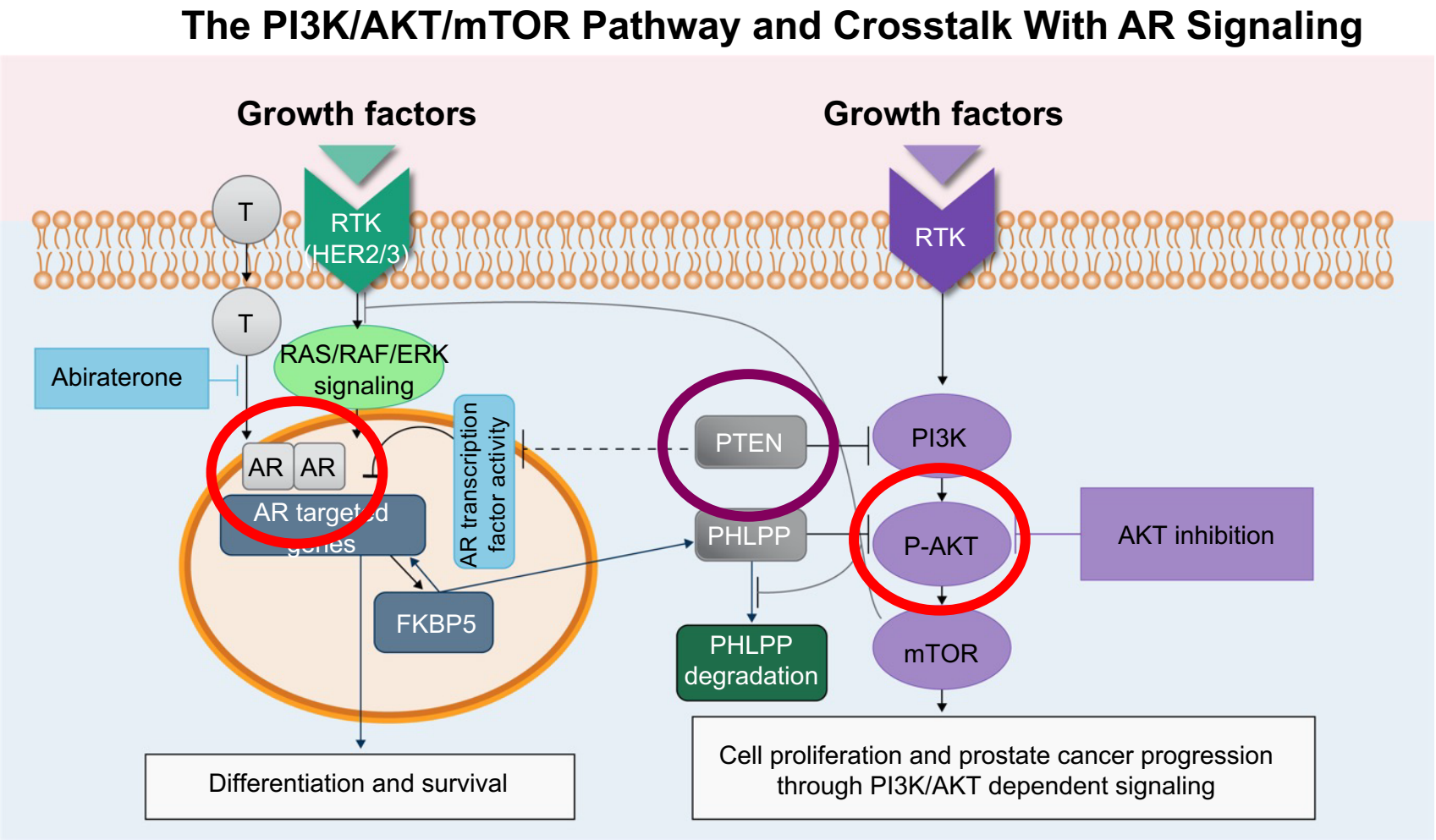
PTEN loss associated with worse outcome in Prostate Cancer



No. at risk								
PTEN negative	57	32	14	3	2	2	2	1
PTEN positive	87	58	30	13	7	6	2	0

Ferraldeschi R et al. *Eur Urol.* 2015 67:795-802.

PI3K/AKT/PTEN pathway crosstalk with the AR pathway

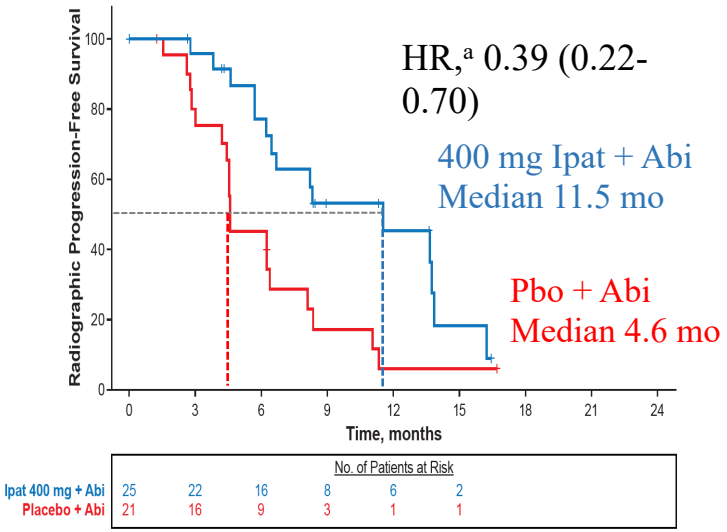


Fizazi K, ASCO GU 2021, Abstr TPS178

Abiraterone +/- Ipatasertib (Akt inhibitor)
Randomised phase 2 trial: PFS

400 mg Ipatasertib

PTEN loss

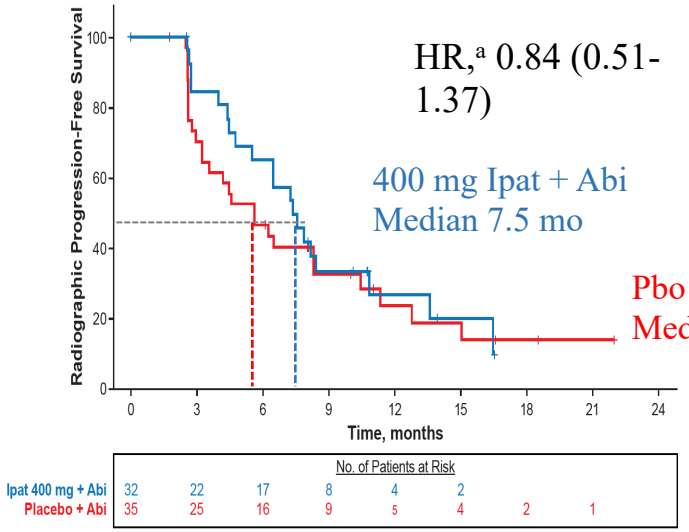


HR,[a] 0.39 (0.22-0.70)

400 mg Ipat + Abi
Median 11.5 mo

Pbo + Abi
Median 4.6 mo

PTEN non-loss



HR,[a] 0.84 (0.51-1.37)

400 mg Ipat + Abi
Median 7.5 mo

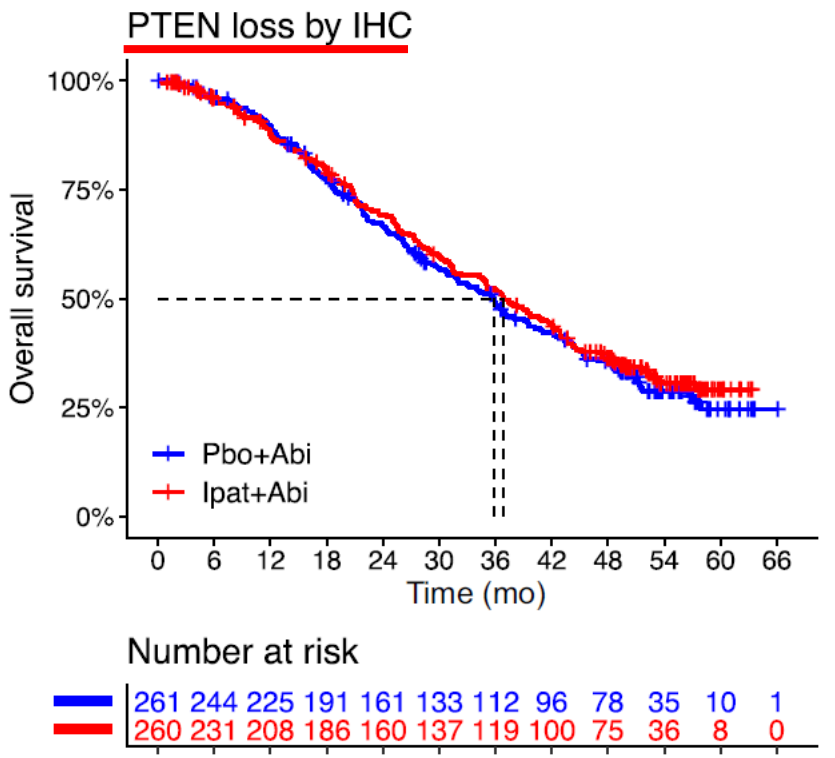
Pbo + Abi
Median 5.6 mo



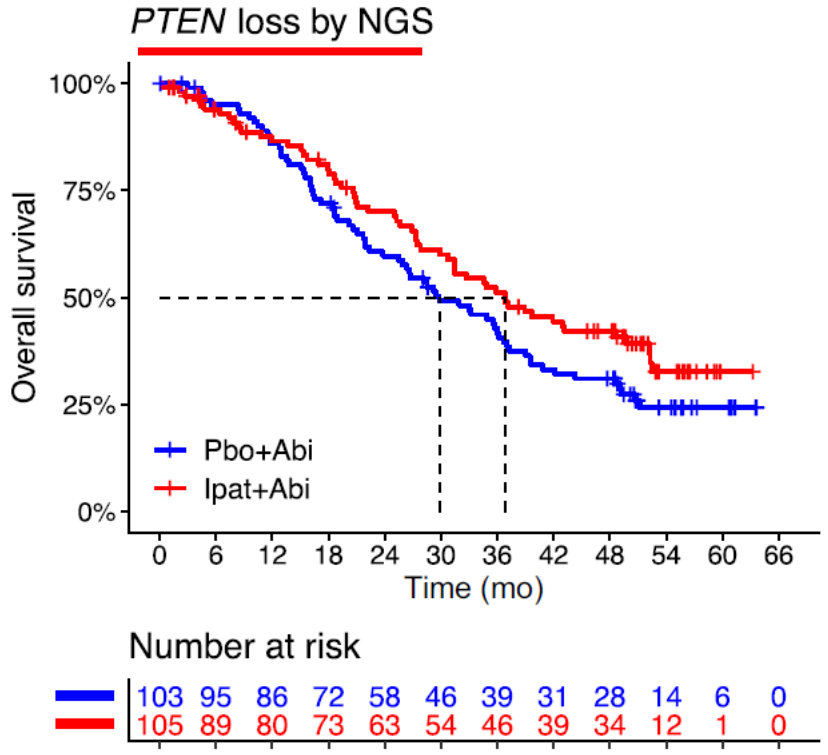


Overall survival is not improved by Ipatasertib in mCRPC (IPATential150 trial)

	Events	Patients	Median OS, mo (95% CI)	HR (95% CI), stratified	*p* value
Pbo+Abi	170	261	35.8 (30.8–39.6)	0.94 (0.76–1.17)	0.57
Ipat+Abi	158	260	36.8 (31.4–42.1)		



	Events	Patients	Median OS, mo (95% CI)	HR (95% CI), non stratified
Pbo+Abi	72	103	29.8 (25.5–37.0)	0.76 (0.54–1.07)
Ipat+Abi	58	105	36.8 (30.7–52.2)	



de Bono J et al. Eur Urol. 2025 Jun;87(6):672-682.

CAPItello-281 Study Design

A global, multicentre, randomized, double-blind, Phase 3 study

Patients with PTEN deficient *de novo* mHSPC

- PTEN deficiency: (diagnostic cut-off of **≥90%** of viable malignant cells with **no specific cytoplasmic staining** by IHC)*
  - i.e. ≤10% of cells expressing PTEN by IHC

Of ~6,200 patients submitting tumour tissue **97%** had a valid IHC result and **25%** were **PTEN deficient**

1,012 patients (R 1:1)

Capivasertib	400 mg BID 4 days on, 3 days off
Abiraterone/pred **+ ADT**	1000 mg/5 mg QD + ADT
Placebo	400 mg BID 4 days on, 3 days off
Abiraterone/pred **+ ADT**	1000 mg/5 mg QD + ADT

Primary endpoint

- **Investigator assessed rPFS**

Secondary endpoints

- Overall survival
- Time to first subsequent therapy
- Symptomatic skeletal-event free survival
- Time to pain progression
- Time to castration resistance
- Time to PSA progression

Exploratory *post-hoc* PTEN deficiency subgroups

Stratification factors:†

- M1 volume (CHAARTED criteria) and visceral mets
- Geography

Study timeline

Current analysis

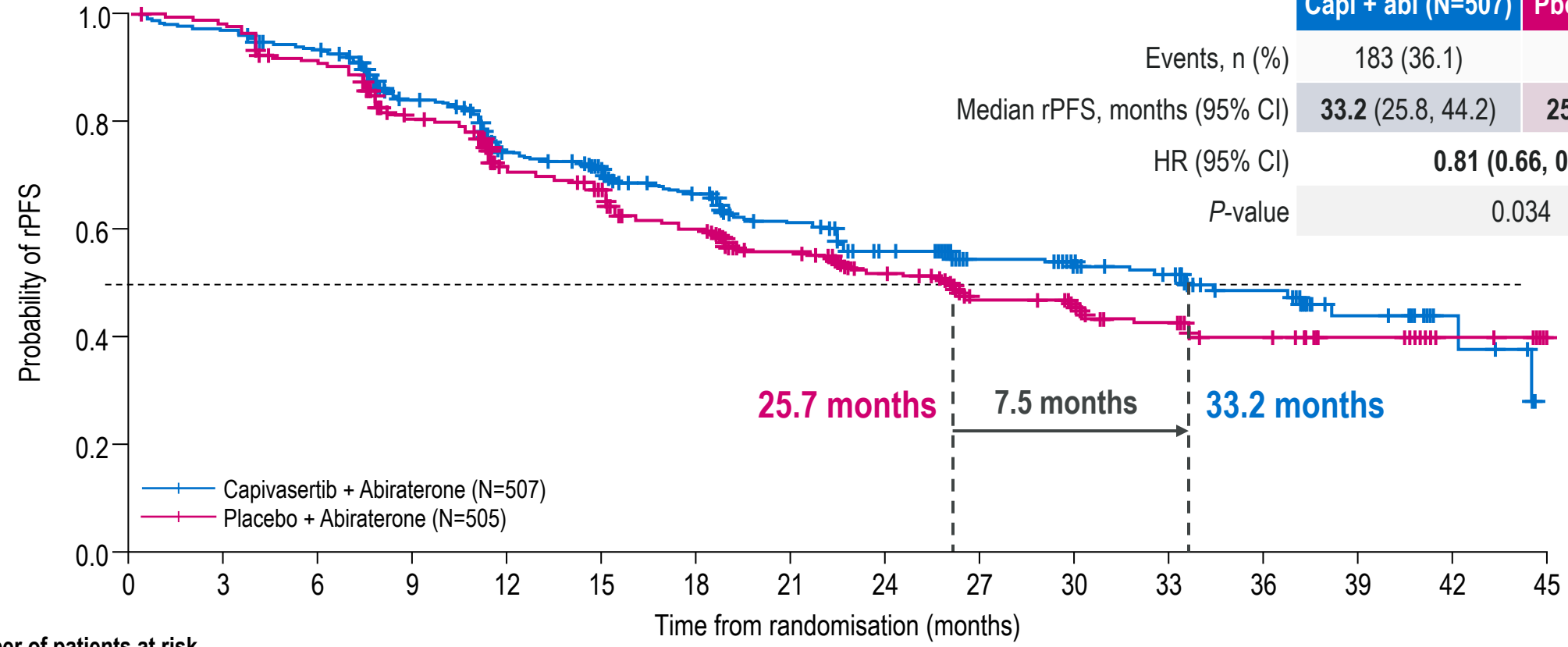
Enrolment period	Primary rPFS DCO	Final OS DCO
13 July 2020–5 Feb 2024	7 Oct 2024 Median rPFS follow-up:‡ ~18 mo	Planned for 522 deaths (52.2% maturity)

NCT04493853. Full eligibility criteria available in the online article. *Determined using investigational antibody for PTEN (SP218) (Roche Diagnostics).
†High-vol. disease with visceral mets, high-vol disease without visceral mets, low-vol. disease; North America; Western Europe and Australia; Latin America and Eastern Europe; Asia. ‡In censored patients.
ADT, androgen deprivation therapy; BID, twice daily; IHC, immunohistochemistry; mHSPC, metastatic hormone-sensitive prostate cancer; pred, prednisone/prednisolone; QD, once daily; rPFS, radiographic progression-free survival

BERLIN 2025 ESMO congress

CAPItello-281 Primary endpoint: investigator-assessed rPFS

	Capi + abi (N=507)	Pbo + abi (N=505)
Events, n (%)	183 (36.1)	215 (42.6)
Median rPFS, months (95% CI)	**33.2** (25.8, 44.2)	**25.7** (22.0, 29.9)
HR (95% CI)	**0.81 (0.66, 0.98)**	
P-value	0.034	



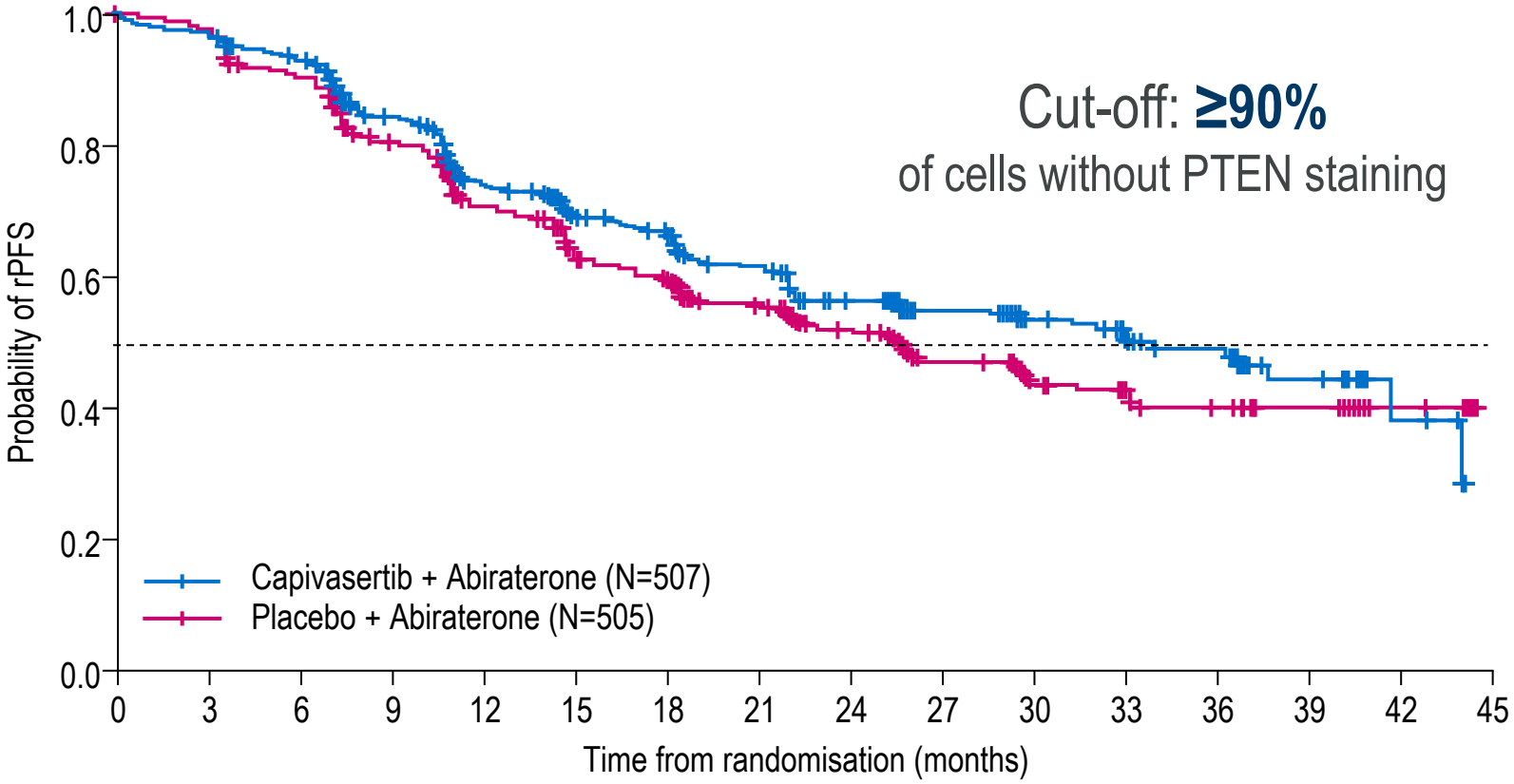
Number of patients at risk

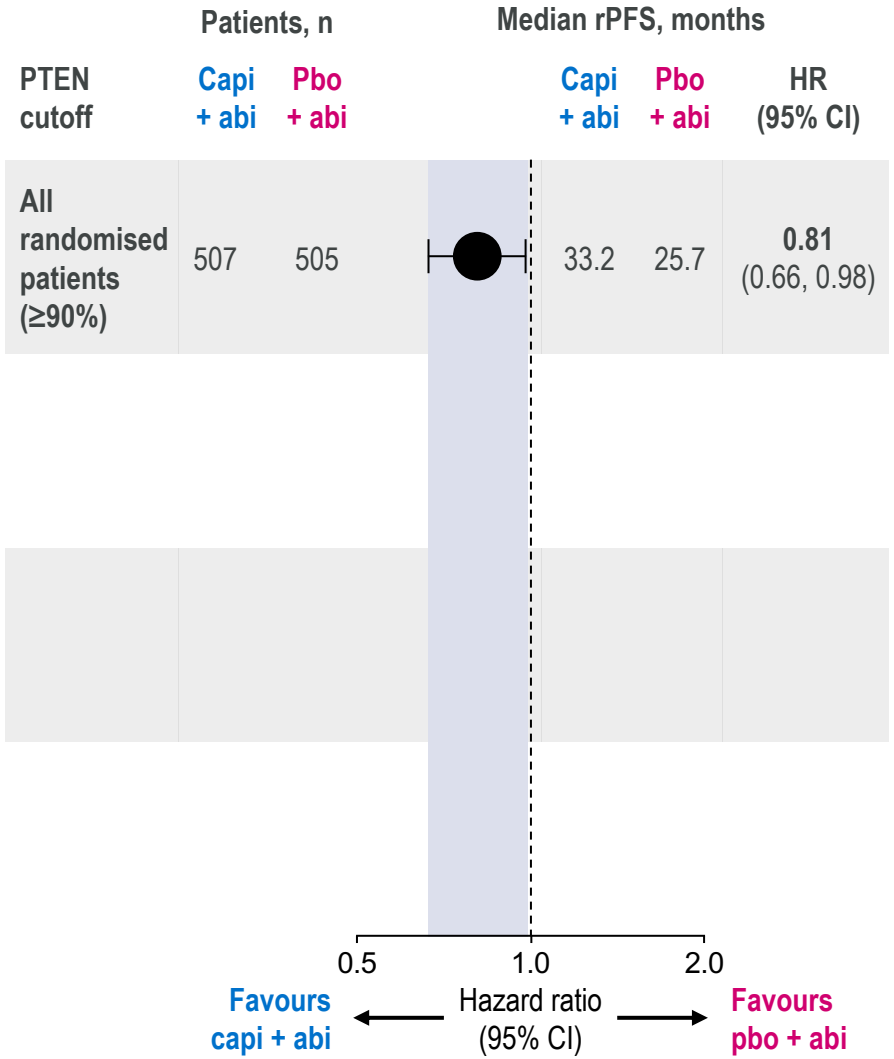
Capi + abi	507	460	435	353	282	233	217	165	123	93	69	62	41	21	6	0
Pbo + abi	505	479	440	359	276	215	198	154	113	83	59	51	37	23	8	0

A stratified log-rank test was used to calculate two-sided P values. HRs and 95% CIs were calculated using a stratified Cox proportional-hazards model. Median follow-up: 18.4 months (capi + abi), 18.5 months (pbo + abi)
abi, abiraterone; capi, capivasertib; CI, confidence interval; HR, hazard ratio; pbo, placebo; rPFS, radiographic progression-free survival

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CAPItello-281 PTEN subgroups: investigator-assessed rPFS



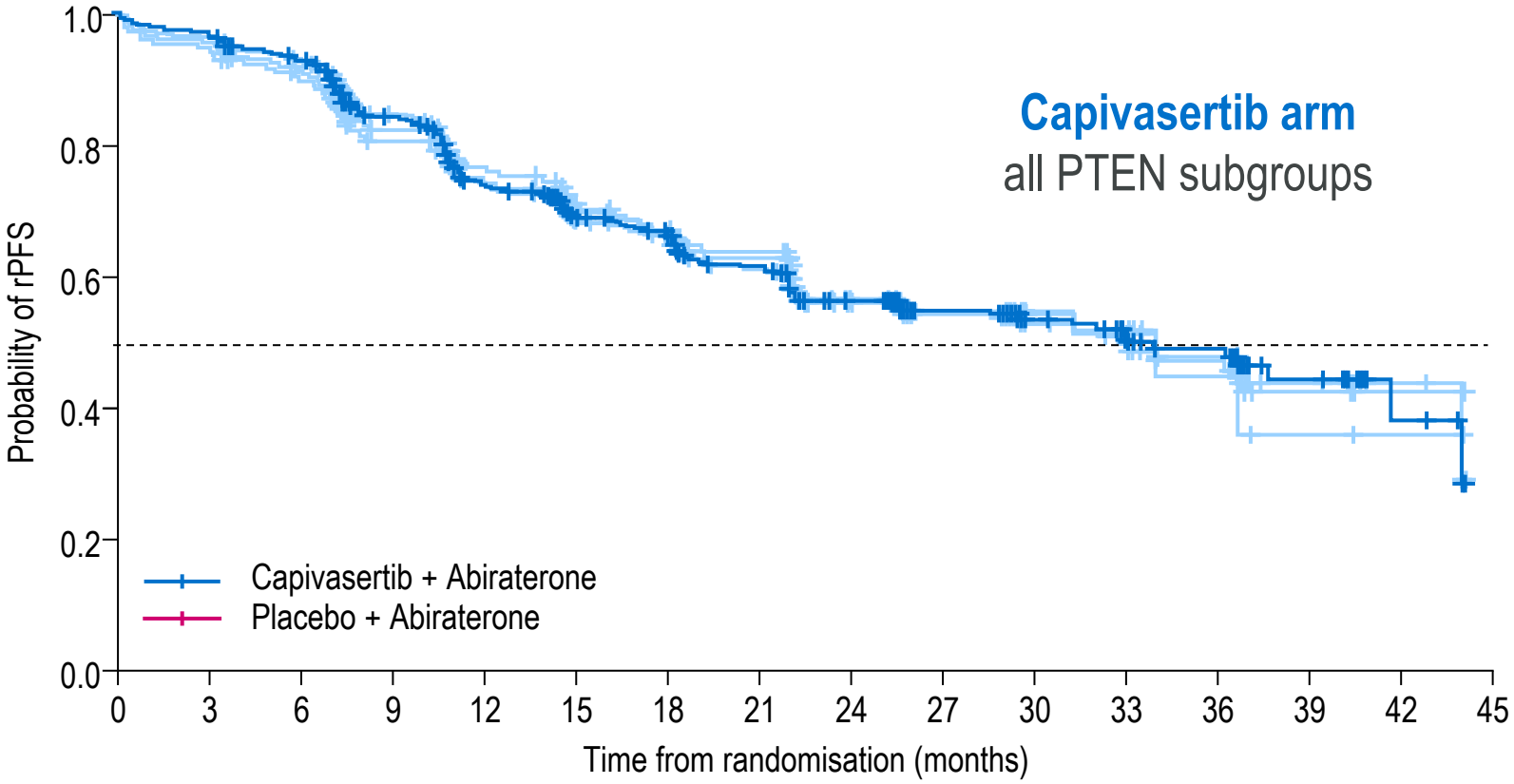


rPFS maturity in PTEN subgroups was consistent with the overall population

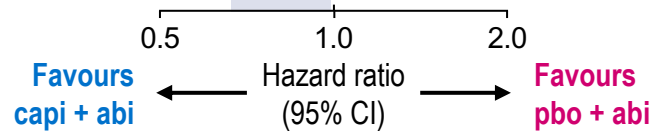
abi, abiraterone; capi, capivasertib; CI, confidence interval; HR, hazard ratio; pbo, placebo; rPFS, radiographic progression-free survival

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CAPItello-281 PTEN subgroups: investigator-assessed rPFS



PTEN cutoff	Patients, n: Capi + abi	Patients, n: Pbo + abi	Median rPFS, months: Capi + abi	Median rPFS, months: Pbo + abi	HR (95% CI)
All randomised patients (≥90%)	507	505	33.2	25.7	**0.81** (0.66, 0.98)
≥95%	404	410	33.2	22.7	**0.75** (0.60, 0.94)
≥99%	205	196	34.1	22.4	**0.71** (0.52, 0.97)
100%	169	162	34.1	22.1	**0.68** (0.48, 0.96)

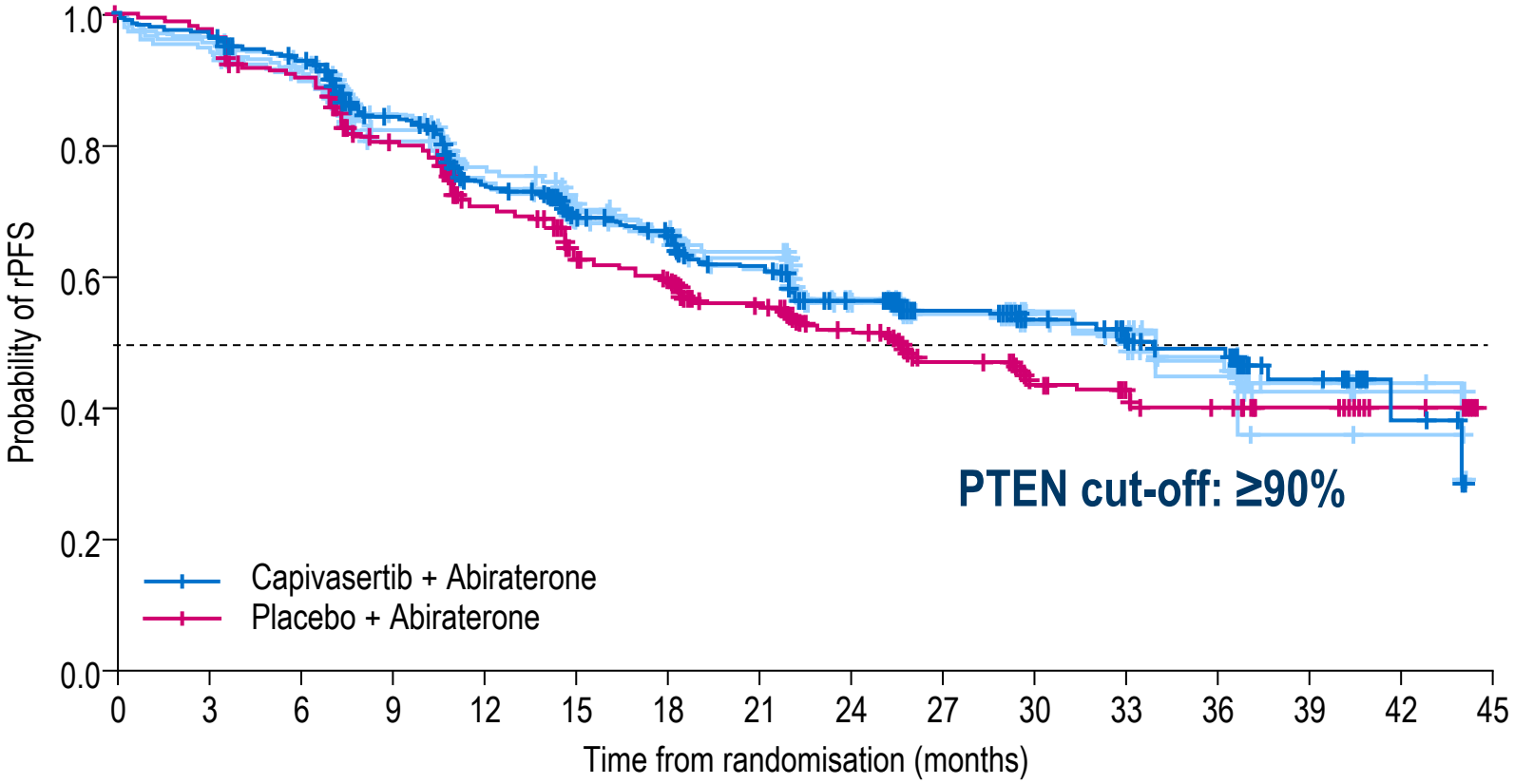


rPFS maturity in PTEN subgroups was consistent with the overall population

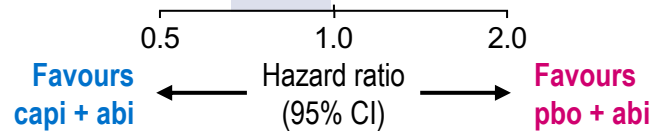
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CAPItello-281 PTEN subgroups: investigator-assessed rPFS



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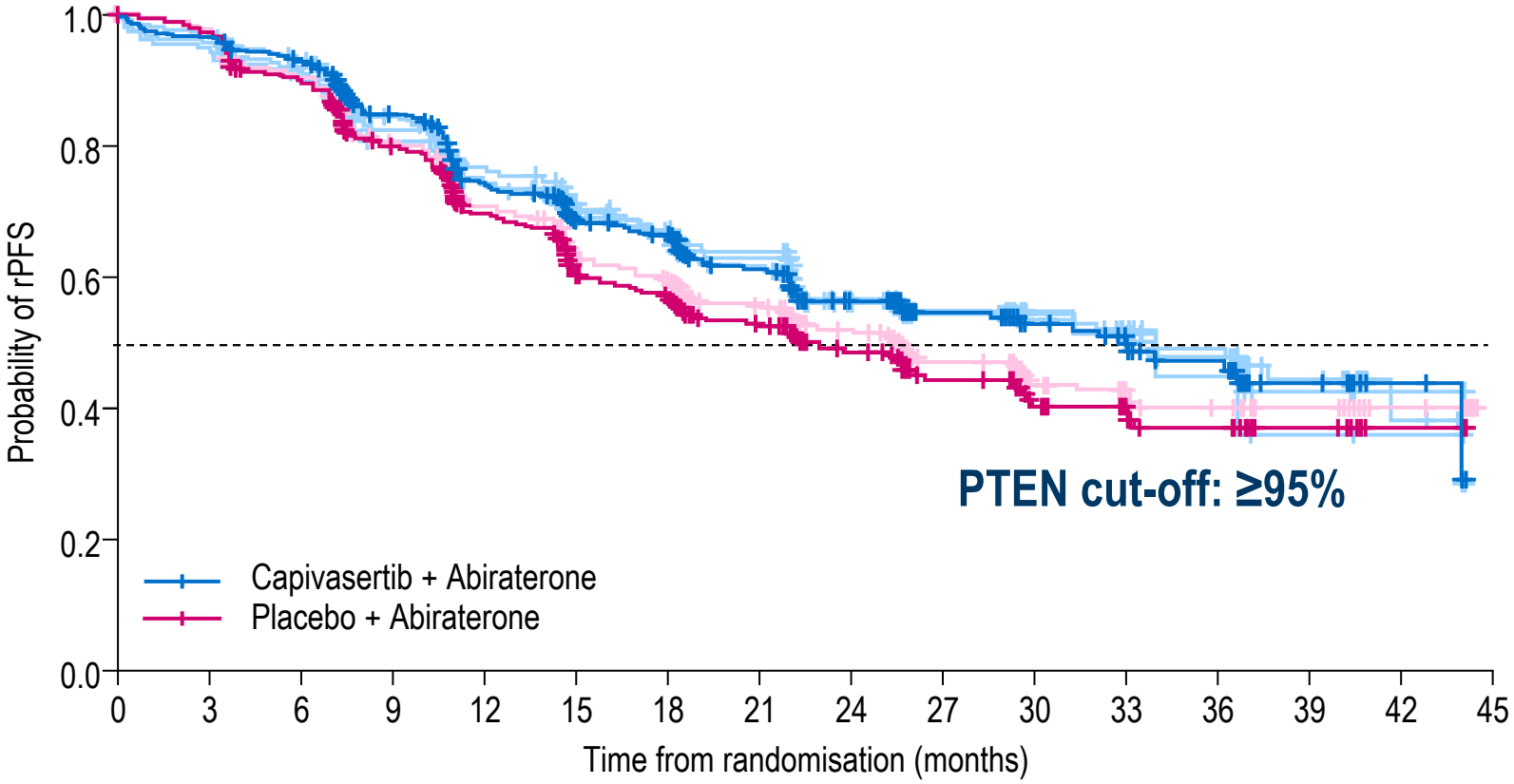


rPFS maturity in PTEN subgroups was consistent with the overall population

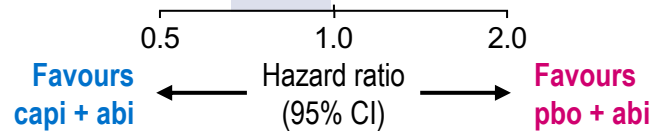
abi, abiraterone; capi, capivasertib; CI, confidence interval; HR, hazard ratio; pbo, placebo; rPFS, radiographic progression-free survival

Karim Fizazi MD, PhD

CAPItello-281 PTEN subgroups: investigator-assessed rPFS



PTEN cutoff	Patients, n: Capi + abi	Patients, n: Pbo + abi	Median rPFS, months: Capi + abi	Median rPFS, months: Pbo + abi	HR (95% CI)
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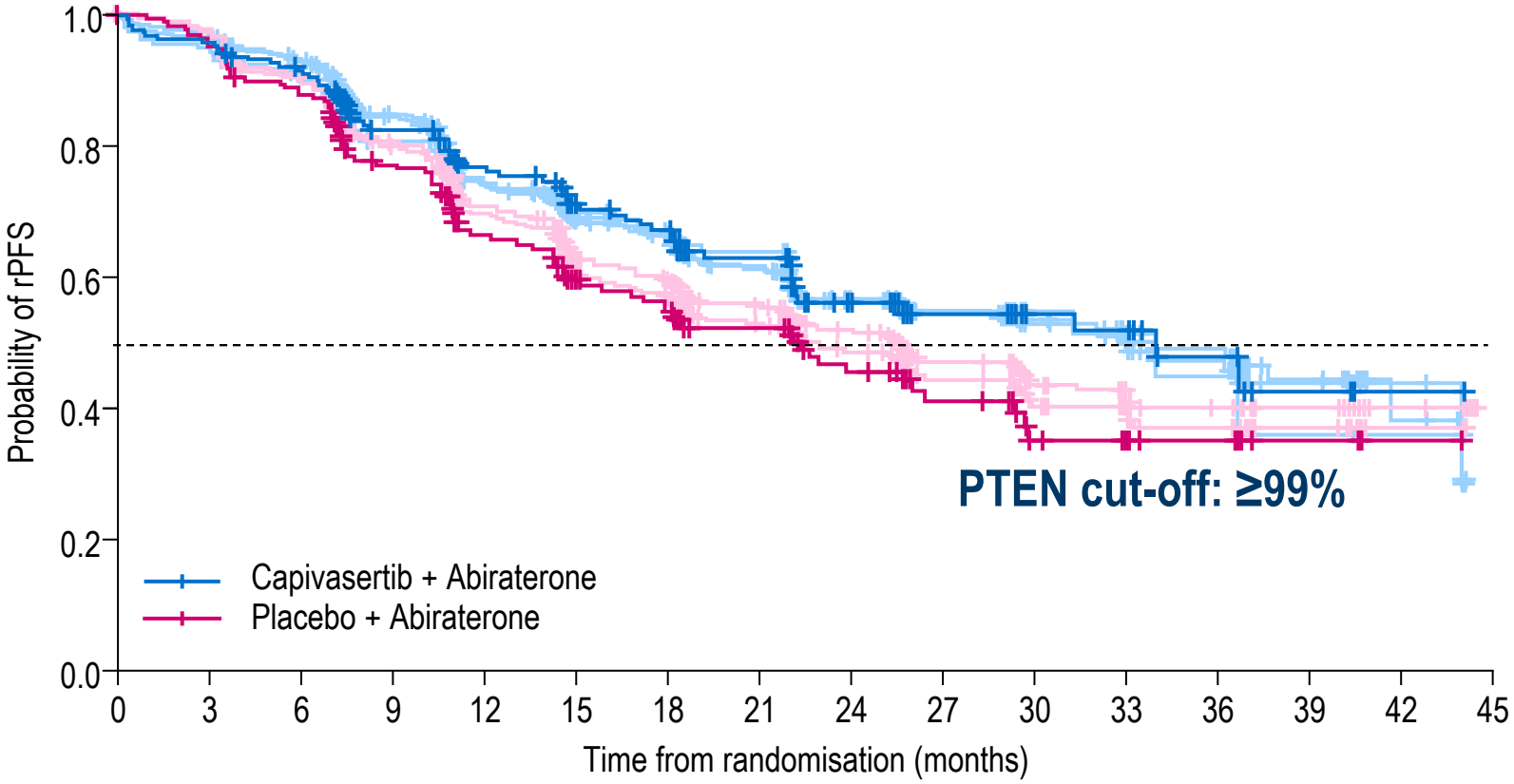


rPFS maturity in PTEN subgroups was consistent with the overall population

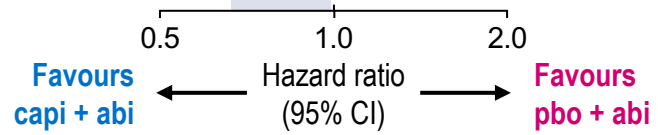
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Karim Fizazi MD, PhD

CAPItello-281 PTEN subgroups: investigator-assessed rPFS



PTEN cutoff	Patients, n: Capi + abi	Patients, n: Pbo + abi	Median rPFS, months: Capi + abi	Median rPFS, months: Pbo + abi	HR (95% CI)
All randomised patients (≥90%)	507	505	33.2	25.7	**0.81** (0.66, 0.98)
≥95%	404	410	33.2	22.7	**0.75** (0.60, 0.94)
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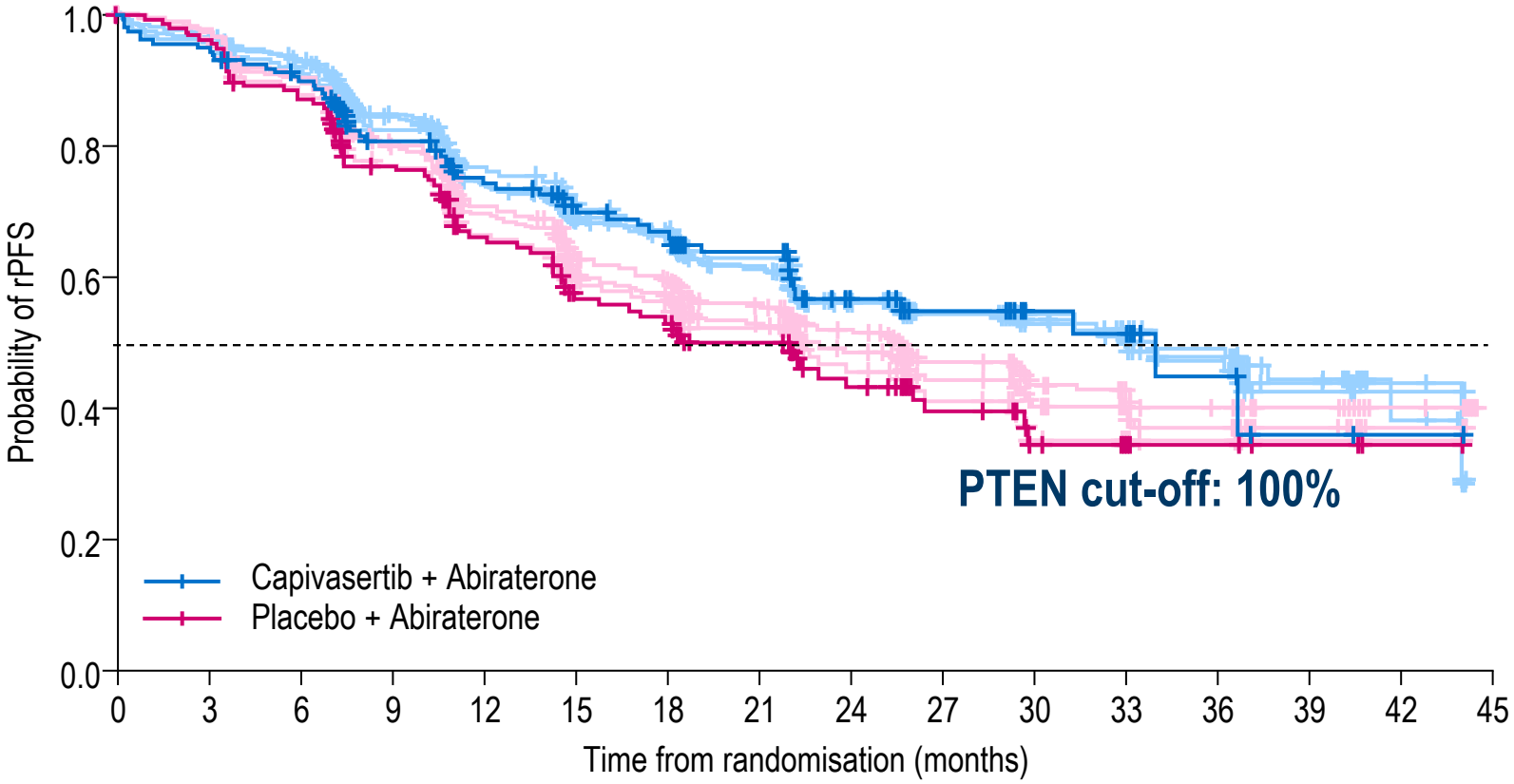


rPFS maturity in PTEN subgroups was consistent with the overall population

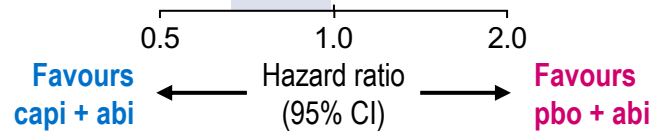
abi, abiraterone; capi, capivasertib; CI, confidence interval; HR, hazard ratio; pbo, placebo; rPFS, radiographic progression-free survival

Karim Fizazi MD, PhD

CAPItello-281 PTEN subgroups: investigator-assessed rPFS



PTEN cutoff	Patients, n: Capi + abi	Patients, n: Pbo + abi	Median rPFS, months: Capi + abi	Median rPFS, months: Pbo + abi	HR (95% CI)
All randomised patients (≥90%)	507	505	33.2	25.7	**0.81** (0.66, 0.98)
≥95%	404	410	33.2	22.7	**0.75** (0.60, 0.94)
≥99%	205	196	34.1	22.4	**0.71** (0.52, 0.97)
100%	169	162	34.1	22.1	**0.68** (0.48, 0.96)



rPFS maturity in PTEN subgroups was consistent with the overall population

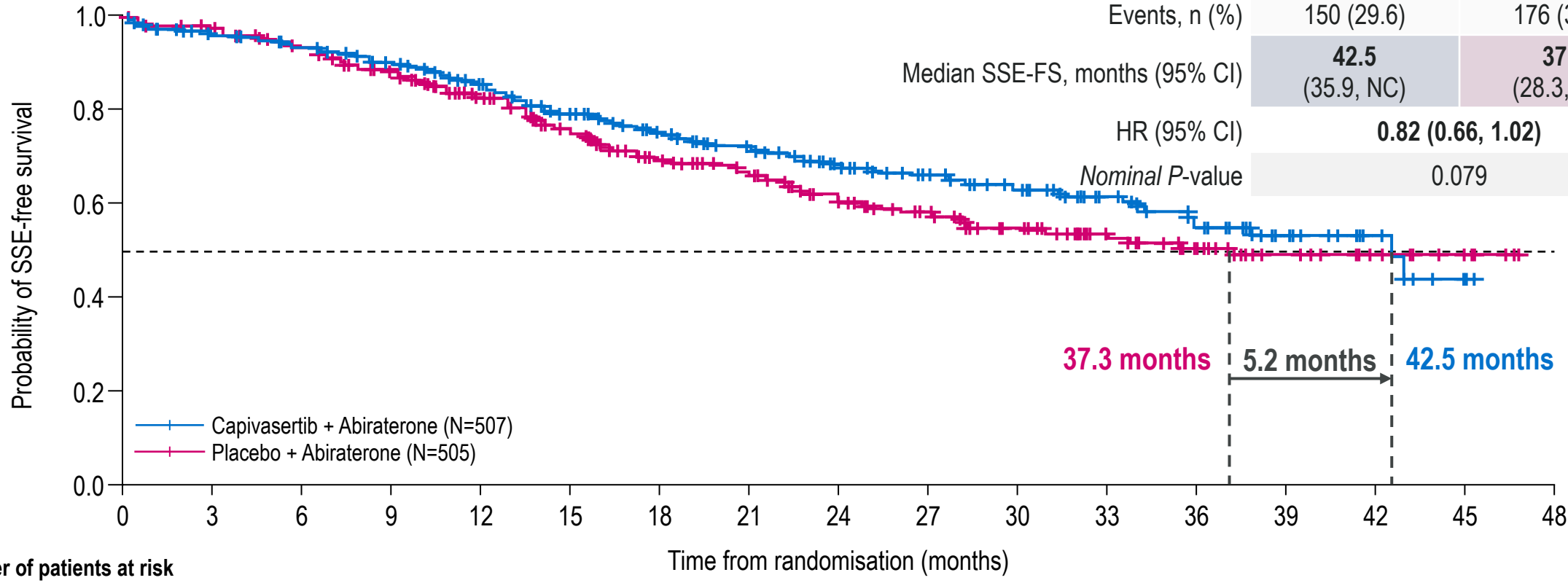
abi, abiraterone; capi, capivasertib; CI, confidence interval; HR, hazard ratio; pbo, placebo; rPFS, radiographic progression-free survival

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CAPItello-281: Symptomatic skeletal event-free survival

Composite endpoint of pathological fracture, spinal cord compression, use of radiation, surgical intervention, and death

	Capi + abi (N=507)	Pbo + abi (N=505)
Events, n (%)	150 (29.6)	176 (34.9)
Median SSE-FS, months (95% CI)	**42.5** (35.9, NC)	**37.3** (28.3, NC)
HR (95% CI)	**0.82 (0.66, 1.02)**	
Nominal P-value	0.079	



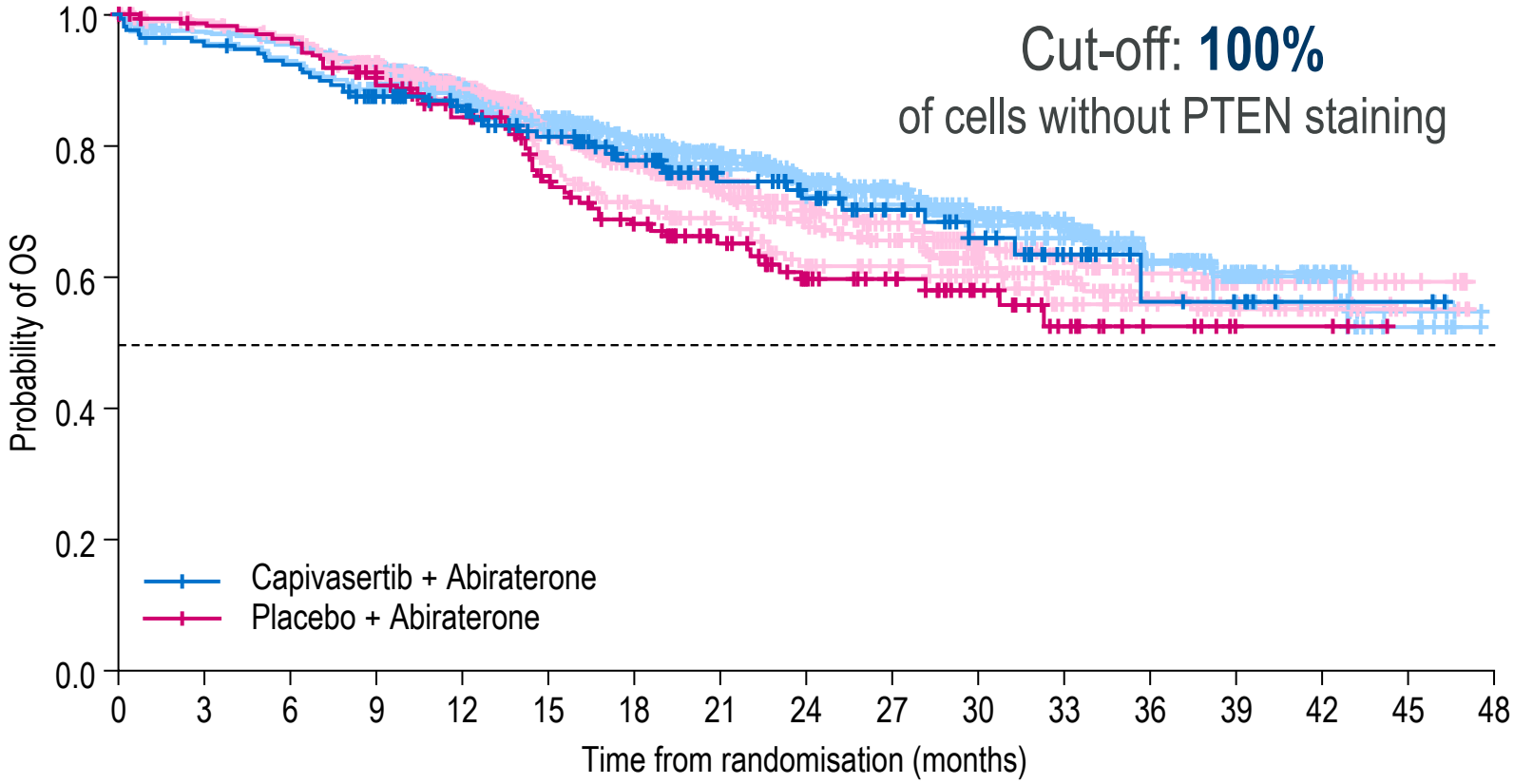
Number of patients at risk

Capi + abi	507	458	439	408	346	292	245	206	162	129	104	67	46	26	13	5	0
Pbo + abi	505	479	455	412	336	285	223	190	142	113	85	59	38	26	13	6	0

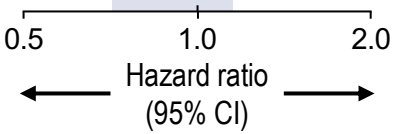
CI, confidence interval; HR, hazard ratio; NC, not calculable; pbo, placebo, SSE-FS, symptomatic skeletal event-free survival

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CAPItello-281 PTEN subgroups: OS



PTEN cutoff	Patients, n: Capi + abi	Patients, n: Pbo + abi	Median OS, months: Capi + abi	Median OS, months: Pbo + abi	HR (95% CI)
All randomised patients (≥90%)	507	505	NC	NC	0.90 (0.71, 1.15)
≥95%	404	410	NC	NC	0.80 (0.62, 1.04)
≥99%	205	196	NC	NC	0.77 (0.53, 1.12)
100%	169	162	NC	NC	0.77 (0.51, 1.14)

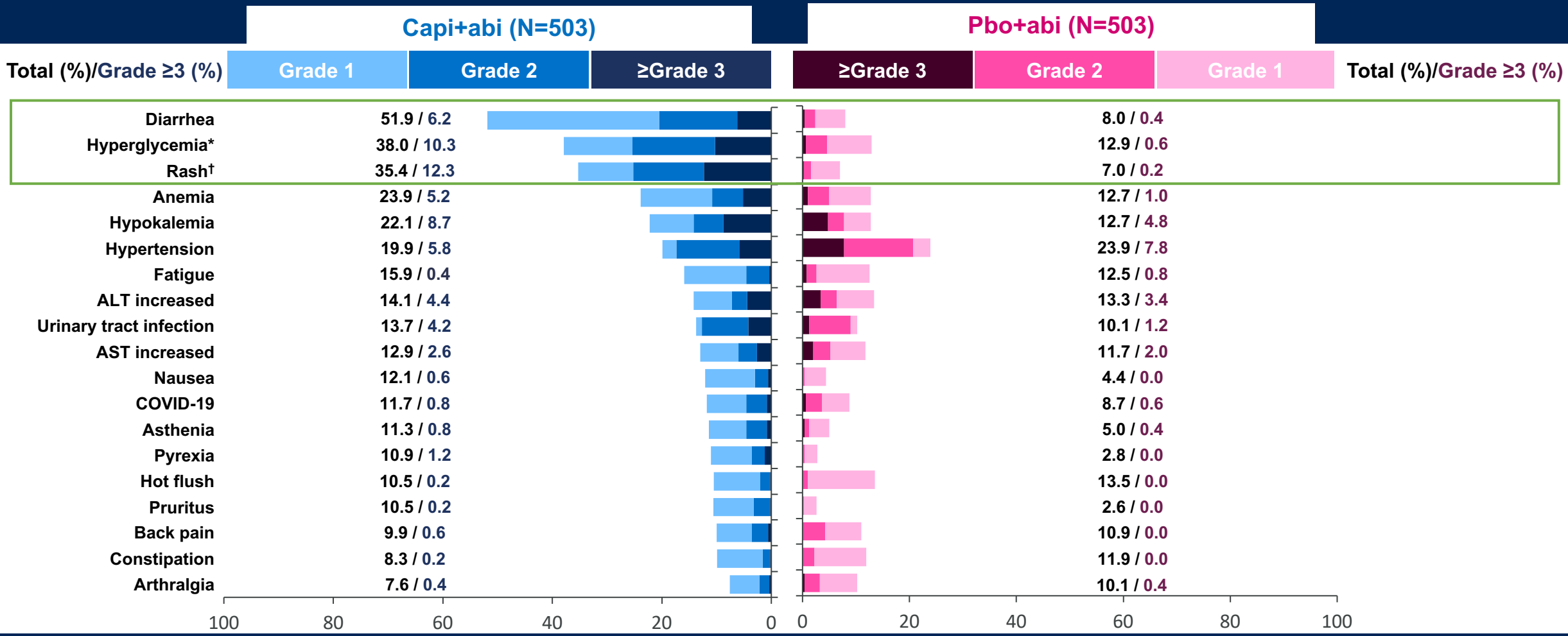


OS maturity in PTEN subgroups was broadly consistent with the overall population

abi, abiraterone; capi, capivasertib; CI, confidence interval; HR, hazard ratio; NC, not calculable; OS, overall survival; pbo, placebo

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CAPItello-281: investigator-reported AEs (≥10% of patients)

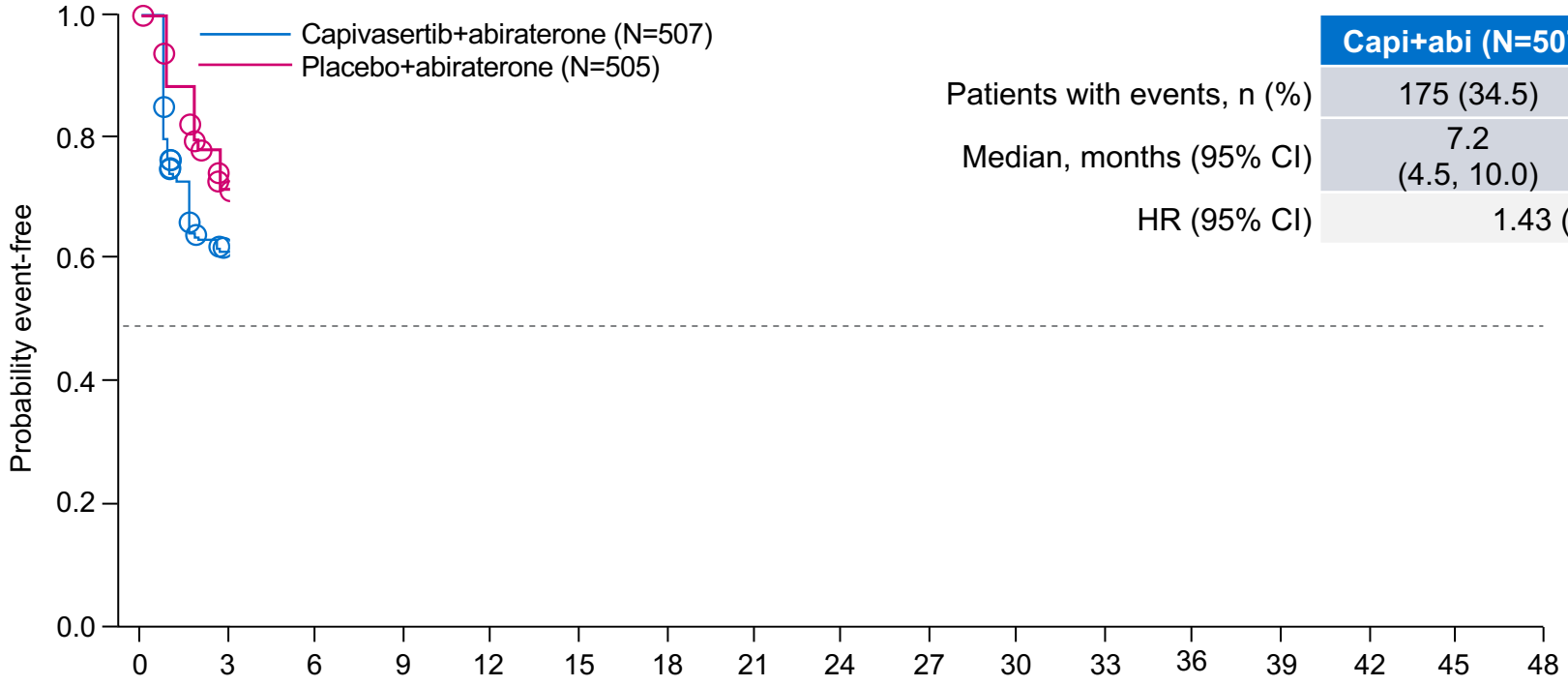


Diabetic ketoacidosis was reported in six patients (1.2%) in the capi+abi arm and zero patients in the pbo+abi arm

*Grouped term (includes the preferred terms of blood glucose increased, hyperglycemia); †Grouped term (includes the preferred terms of erythema, rash, rash erythematous, rash macular, rash maculo-papular, rash popular, rash pruritic). abi, abiraterone; AE, adverse event; ALT, alanine aminotransferase; AST, aspartate aminotransferase; capi, capivasertib; pbo, placebo; PTEN, phosphatase and tensin homolog

CAPItello-281: time to clinically meaningful decrease in FACT–P physical wellbeing (PWB)

PWB includes outcomes such as side effects, energy, nausea and pain



	Capi+abi (N=507)	Pbo+abi (N=505)
Patients with events, n (%)	175 (34.5)	152 (30.1)
Median, months (95% CI)	7.2 (4.5, 10.0)	14.6 (8.1, NC)
HR (95% CI)	1.43 (1.15, 1.78)	

Number of patients at risk

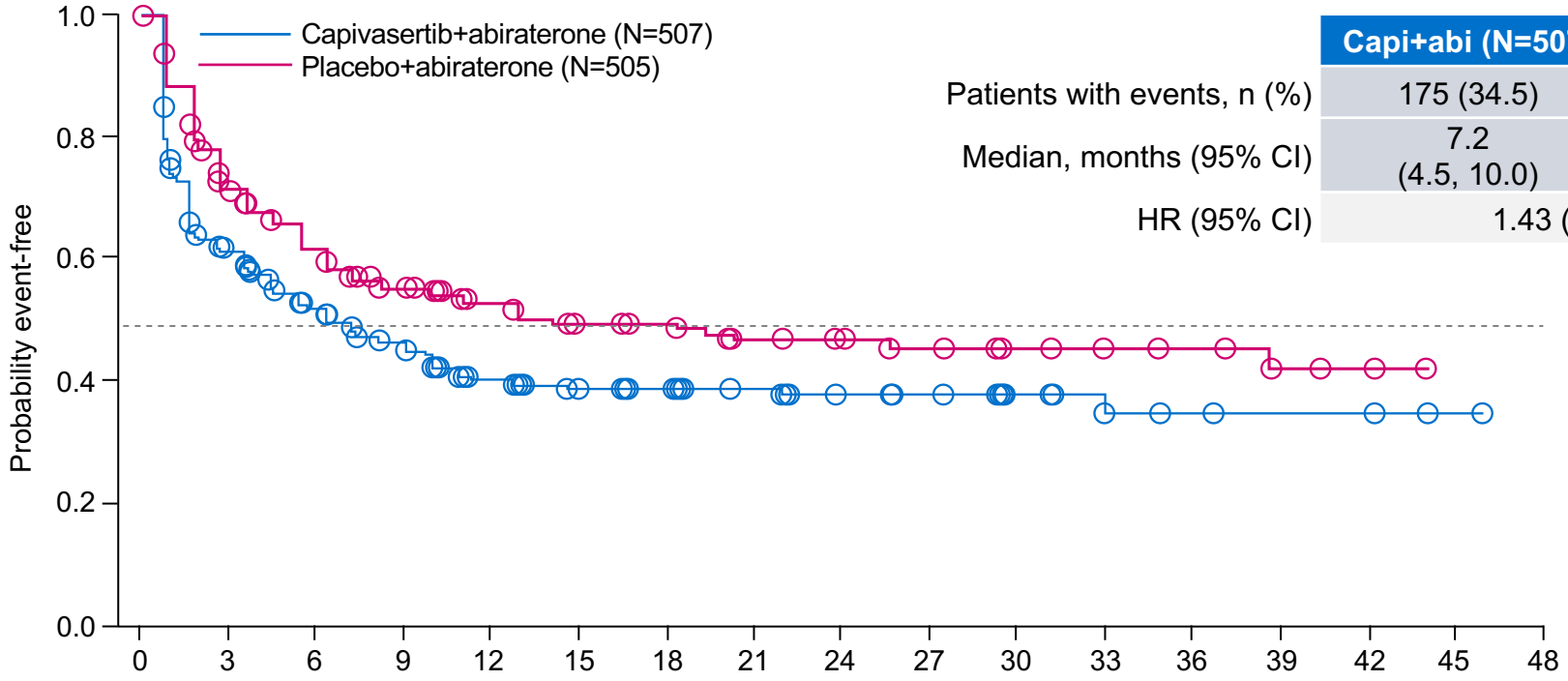
Capi+abi	507	179	143	114	81	65	55	42	34	31	17	8	7	4	4	1	0
Pbo+abi	505	214	180	142	111	77	68	51	39	32	23	17	14	10	5	0	0

Time to decrease in ≥3 points in FACT–P PWB is defined as the time from randomization until date of first confirmed clinically meaningful decrease before receiving subsequent anticancer therapy. If the decrease occurred after two or more missed PRO assessments, patients were censored at the time of last PRO assessment prior to the two missed visits. Circles represent censored observations

abi, abiraterone; capi, capivasertib; CI, confidence interval; FACT–P, Functional Assessment of Caner Therapy–Prostate Cancer; HR, hazard ratio; NC, non-calculable; pbo, placebo; PRO, patient-reported outcome

CAPItello-281: time to clinically meaningful decrease in FACT–P physical wellbeing (PWB)

PWB includes outcomes such as side effects, energy, nausea and pain



	Capi+abi (N=507)	Pbo+abi (N=505)
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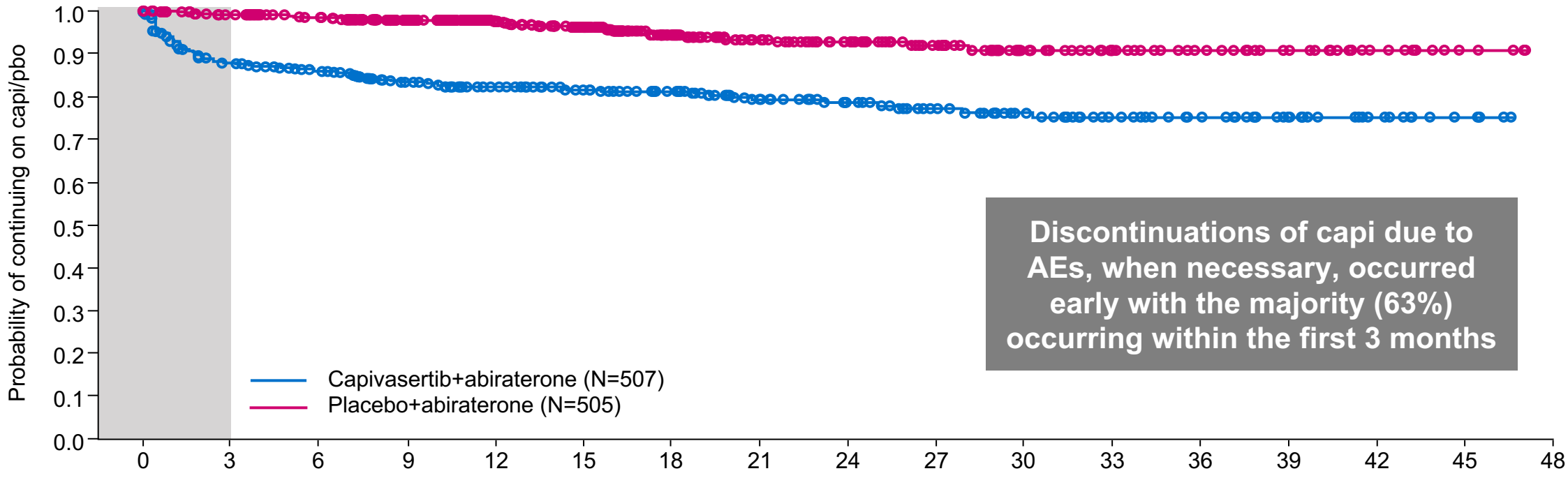
Number of patients at risk

Capi+abi	507	179	143	114	81	65	55	42	34	31	17	8	7	4	4	1	0
Pbo+abi	505	214	180	142	111	77	68	51	39	32	23	17	14	10	5	0	0

Time to decrease in ≥3 points in FACT–P PWB is defined as the time from randomization until date of first confirmed clinically meaningful decrease before receiving subsequent anticancer therapy. If the decrease occurred after two or more missed PRO assessments, patients were censored at the time of last PRO assessment prior to the two missed visits. Circles represent censored observations

abi, abiraterone; capi, capivasertib; CI, confidence interval; FACT–P, Functional Assessment of Caner Therapy–Prostate Cancer; HR, hazard ratio; NC, non-calculable; pbo, placebo; PRO, patient-reported outcome

CAPItello-281: Discontinuations due to AEs



Discontinuations of capi due to AEs, when necessary, occurred early with the majority (63%) occurring within the first 3 months

Number of patients at risk

Capi+abi	507	403	369	321	271	230	191	147	113	91	70	53	35	24	13	4	0
Pbo+abi	505	473	435	376	309	253	197	158	124	97	72	57	38	25	13	4	0

*AEs leading to death, considered by the investigator to be related to capi/pbo were reported in 6 (1.2%) and 1 (0.2%) patient(s), respectively. Patients not known to have discontinued capi/pbo because of AEs were censored at the earliest of the following: death date, end of study date, DCO date and when discontinuation of capi/pbo was not due to AEs. AE, adverse event; capi, capivasertib; DCO, data cutoff; pbo, placebo; SAE, serious AE

Conclusion

- PTEN loss associated with poor outcomes in Prostate cancer
- Targeting both AR and Akt pathways makes sense
- ADT + Abiraterone + Capivasertib:
  - Improves rPFS
  - Benefit more profound if deep PTEN deficiency
  - OS data pending
- Main AEs: Diarrhea, Hyperglycemia, rash

Discussion Questions

49 yo man with PTEN-deficient prostate cancer, high-volume de novo disease. Has diabetes with HbA1c 7.5.

What is the optimal front-line treatment for patients with high-volume, PTEN-deficient mHSPC and diabetes — would you opt for ADT + APRI + docetaxel or would you consider a capivasertib combination? What HbA1c level is needed to treat with capivasertib?

What is the best type of assay to detect PTEN deficiency? Is it important to obtain a quantitative assay?

If capivasertib is approved in prostate cancer, in which situations would you use it in hormone-sensitive metastatic disease?

RTP
RESEARCH
TO PRACTICE

Discussion Questions (Continued)

49 yo man with PTEN-deficient prostate cancer, high-volume de novo disease. Has diabetes with HbA1c 7.5.

Is chemotherapy more or less effective in patients with PTEN deficiency?

What would you do for a patient with PTEN deficiency with suboptimal response to ADT/ARPI/docetaxel? Layer on capivasertib? Switch to capivasertib/abiraterone/ADT?

RTP
RESEARCH
TO PRACTICE

Discussion Questions

71 y/o man, de novo high-volume mHSPC (Gleason 8, >10 bone metastases).
Atrial fibrillation (on apixaban), CKD Stage 3a. PTEN deficiency.
Receives ADT + darolutamide + docetaxel with progression at 2 years.
Now receiving lutetium (4 of 6 cycles completed).
Mixed response: Bone/nodal disease improved, but a PSMA-negative liver metastasis is growing.

Is there a role for AKT inhibition in this patient?

What's the best way to follow patients with PTEN deficiency? Do they require more frequent imaging?

How do you recommend prevention and management of side effects of capivasertib?

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RESEARCH
TO PRACTICE

Agenda

Module 1: Evolving Management of Nonmetastatic Hormone-Sensitive Prostate Cancer (HSPC) — Dr Antonarakis

Module 2: Current and Future Hormonal Treatment for Metastatic HSPC (mHSPC) — Dr Aggarwal

Module 3: Current and Future Role of Regimens Combining PARP Inhibitors and AR Pathway Inhibitors in Metastatic Prostate Cancer — Dr Abida

Module 4: Emerging Role of AKT Inhibition for Patients with mHSPC — Dr Fizazi

Module 5: Current and Future Use of Radiopharmaceuticals in Metastatic Prostate Cancer — Dr McKay

RTP RESEARCH TO PRACTICE

UC San Diego Health

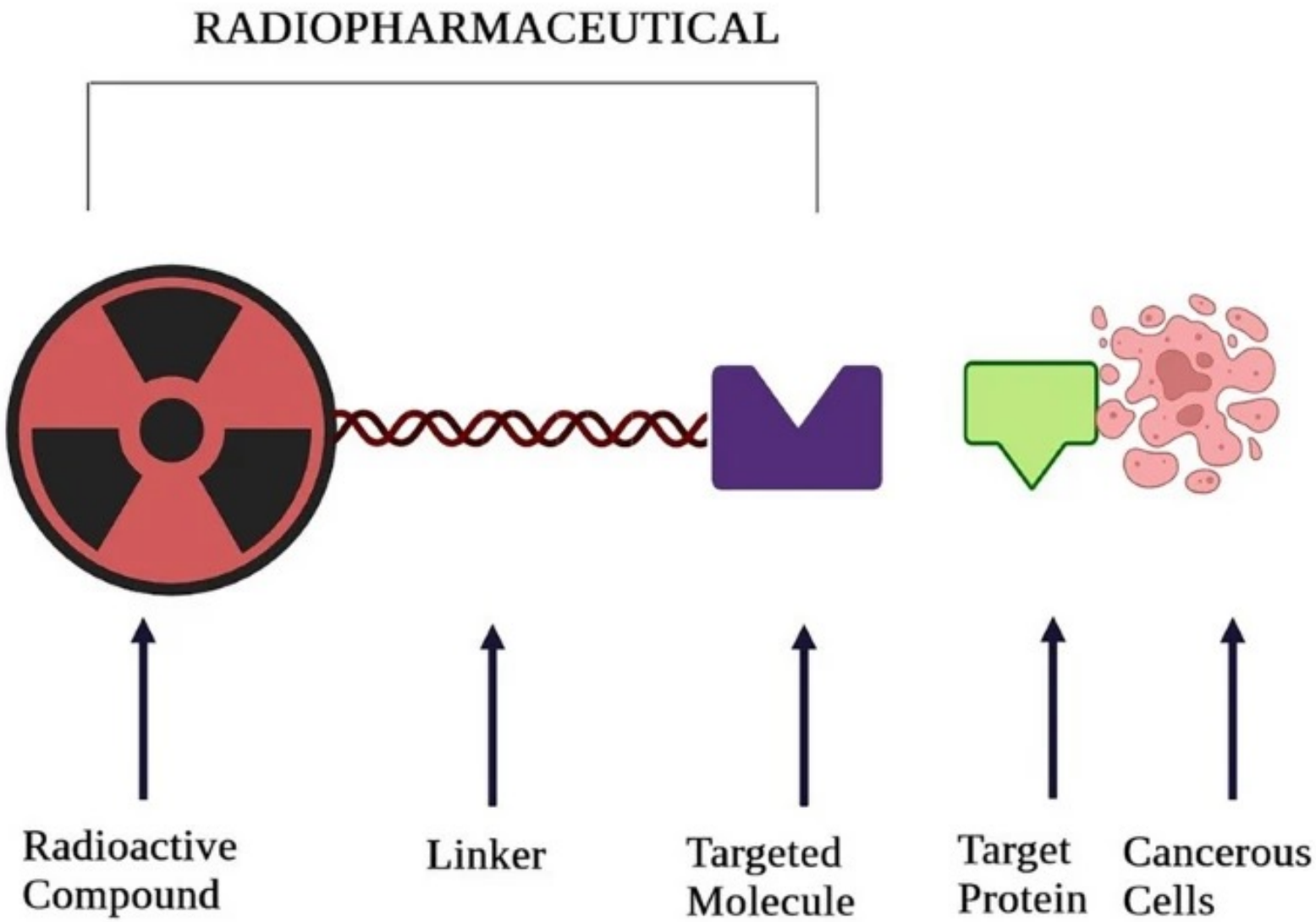
Current and Future Use of Radiopharmaceuticals in Metastatic Prostate Cancer

Rana R. McKay, MD, FASCO

Professor of Medicine, Urology, and Radiation Medicine

Moores Cancer Center, University of California San Diego

What Are Radiopharmaceuticals?



Dhoundiyal S et al. Eur J Med Res 2024

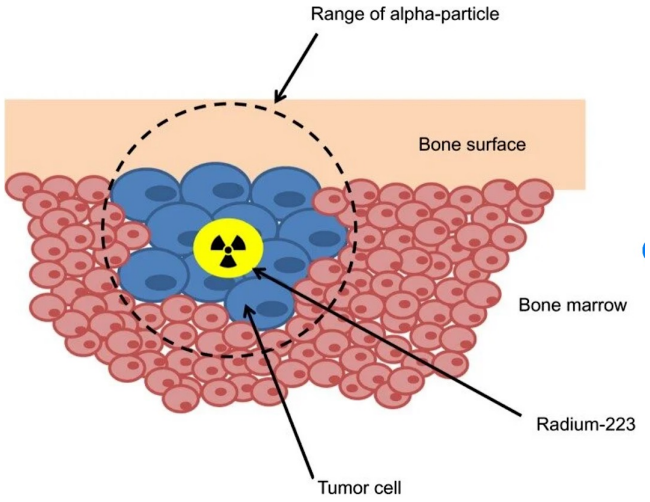
Radium-223 – The First Life Prolonging Radiopharmaceutical

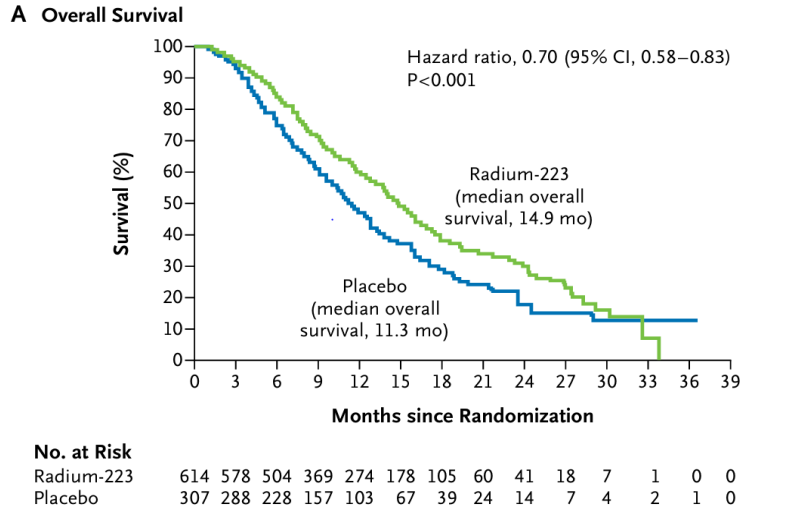
The NEW ENGLAND JOURNAL of MEDICINE

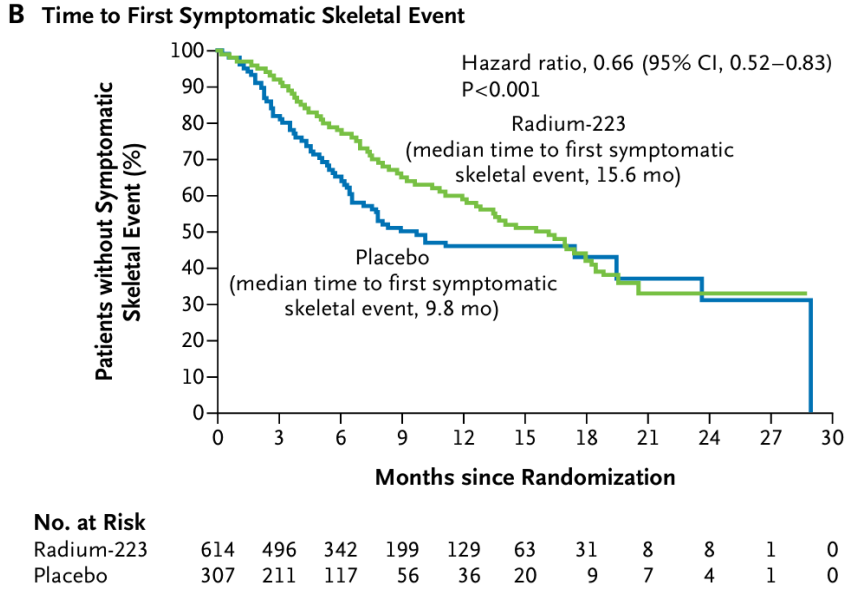
ESTABLISHED IN 1812 JULY 18, 2013 VOL. 369 NO. 3

Alpha Emitter Radium-223 and Survival in Metastatic Prostate Cancer

C. Parker, S. Nilsson, D. Heinrich, S.I. Helle, J.M. O'Sullivan, S.D. Fosså, A. Chodacki, P. Wiechno, J. Logue, M. Seke, A. Widmark, D.C. Johannessen, P. Hoskin, D. Bottomley, N.D. James, A. Solberg, I. Syndikus, J. Kliment, S. Wedel, S. Boehmer, M. Dall'Oglio, L. Franzén, R. Coleman, N.J. Vogelzang, C.G. O'Bryan-Tear, K. Staudacher, J. Garcia-Vargas, M. Shan, Ø.S. Bruland, and O. Sartor, for the ALSYMPCA Investigators*

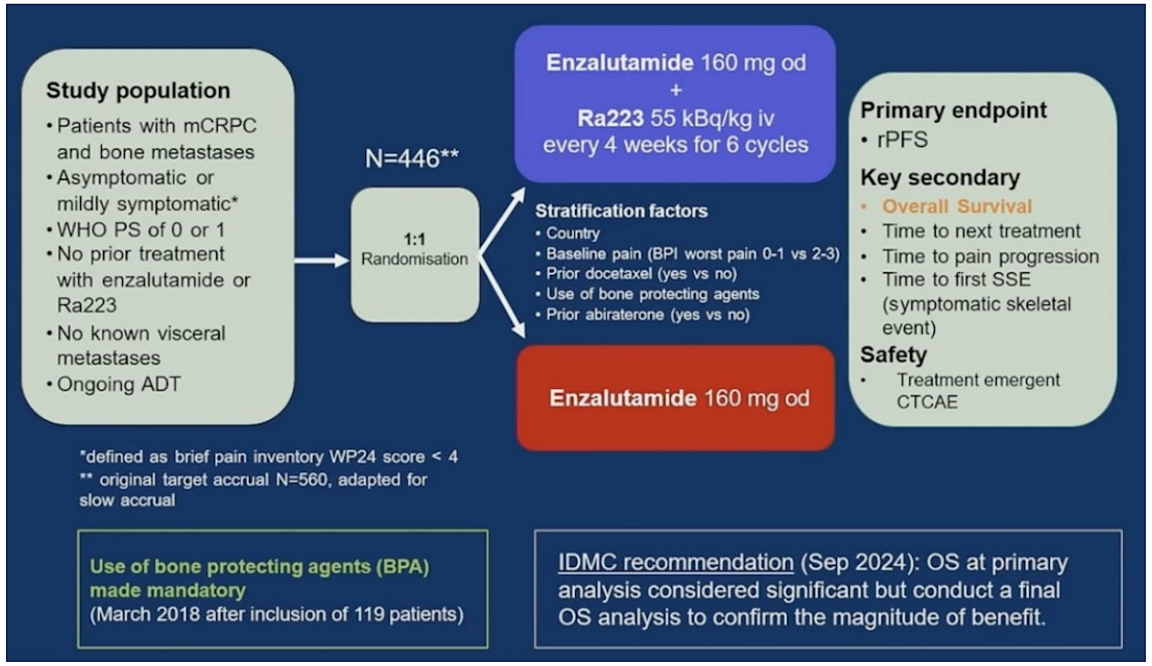






Mukherji D et al. Ther Clin Risk Manag. 2014; Parker C et al. New Eng J Med. 2013

PEACE 3 – Enzalutamide +/- Radium-223



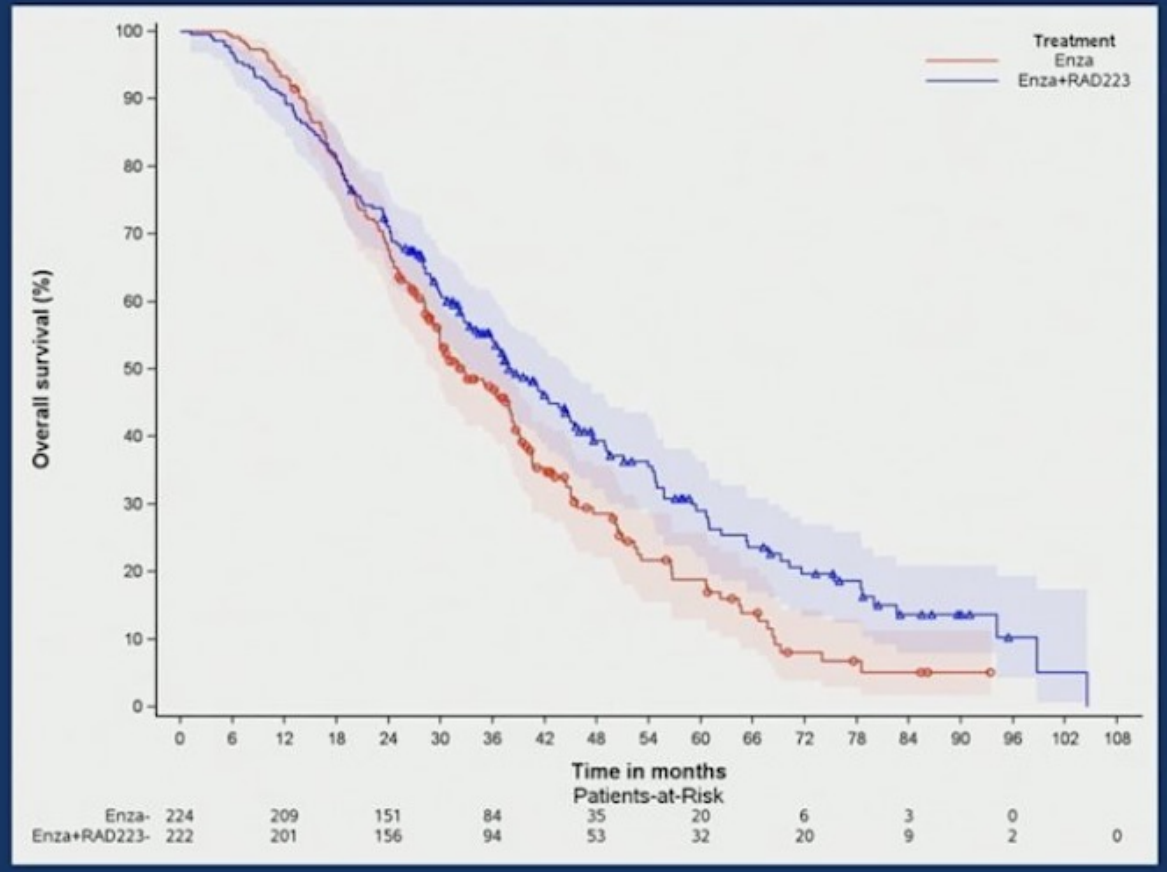
	Enza+Ra223 (N=222)	Enza (N=224)
	N (%)	N (%)
Age, Median (range) years	70.0 (43.0 - 90.0)	70.0 (47.0 - 90.0)
PSA, Median (Q25-Q75) ng/mL	24.5 (7.8-68.8) N = 190	21.2 (7.9-53.1) N = 191
WHO Performance status 0	153 (69)	154 (69)
Prior docetaxel(1)	67 (30.2)	66 (30)
Prior abiraterone(1)	4 (2)	7 (3)
Bone lesions(2)		
<10	109 (49)	105 (47)
≥10	93 (42)	99 (44)
Missing or diffuse lesions	20 (9)	20 (9)
Alkaline phosphatase		
≤ULN	127 (57)	109 (49)
>ULN	82 (37)	109 (49)
Missing	13 (6)	6 (3)
Extra-skeletal disease at baseline	77 (35)	73 (33)

(1) Prior docetaxel or abiraterone was allowed for mHSPC

Gallardo E et al. ASCO GU 2026

PEACE 3 – Enzalutamide +/- Radium-223 – FINAL OS

Final OS results



	Event/ Total	Median (95%CI) (months)[1]	Hazard Ratio (95% CI)[2]	1-sided P-value
Treatment				Logrank: 0.0096[3] Permutation:0 .0105
Enza	165/224	32.62 (29.31-38.24)	0.76 (0.60-0.96)	
Enza+Ra223	152/222	38.21 (33.08-44.75)		

[1]Kaplan-Meier method; [2]Cox model; [3] Stratified Logrank test

	Enza+Ra223 (N=222)	Enza (N=224)
	(95% CI)	(95% CI)
6 mths	96.8 (93.5-98.5%)	99.1 (96.5-99.8%)
12 mths	90.5 (85.9-93.7%)	92.9 (88.6-95.6%)
18 mths	81.1 (75.3-85.6%)	80.8 (74.9-85.3%)
24 mths	71.1 (64.7-76.6%)	67.7 (61.2-73.4%)
36 mths	54.2 (47.1-60.6%)	47.4 (40.6-54.0%)

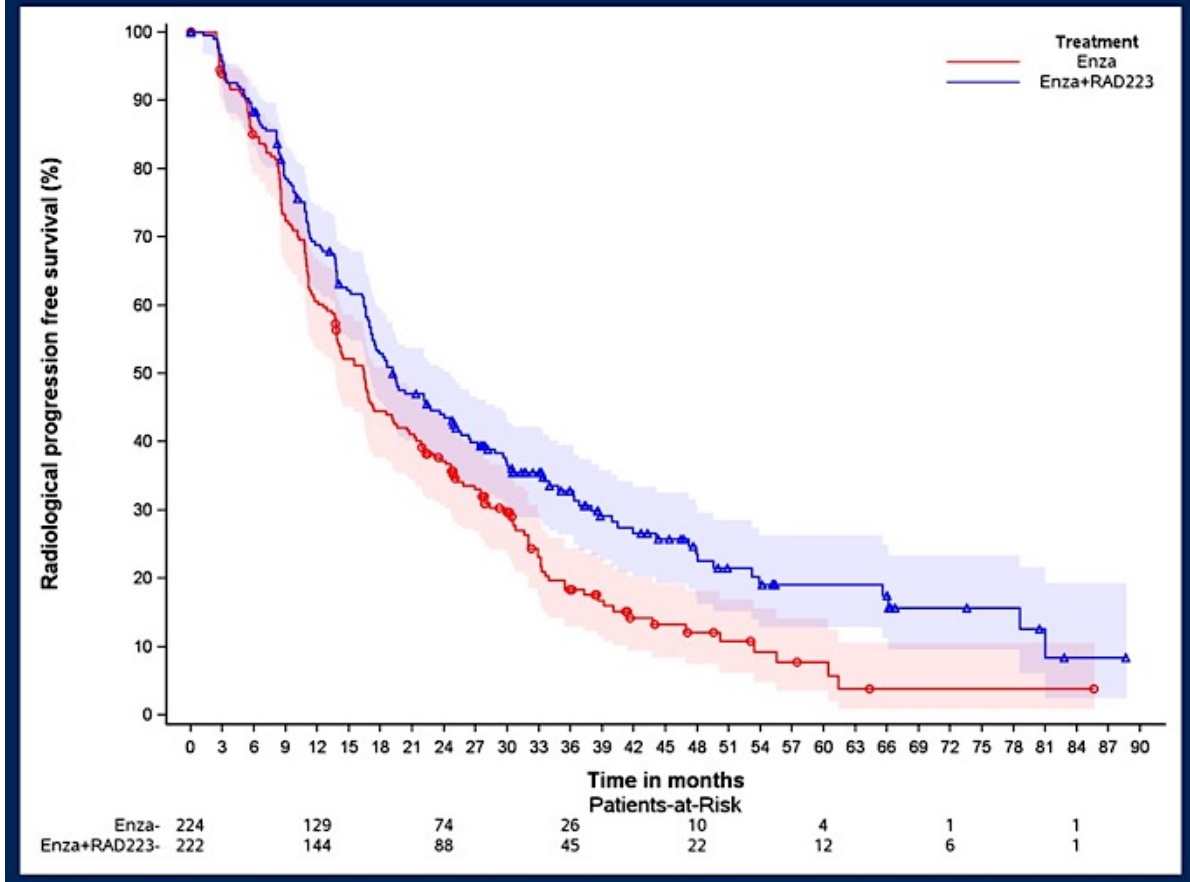
Preset level of significance at final analysis: <0.0248
Crossing of the curves (still) present at month 18
Only 3 patients censored < 24 months

Database locked on 12 January 2026 (cut off date: 01 May 2025)

Gallardo E et al. ASCO GU 2026

PEACE 3 – Enzalutamide +/- Radium-223 – FINAL rPFS

Final rPFS results



	Event/ Total	Median (95% CI) (months)[1]	Hazard Ratio (95% CI)[2]
Treatment			
Enza	176/224	**16.43** (13.77-19.15)	Reference
Enza+Ra223	156/222	**19.19** (16.92-24.57)	**0.71** **(0.57-0.89)**

[1]Kaplan-Meier method; [2]Cox model

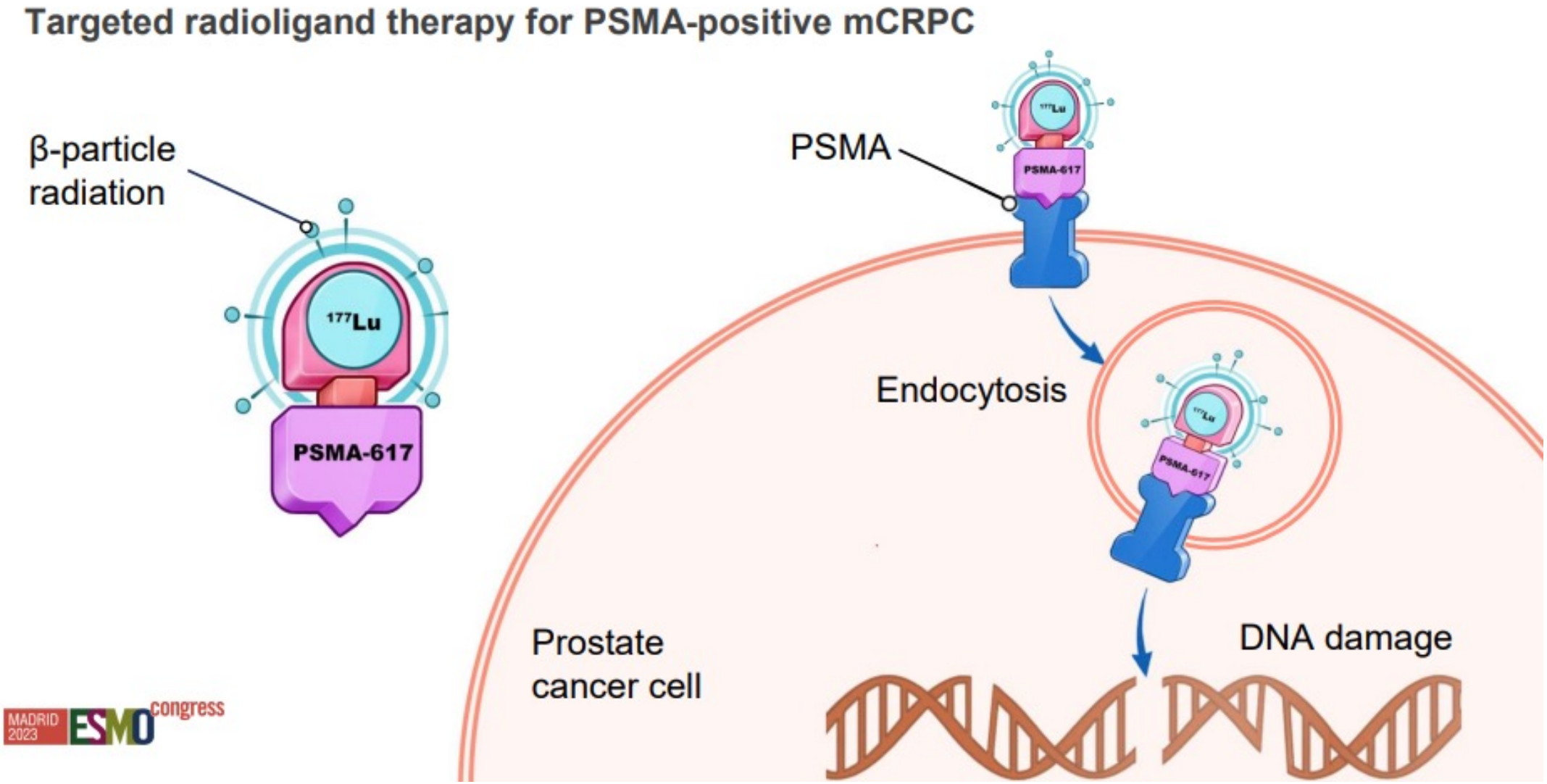
	Enza+RAD223 (N=222)	Enza (N=224)
	(95% CI)	(95% CI)
6 months	88.4 (83.3-92.0%)	84.6 (79.1-88.8%)
12 months	68.9 (62.2-74.7%)	60.6 (53.8-66.8%)
18 months	52.9 (45.9-59.4%)	44.5 (37.7-51.0%)
24 months	44.1 (37.2-50.7%)	37.2 (30.7-43.7%)

rPFS results confirmed with longer follow-up and appear little changed from primary analysis.

Database locked on 12 January 2026 (cut off date: 01 May 2025)

Gallardo E et al. ASCO GU 2026

^{177}Lu-PSMA-617



Sartor O et al. ESMO 2023

VISION – ^{177}Lu-PSMA-617 + SoC vs SoC

Eligible patients
- Previous treatment with both
  - ≥ 1 androgen receptor pathway inhibitor
  - 1 or 2 taxane regimens
- Protocol-permitted standard of care (SOC) planned before randomization
  - Excluding chemotherapy immunotherapy, radium-223, investigational drugs
- ECOG performance status 0–2
- Life expectancy > 6 months
- PSMA-positive mCRPC on PET/CT with ^{68}Ga-PSMA-11

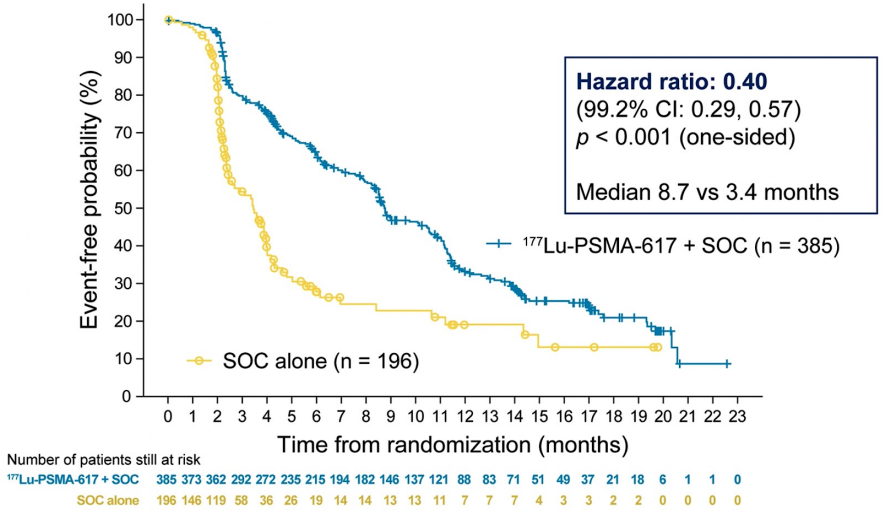
2:1

Protocol-permitted SOC + ^{177}Lu-PSMA-617
7.4 GBq (200 mCi) every 6 weeks
4 cycles, increasable to 6

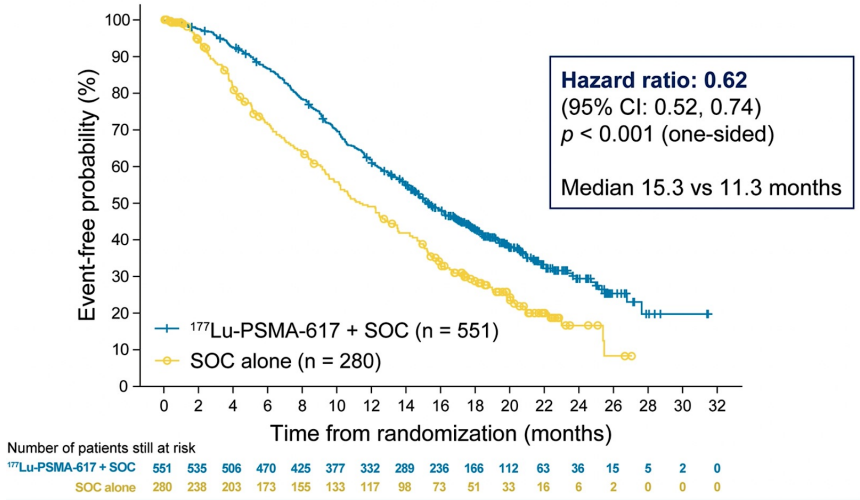
Protocol-permitted SOC alone

Treatment → Follow-up → Final analysis

- Randomization stratified by
  - ECOG status (0–1 or 2)
  - LDH (high or low)
  - Liver metastases (yes or no)
  - Androgen receptor pathway inhibitors in SOC (yes or no)
- CT/MRI/bone scans
  - Every 8 weeks (treatment)
  - Every 12 weeks (follow-up)
  - Blinded independent central review



	rPFS analysis set (n = 581)		All randomized (N = 831)	
	^{177}Lu-PSMA-617 + SOC (n = 385)	SOC alone (n = 196)	^{177}Lu-PSMA-617 + SOC (n = 551)	SOC alone (n = 280)
Age, median (range)	71.0 (52–94)	72.0 (51–89)	70.0 (48–94)	71.5 (40–89)
Race, n (%)				
White	336 (87.3)	166 (84.7)	486 (88.2)	235 (83.9)
Black/African-American	29 (7.5)	14 (7.1)	34 (6.2)	21 (7.5)
Asian	6 (1.6)	9 (4.6)	9 (1.6)	11 (3.9)
ECOG status, n (%)				
0 or 1	352 (91.4)	179 (91.3)	510 (92.6)	258 (92.1)
2	33 (8.6)	17 (8.7)	41 (7.4)	22 (7.9)
Site of disease, n (%)				
Lung	35 (9.1)	20 (10.2)	49 (8.9)	28 (10.0)
Liver	47 (12.2)	26 (13.3)	63 (11.4)	38 (13.6)
Lymph node	193 (50.1)	99 (50.5)	274 (49.7)	141 (50.4)
Bone	351 (91.2)	179 (91.3)	504 (91.5)	256 (91.4)

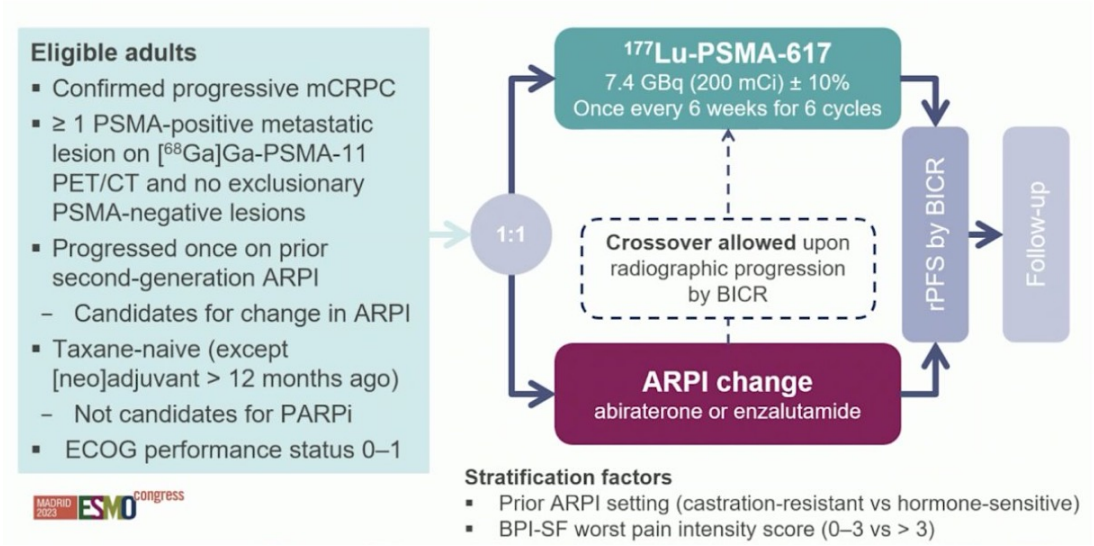


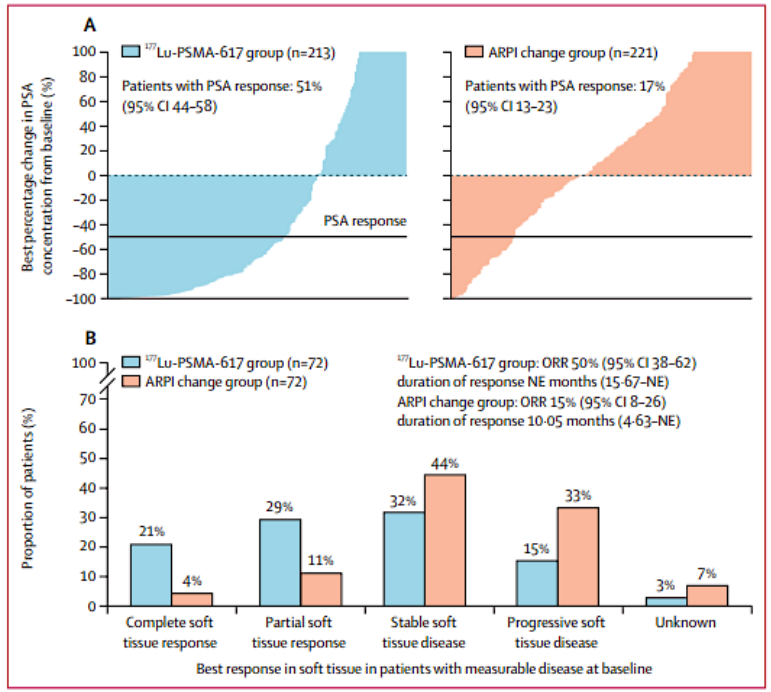
Morris MJ et al. ASCO 2021; Sartor O et al. NEJM. 2021;385:1091.

PSMAFore – ^{177}Lu-PSMA-617 vs ARPI

Eligible adults

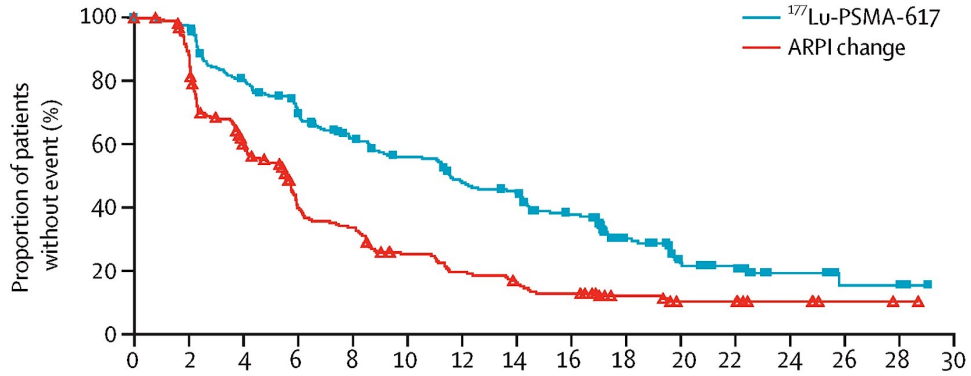
- Confirmed progressive mCRPC
- ≥ 1 PSMA-positive metastatic lesion on [^{68}Ga]Ga-PSMA-11 PET/CT and no exclusionary PSMA-negative lesions
- Progressed once on prior second-generation ARPI
  - Candidates for change in ARPI
- Taxane-naive (except [neo]adjuvant > 12 months ago)
  - Not candidates for PARPi
- ECOG performance status 0–1





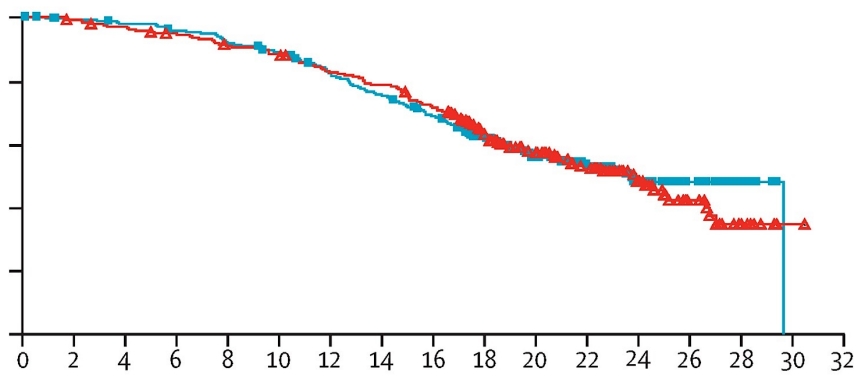
A Radiographic progression-free survival

^{177}Lu-PSMA-617 group: median 11·60 months (95% CI 9·30–14·19), 154 events
ARPI change group: median 5·59 months (95% CI 4·21–5·95), 180 events
HR 0·49 (95% CI 0·39–0·61)



B Overall survival (intention-to-treat analysis)

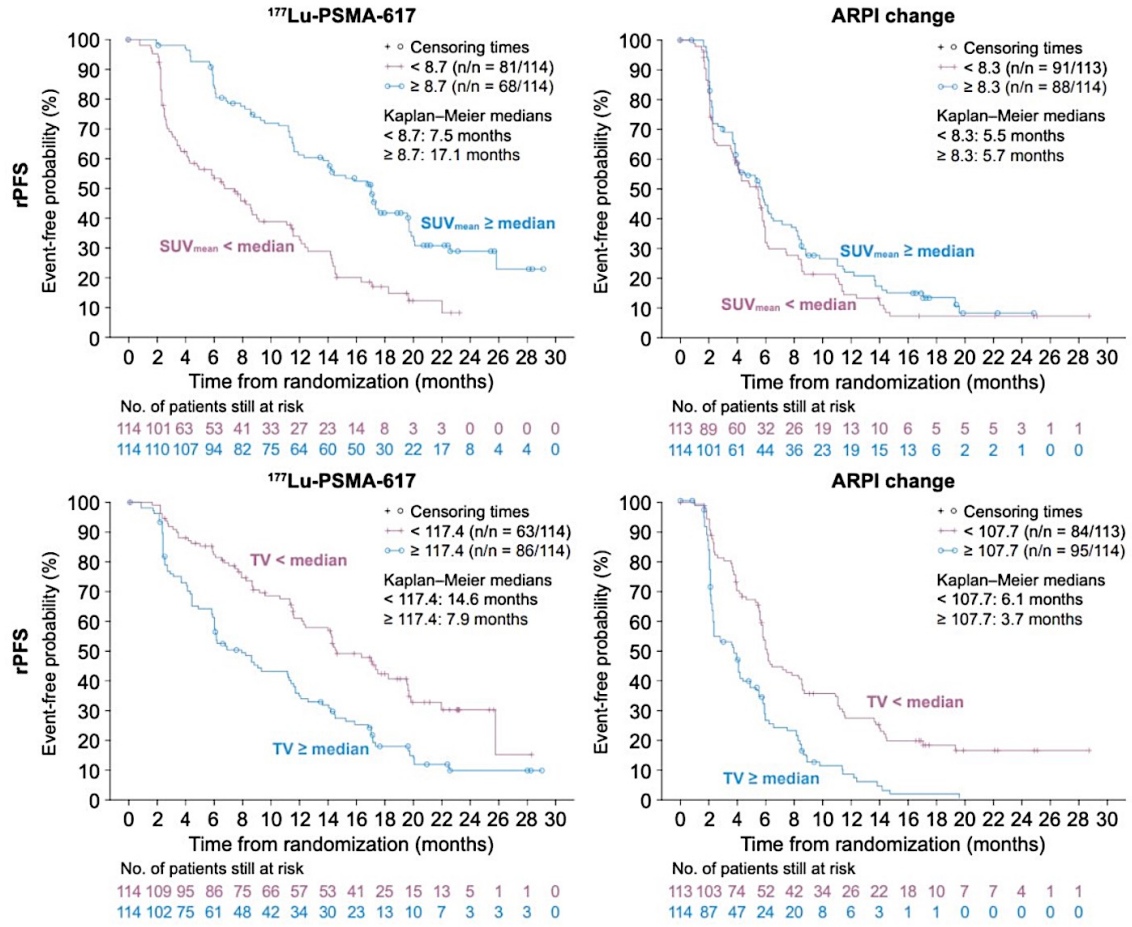
^{177}Lu-PSMA-617 group: median 23·66 months (95% CI 19·75–NE), 104 events
ARPI change group: 23·85 months (20·60–26·55), 112 events
HR 0·98 (95% CI 0·75–1·28), p=0·44



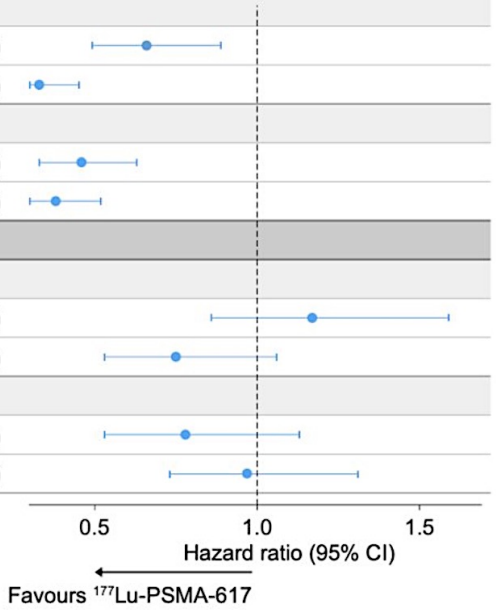
Sartor O et al. ESMO 2023; Morris MJ et al. Lancet. 2024

PSMAFore – Imaging Based Predictors of Response

Figure 2. Kaplan–Meier analysis for rPFS by median SUV_{mean} and PSMA+ TV



	177Lu-PSMA-617 N = 228		ARPI change N = 227		
	Events (n/n)	Median, months	Events (n/n)	Median, months	HR (95% CI)
rPFS					
SUV_{mean}					
< median[a]	78/110	6.7	94/117	5.5	0.66 (0.49, 0.89)
≥ median[a]	71/118	17.0	85/110	5.8	0.33 (0.23, 0.45)
PSMA+ TV					
< median[b]	62/112	14.6	86/115	6.0	0.46 (0.33, 0.63)
≥ median[b]	87/116	7.9	93/112	3.7	0.38 (0.27, 0.52)
OS (intent to treat, unadjusted for crossover)[c]					
SUV_{mean}					
< median[a]	76/110	17.0	88/117	19.6	1.17 (0.86, 1.59)
≥ median[a]	61/118	31.5	65/110	26.7	0.75 (0.53, 1.06)
PSMA+ TV					
< median[b]	48/112	35.9	63/115	28.6	0.78 (0.53, 1.13)
≥ median[b]	89/116	16.3	90/112	18.1	0.97 (0.73, 1.31)



Herrmann K et al. ESMO. 2025

ENZA-P – Enzalutamide +/- ^{177}Lu-PSMA-617

ENZA-p Schema

Eligibility

mCRPC with PSA rising and >5ng/mL
No chemotherapy for mCRPC
≥2 risk features for early enzalutamide failure
Positive ^{68}Ga PSMA PET/CT

R

1:1

Enzalutamide 160 mg

Enzalutamide 160 mg
+ [^{177}Lu]Lu- PSMA-617 7.5 GBq
2-4 doses

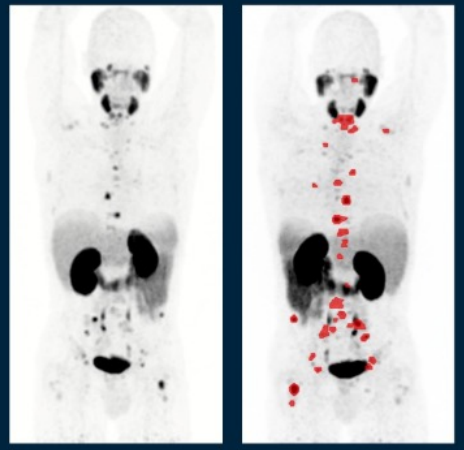
Stratification

Study Site
Volume of disease (>20 vs ≤20)
Early docetaxel for hormone-sensitive disease
Prior treatment with abiraterone

Objectives

PSA-PFS (primary endpoint)
Overall survival
Health-related Quality of Life
Radiographic PFS
PSA response rate
Pain response and PFS
Clinical PFS
Adverse events
Health economic analyses
Translational/correlative

Screening Criteria



PSMA-PET screening criteria

SUV_{max} ≥15 at one site AND ≥10 at all measurable sites
Mismatch on diagnostic CT not an exclusion

Risk Factors for Early Treatment Failure on Enzalutamide

LDH ≥ULN
ALP ≥ULN
Albumin <35g/L
De novo metastatic disease at diagnosis
<3 Years since initial diagnosis
>5 Bone metastases
Visceral metastases
PSA doubling time <84 days
Pain requiring opiates >14 days
Prior abiraterone

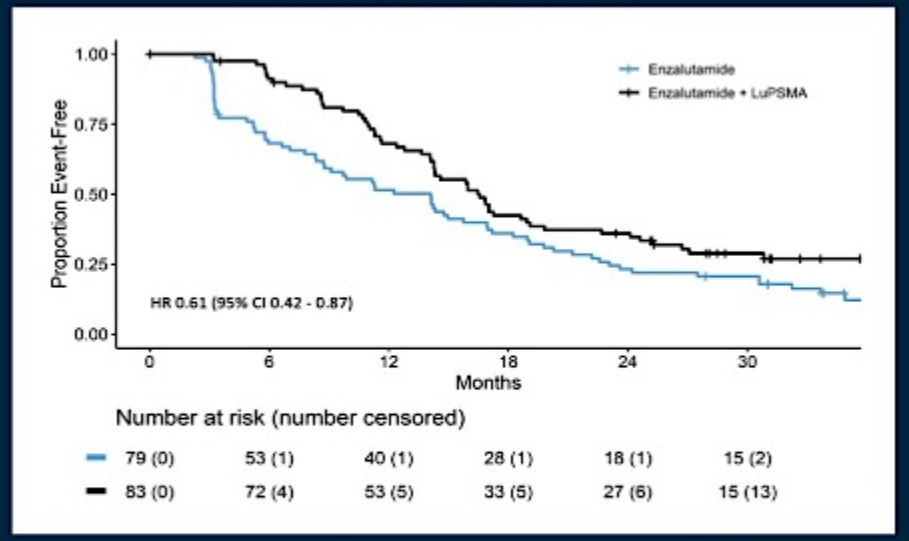
Imaging screen failure rate 18%

Emmett L et al, GU ASCO, 2025

ENZA-P – Enzalutamide +/- ^{177}Lu-PSMA-617

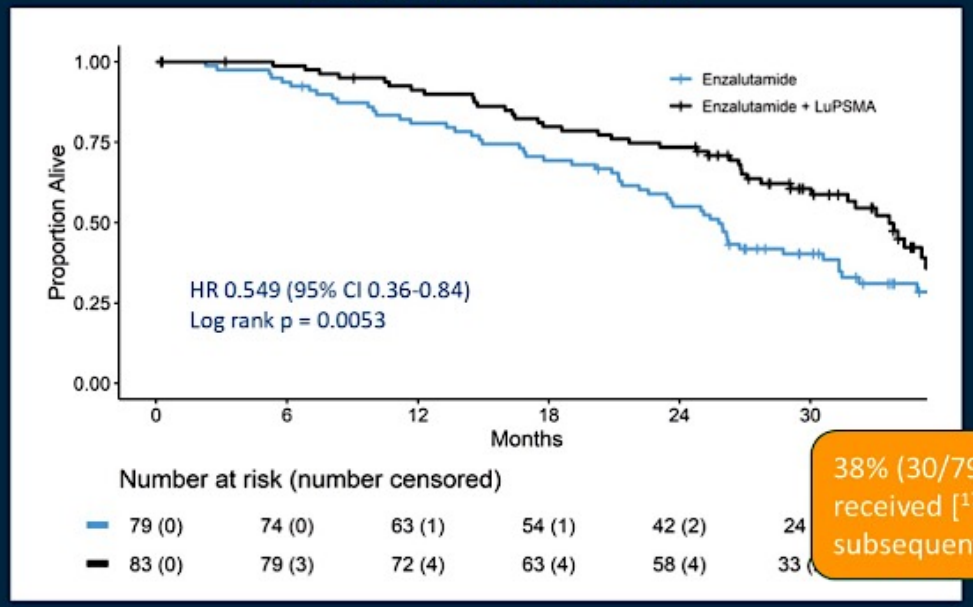
R-PFS

HR 0.61 (95% CI 0.42-0.87)



R-PFS	Participants	Events	Censored	Median Months
Enzalutamide	79	69	10	14
Enzalutamide+[^{177}Lu]LuPSMA617	83	56	27	17

Overall Survival



38% (30/79) on enzalutamide-alone received [^{177}Lu]Lu-PSMA 617 as subsequent treatment off protocol

Overall Survival	Participants	Events	Censored	Median Months
Enzalutamide	79	53	26	26 (C195% 23-31)
Enzalutamide + Lu-PSMA 617	83	43	40	34 (CI95% 30-37)

Emmett L et al, GU ASCO, 2025; Emmett L et al. Lancet Oncol. 2025

CCTG PR.21 (PLUDO) – ^{177}Lu-PSMA-617 vs Docetaxel

Study Design

Open label randomized phase II with cross-over

Key Eligibility
- Chemotherapy-naïve mCRPC progressing after ARPI
- PSMA PET positive (uptake >liver)
  - Excluded if >50% of extra-osseous lesions or >5 cm soft tissue lesion were PSMA negative
- ANC ≥ 1.5 x 10^9/L
- Platelet count ≥ 100 x 10^9/L
- Hemoglobin ≥ 90 g/L
- CrCl ≥ 30 ml/min
- Bilirubin < 1.5 x ULN
- ALT < 3.5 x ULN (<5 x UN for liver metastases)
- ECOG PS of 0 - 2
- Prior docetaxel for mHSPC permitted if ≥12 months prior

Randomize 1:1

- ^{177}Lu-PSMA-617 7.4 GBq (+/- 10%) IV Every 6 weeks x 6 cycles → Radiographic Progression (rPFS1) → Docetaxel 75 mg/m² IV Every 3 weeks x 12 cycles → Radiographic Progression (rPFS2)
- Docetaxel 75 mg/m² IV Every 3 weeks x 12 cycles → Radiographic Progression (rPFS1) → ^{177}Lu-PSMA-617 7.4 GBq (+/- 10%) IV Every 6 weeks x 6 cycles → Radiographic Progression (rPFS2)

Stratification factors:
- ECOG PS (0-1 vs 2)
- LDH (>ULN, yes vs no)
- Visceral metastases (yes vs no)
- Prior docetaxel for mHSPC (yes vs no)

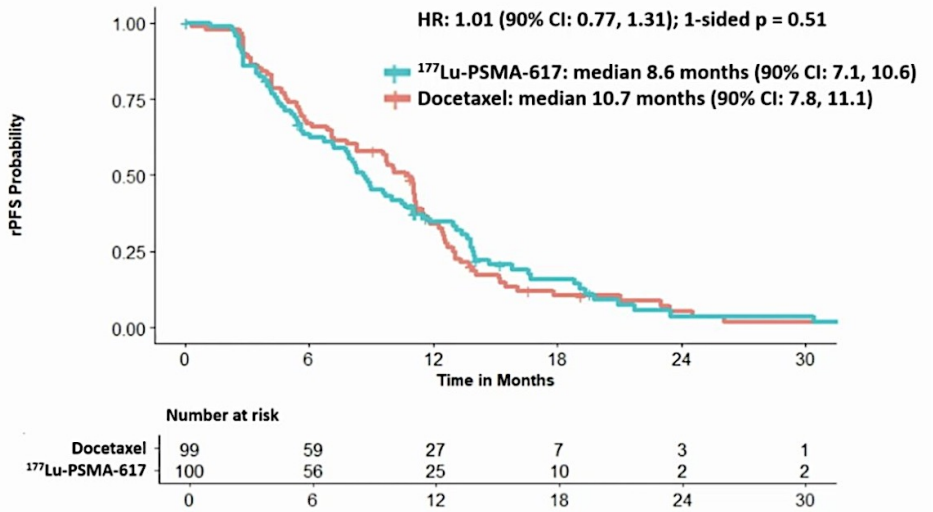
Primary Objective
- Radiographic Progression Free Survival (rPFS1)

Additional Objectives
- Overall survival
- rPFS2
- PSA decline
- Adverse events
- PRO QoL
- Correlative studies

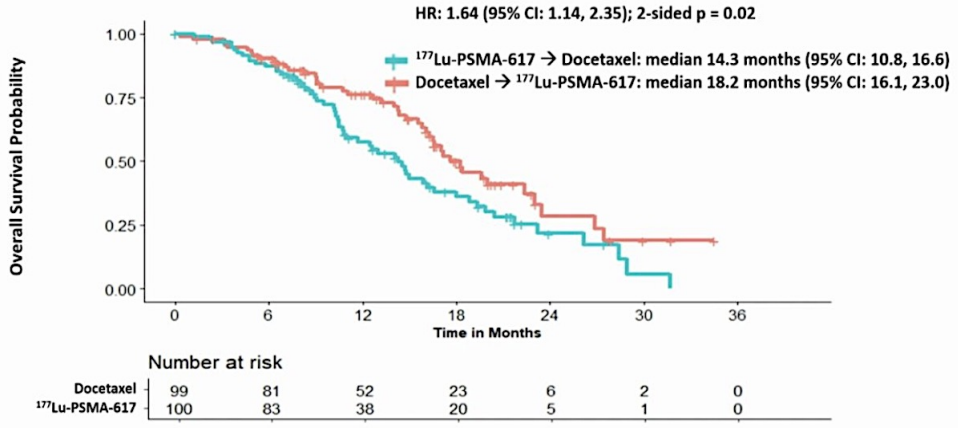
Baseline Characteristics

Characteristic	^{177}LU-PSMA-617 N = 100	Docetaxel n = 99	Total Population N = 199
Age, years, median (range)	70 (48-86)	73 (50-89)	70 (48-89)
ECOG performance status 0/1/2 (%)	53/44/4	53/44/4	53/44/4
Race (%)			
White	94	89	92
Asian	3	7	5
Black	1	0	1
Other	2	4	2
Type of metastasis (%)			
Visceral	22	20	21
Bone	81	86	83
Elevated LDH (%)			
Yes	25	27	26
No	75	73	74
PSA value, ng/ml, median (range)	24 (0.08-716.3)	18 (0.4-2789)	19.6 (0.08-2789)
Hemoglobin, g/L, median (range)	130 (92-155)	128 (93-161)	128 (92-161)
Prior docetaxel for mHSPC (%)	14	13	14
PSMA PET SUV_{mean}, median (range)	8.7 (3.9-26.2)	9.2 (4.1-32.8)	9.1 (3.9-32.7)
PSMA PET meeting VISION criteria (%)	98	96	97

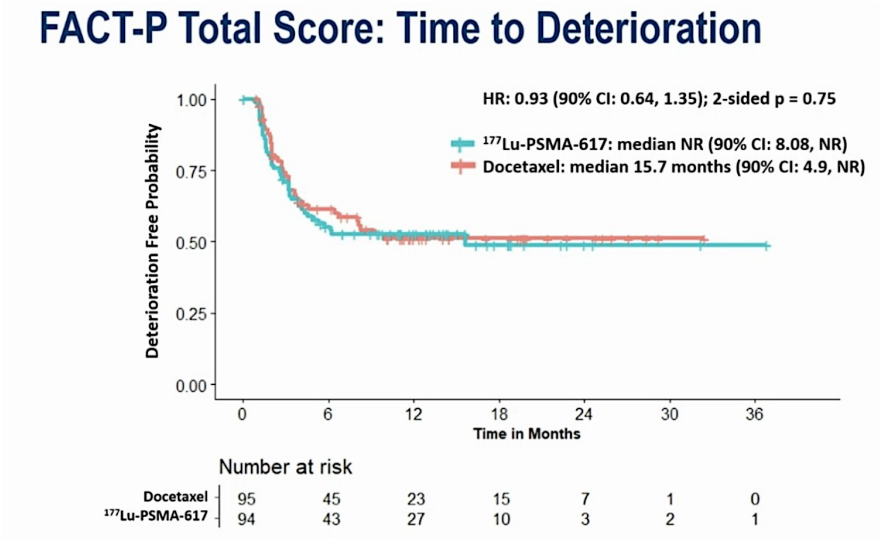
Primary Endpoint: rPFS (ITT)

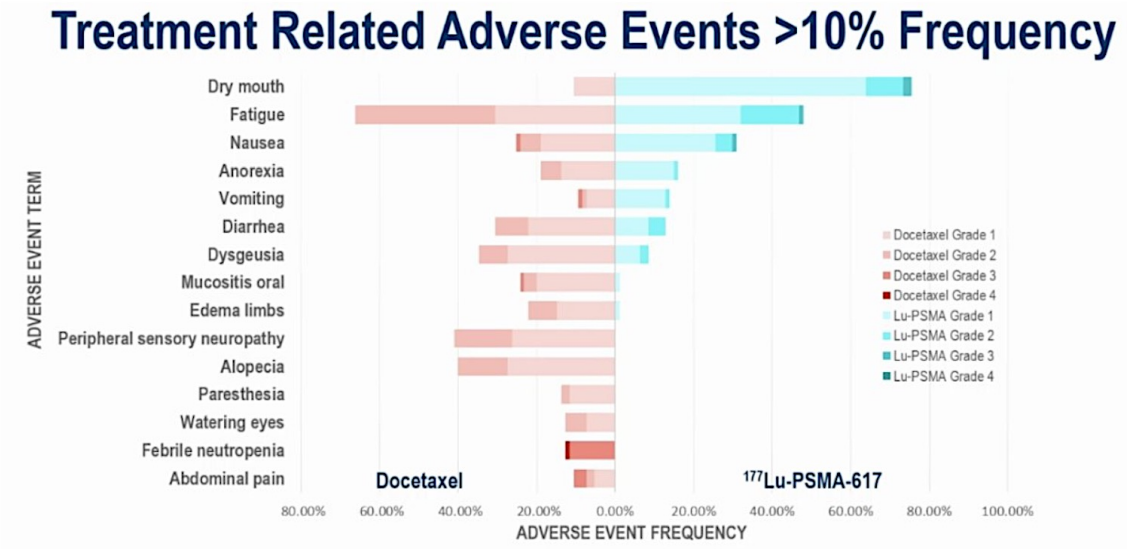


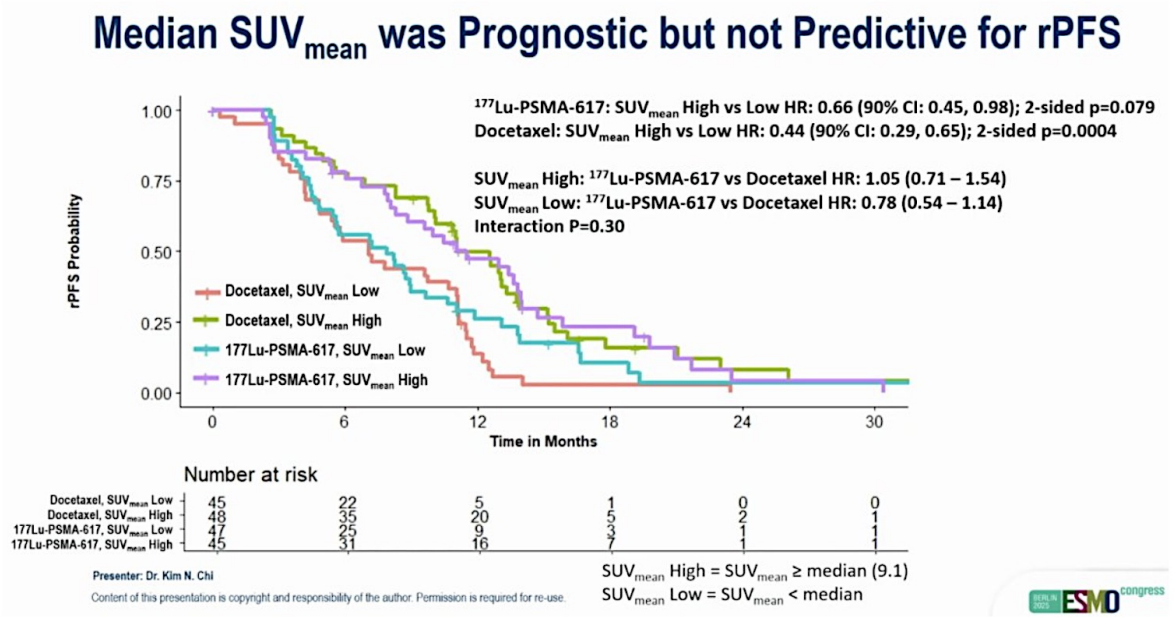
Overall Survival



CCTG PR.21 (PLUDO) – ^{177}Lu-PSMA-617 vs Docetaxel

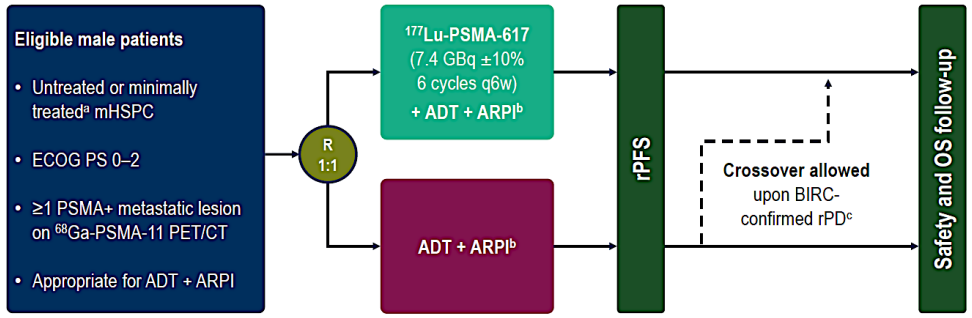






PSMAddition – ^{177}Lu-PSMA-617 + ARPI vs. ARPI in mHSPC

PSMAddition: randomized phase 3 trial of ^{177}Lu-PSMA-617 in mHSPC



Stratification factors
- Disease volume (high/low) – per CHAARTED criteria[1]
- Age ≥ 70 years (yes/no)
- Previous or planned treatment of primary tumour by radiation or prostatectomy (yes/no)

Follow-up periods
- rPFS: until event in all patients
- Safety: 30 days then 24 and 48 weeks after treatment discontinuation
- OS: every 90 days after last contact

[a] ADT in the neo-/adjuvant setting and/or up to 45 days of ADT/ARPI for metastatic disease was allowed before study entry | [b] Any ARPI with one switch allowed | [c] ADT/ARPI not mandatory after crossover
ADT, androgen deprivation therapy; BIRC, blinded independent review committee; ECOG PS, Eastern Cooperative Oncology Group performance status; OS, overall survival; PET/CT, positron-emission tomography/computed tomography; q6w, every 6 weeks; rPD, radiographic disease progression; rPFS, radiographic progression-free survival
1. Sweeney CT *et al. N Engl J Med* 2015;373:737–46

Baseline characteristics were balanced between arms

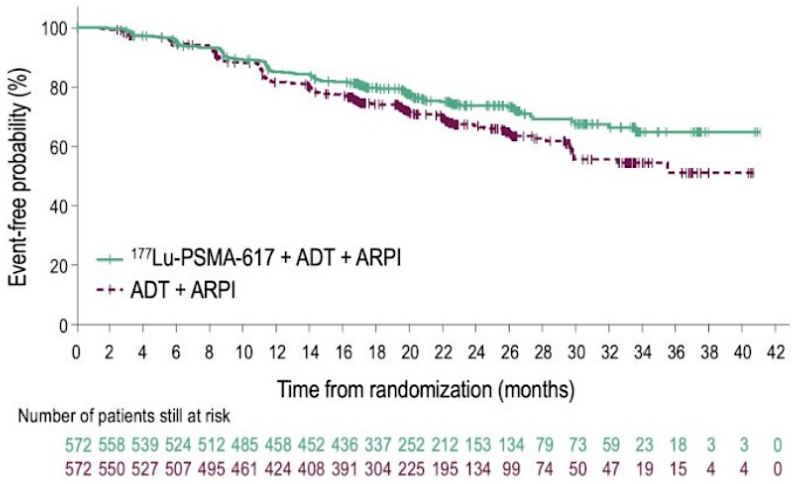
Data cut-off: 15 January

	^{177}Lu-PSMA-617 + ADT + ARPI (N = 572)	ADT + ARPI (N = 572)	Overall (N = 1144)
Age, years – median (range)	68.0 (38–91)	68.0 (36–90)	68.0 (36–91)
≥70 years – n (%)	246 (43.0)	246 (43.0)	492 (43.0)
ECOG performance status – n (%)			
0	397 (69.4)	407 (71.2)	804 (70.3)
1	174 (30.4)	155 (27.1)	329 (28.8)
2	1 (0.2)	7 (1.2)	8 (0.7)
Site of disease – n (%)			
Soft tissue	342 (59.8)	338 (59.1)	680 (59.4)
Bone	522 (91.3)	521 (91.1)	1043 (91.2)
Visceral	225 (39.3)	238 (41.6)	463 (40.5)
PSA level, ng/mL – median (interquartile range)	12.06 (3.16–53.24)	11.64 (2.83–44.15)	11.91 (3.01–50.34)
Gleason score 8–10 (grade group 4–5) – n (%)	389 (68.0)	406 (71.0)	795 (69.5)
High tumour volume[a] – n (%)	389 (68.0)	390 (68.2)	779 (68.1)
De novo mHSPC[b] – n (%)	298 (52.1)	274 (47.9)	572 (50.0)

Tagawa ST et al, ESMO 2025

PSMAddition – ^{177}Lu-PSMA-617 + ARPI vs. ARPI in mHSPC – rPFS

rPFS by BIRC – the primary endpoint was met



	^{177}Lu-PSMA-617 + ADT + ARPI (N = 572)	ADT + ARPI (N = 572)
Events – n (%) rPD Death without rPD	139 (24.3) 112 (19.6) 27 (4.7)	172 (30.1) 152 (26.6) 20 (3.5)
HR (95% CI)	0.72 (0.58, 0.90)	
p value	0.002[a]	
Median rPFS (95% CI) – months	NR (NE, NE)	NR (29.7, NE)

rPFS benefit was consistent across subgroups

		HR (95% CI)	Events, n/N (%) ^{177}Lu-PSMA-617 + ADT + ARPI	Events, n/N (%) ADT + ARPI
All patients		0.72 (0.58, 0.90)	139/572 (24.3)	172/572 (30.1)
Tumour volume[a]	High	0.72 (0.56, 0.92)	116/389 (29.8)	144/390 (36.9)
	Low	0.73 (0.42, 1.27)	23/183 (12.6)	28/182 (15.4)
Age	< 70 years	0.68 (0.50, 0.92)	75/326 (23.0)	99/326 (30.4)
	≥ 70 years	0.78 (0.56, 1.10)	64/246 (26.0)	73/246 (29.7)
mHSPC[b]	De novo[c]	0.74 (0.54, 1.01)	74/298 (24.8)	89/274 (32.5)
	Recurrent	0.74 (0.53, 1.04)	60/249 (24.1)	77/274 (28.1)
Previous/planned treatment to primary tumour[d]	Yes	0.75 (0.46, 1.22)	31/156 (19.9)	35/155 (22.6)
	No	0.71 (0.55, 0.92)	108/416 (26.0)	137/417 (32.9)
Initial Gleason score	< 8	0.54 (0.33, 0.88)	29/152 (19.1)	39/134 (29.1)
	≥ 8	0.76 (0.58, 0.99)	99/389 (25.4)	122/406 (30.0)
Baseline LDH level	≤ 260 IU/L	0.69 (0.53, 0.89)	104/471 (22.1)	143/489 (29.2)
	> 260 IU/L	0.61 (0.30, 1.26)	20/37 (54.1)	18/33 (54.5)
Baseline PSA level	< Median	0.85 (0.62, 1.16)	73/279 (26.2)	83/287 (28.9)
	≥ Median	0.60 (0.44, 0.83)	63/284 (22.2)	89/283 (31.4)
ECOG performance status	0	0.78 (0.59, 1.03)	92/397 (23.2)	107/407 (26.3)
	1–2	0.63 (0.43, 0.92)	47/175 (26.9)	64/162 (39.5)



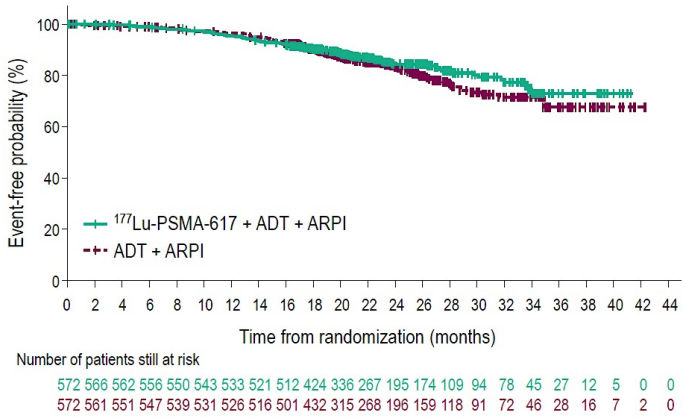
[a] Per CHAARTED criteria | [b] Subgroup not prespecified | [c] Stage IVb (*AJCC Cancer Staging Manual* 8th edition) | [d] Prostatectomy or definitive radiotherapy to prostate only
LDH, lactate dehydrogenase

Tagawa ST et al, ESMO 2025

PSMAddition – ^{177}Lu-PSMA-617 + ARPI vs. ARPI in mHSPC – OS

Interim OS (prespecified intent-to-treat analysis – follow-up continues)

Data cut-off: 13 January 2025



	^{177}Lu-PSMA-617 + ADT + ARPI (N = 572)	ADT + ARPI (N = 572)
Events – n (%)	85 (14.9)	99 (17.3)
Censored – n (%)	487 (85.1)	473 (82.7)
HR (95% CI)	0.84 (0.63, 1.13)	
p value	0.125[a]	
Median OS (95% CI) – months	NR (NE, NE)	NR (NE, NE)

[a] Significance threshold at OS IA1: 0.0011 (one-sided; stratified log-rank test); information fraction, 47.3%

Selected other secondary efficacy endpoints

Data cut-off: 13 January 2025

	^{177}Lu-PSMA-617 + ADT + ARPI (n = 224)	ADT + ARPI (n = 213)
Best response per RECIST v1.1[a]		
ORR – % (95% CI)	85.3 (79.9, 89.6)	80.8 (74.8, 85.8)
Complete response – %	57.1	42.3

	^{177}Lu-PSMA-617 + ADT + ARPI (n = 572)	ADT + ARPI (n = 572)
Time to PSA progression		
Median (95% CI) – months	NR (NE, NE)	NR (NE, NE)
HR (95% CI)	0.42 (0.30, 0.59)	
PSA nadir < 0.2 ng/mL at 48 weeks		
% (95% CI)	87.4 (83.6, 90.6)	74.9 (70.3, 79.1)

	^{177}Lu-PSMA-617 + ADT + ARPI (n = 572)	ADT + ARPI (n = 572)
Time to symptomatic skeletal event		
Median (95% CI) – months	NR (NE, NE)	NR (NE, NE)
HR (95% CI)	0.89 (0.62, 1.26)	
Time to mCRPC		
Median (95% CI) – months	NR (33.58, NE)	29.63 (25.30, NE)
HR (95% CI)	0.70 (0.58, 0.84)	
PFS per investigator assessment		
Median (95% CI) – months	NR (NE, NE)	NR (29.63, NE)
HR (95% CI)	0.64 (0.51, 0.79)	

[a] Patients with measurable soft tissue disease at baseline, accounting for bone disease

Tagawa ST et al, ESMO 2025

PSMAddition – ^{177}Lu-PSMA-617 + ARPI vs. ARPI in mHSPC – Safety

Overall incidence of adverse events

Data cut-off

Patients with on-treatment AE – n (%)[a]	^{177}Lu-PSMA-617 + ADT + ARPI (N = 564)		ADT + ARPI (N = 565)	
	Any grade	Grade ≥3	Any grade	Grade ≥3
Any	555 (98.4)	286 (50.7)	546 (96.6)	243 (43.0)
Related to any study treatment	504 (89.4)	128 (22.7)	394 (69.7)	69 (12.2)
^{177}Lu-PSMA-617-related	441 (78.2)	78 (13.8)	–	–
ADT and/or ARPI-related	418 (74.1)	81 (14.4)	394 (69.7)	69 (12.2)
Serious	180 (31.9)	150 (26.6)	162 (28.7)	129 (22.8)
Related to any study treatment	39 (6.9)	31 (5.5)	14 (2.5)	12 (2.1)
^{177}Lu-PSMA-617-related	17 (3.0)	13 (2.3)	–	–
ADT and/or ARPI-related	27 (4.8)	22 (3.9)	14 (2.5)	12 (2.1)
Leading to death	15 (2.7)	15 (2.7)	14 (2.5)	14 (2.5)
Treatment-related	0	0	0	0
Leading to discontinuation of any study treatment	91 (16.1)	46 (8.2)	51 (9.0)	23 (4.1)
Leading to ^{177}Lu-PSMA -617…				
Discontinuation	45 (8.0)	28 (5.0)	–	–
Dose reduction	22 (3.9)	13 (2.3)	–	–
Dose delay	68 (12.1)	27 (4.8)	–	–

[a] Investigator-assessed grade, seriousness and relationship

AEs grouped by safety topics of interest

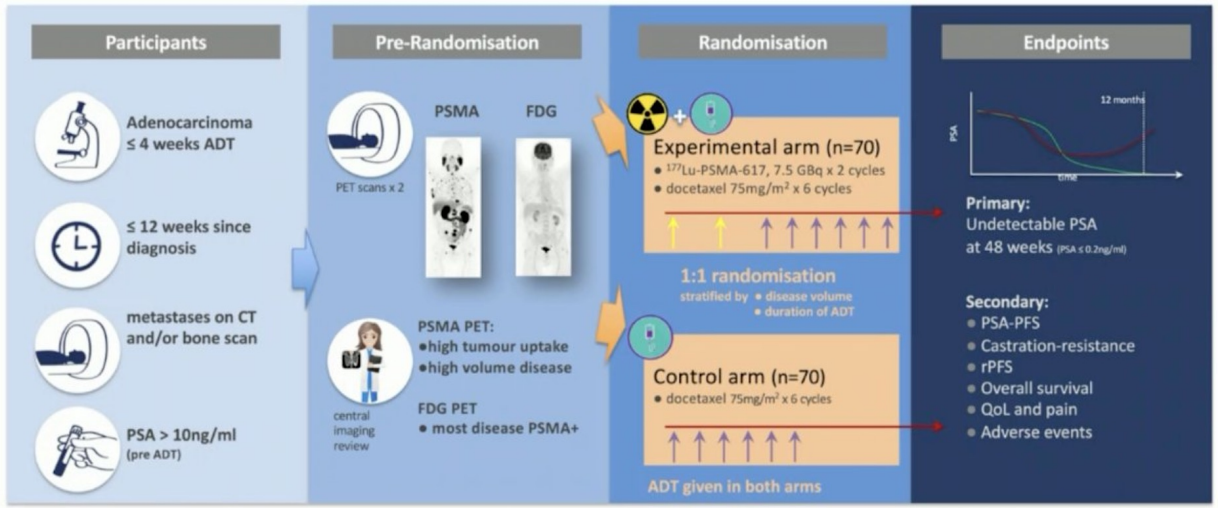
Data cut-off: 13 J

Patients with AE in grouping – n (%)	^{177}Lu-PSMA-617 + ADT + ARPI (N = 564)		ADT + ARPI (N = 565)	
	Any grade	Grade ≥3	Any grade	Grade ≥3
Dry mouth[a]	262 (46.5)	0	22 (3.9)	0
Cytopenias	248 (44.0)	81 (14.4)	115 (20.4)	28 (5.0)
Anaemia[b]	158 (28.0)	28 (5.0)	79 (14.0)	16 (2.8)
Neutropenia[c]	83 (14.7)	21 (3.7)	24 (4.2)	5 (0.9)
Thrombocytopenia[d]	63 (11.2)	10 (1.8)	19 (3.4)	3 (0.5)
Fractures	52 (9.2)	16 (2.8)	43 (7.6)	15 (2.7)
Renal events[e]	40 (7.1)	9 (1.6)	26 (4.6)	5 (0.9)
Second primary malignancies	37 (6.6)	24 (4.3)	31 (5.5)	14 (2.5)
Dry eye	33 (5.9)	0	3 (0.5)	0
Intracranial haemorrhage	6 (1.1)	4 (0.7)	4 (0.7)	2 (0.4)

[a] Dry mouth, dry throat, salivary hyposecretion, lip dry, mucosal dryness, salivary hypersecretion, Sjogren's syndrome
[b] Anaemia, anaemia macrocytic, haematocrit decreased, haemoglobin decreased, normocytic anaemia, red blood cell count decreased
[c] Agranulocytosis, febrile neutropenia, neutropenia, neutrophil count decreased
[d] Platelet count decreased, thrombocytopenia
[e] Blood creatinine increased, acute kidney injury, renal failure, chronic kidney disease, blood urea increased, glomerular filtration rate decreased, creatinine renal clearance decreased, proteinuria, renal impairment, uraemic encephalopathy

Tagawa ST et al, ESMO 2025

UpFrontPSMA – Docetaxel +/- ^{177}Lu-PSMA-617 x 2 in mHSPC



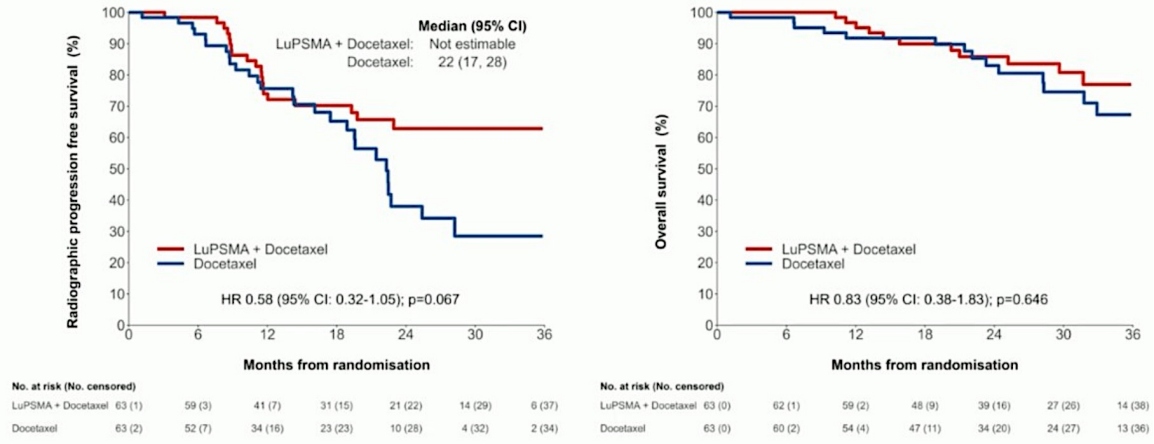
Undetectable PSA at week 48

Treatment	Lu-PSMA + docetaxel (n=61)*	Docetaxel (n=61)*
Undetectable PSA at week 48, %	41% (95% CI 30-54)	16% (95% CI 9-28)
	OR 3.88 (95% CI 1.61-9.38); p=0.002	
Undetectable PSA at any time point, %	51% (95% CI 39-63)	32% (95% CI 22-45)
	OR 2.14 (95% CI 1.03-4.46); p=0.042	
Undetectable PSA at week 12, %	17% (95% CI 10-29)	18% (95% CI 10-29)
	OR 0.94 (95% CI 0.37-2.36); p=0.895	

Patient characteristics

	Lu-PSMA + docetaxel (n=63)	Docetaxel (n=63)	All (n=126))
Age (median, IQR)	69 (69 - 73)	69 (64 - 74)	69 (63 - 74)
ECOG PS 0-1, n (%)	63 (100%)	62 (98%)	125 (99%)
T3-4 stage, n (%)	38 (67%)	45 (75%)	83 (72%)
N1 stage, n (%)	47 (77%)	48 (77%)	95 (77%)
Visceral disease, n (%)	4 (6%)	6 (10%)	10 (8%)
Grade Group 4-5, n (%)	48 (89%)	51 (92%)	99 (91%)
PSA (ng/ml), median (IQR)	48 (18 - 113)	31 (15 - 93)	38 (16 - 102)
High-volume disease (conventional imaging) , n (%)	60 (95%)	55 (87%)	115 (91%)
≤ 28 days on ADT at registration, n (%)	54 (86%)	51 (81%)	105 (83%)

Time-to-event analyses



Azad AA et al, ESMO 2024

Where The Field Is Heading?

- Earlier Lines of Therapy
- Patient Selection
- Sequencing of α and β
- Combinations
- Beyond PSMA
- Salivary Preservation

Therapeutic Radionuclides in Prostate Cancer

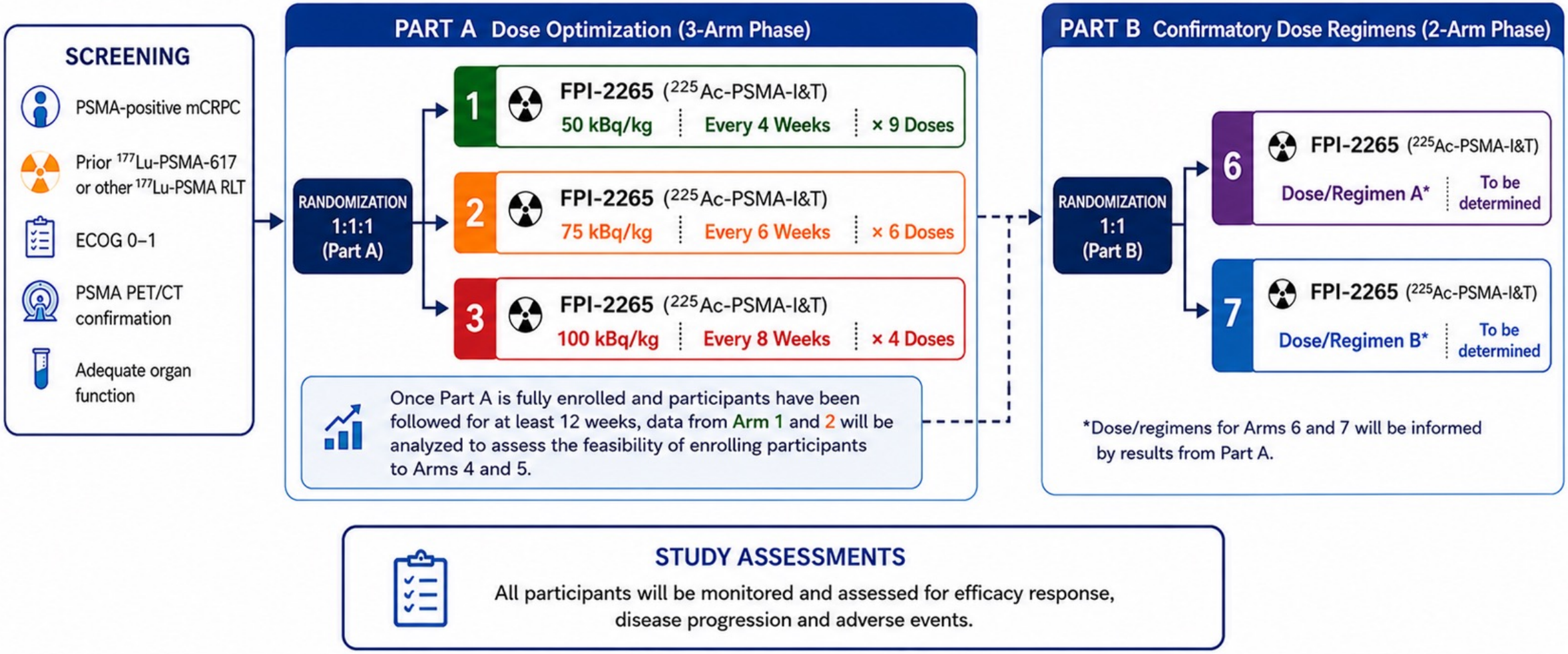
Radionuclide	Emission	Half-life	Tissue range	Example agent
^{177}Lu	Beta	6.7 d	~2 mm	PSMA-targeted (small molecule): **^{177}Lu-PSMA-617**, ^{177}Lu-PSMA-I&T GRPR-targeted (small molecule): LY4257496
^{225}Ac	Alpha	9.9 d	50–100 µm	PSMA-targeted (small molecule): ^{225}Ac-PSMA-617, ^{225}Ac-PSMA-I&T (FPI-2265), ^{225}Ac-PSMA-Trillium (BAY 3563254) PSMA-targeted (antibody): ^{225}Ac-rosopatamab tetraxetan (CONV01-α) STEAP2-targeted (antibody): AZD2284
^{223}Ra	Alpha	11.4 d	<100 µm	Bone-targeted (Ca^{2+} mimetic): **^{223}Ra-dichloride**
^{227}Th	Alpha	18.7 d	40–80 µm	PSMA-targeted (antibody): ^{227}Th-pelgifatamab (BAY 2315497)
^{212}Pb	Alpha	10.6 h	50–90 µm	PSMA-targeted (small molecule): ^{212}Pb-ADVC001
^{67}Cu	Beta	2.6 d	~0.6 mm	PSMA-targeted (small molecule): ^{67}Cu-SAR-bisPSMA

Red = FDA Approved agents.

AlphaBreak Study (NCT06402331)

FPI-2265 (^{225}Ac-PSMA-I&T) in PSMA-Positive mCRPC Previously Treated With ^{177}Lu-PSMA RLT

Open-label, randomized, multicenter Phase 2 dose-optimization study



STUDY ASSESSMENTS

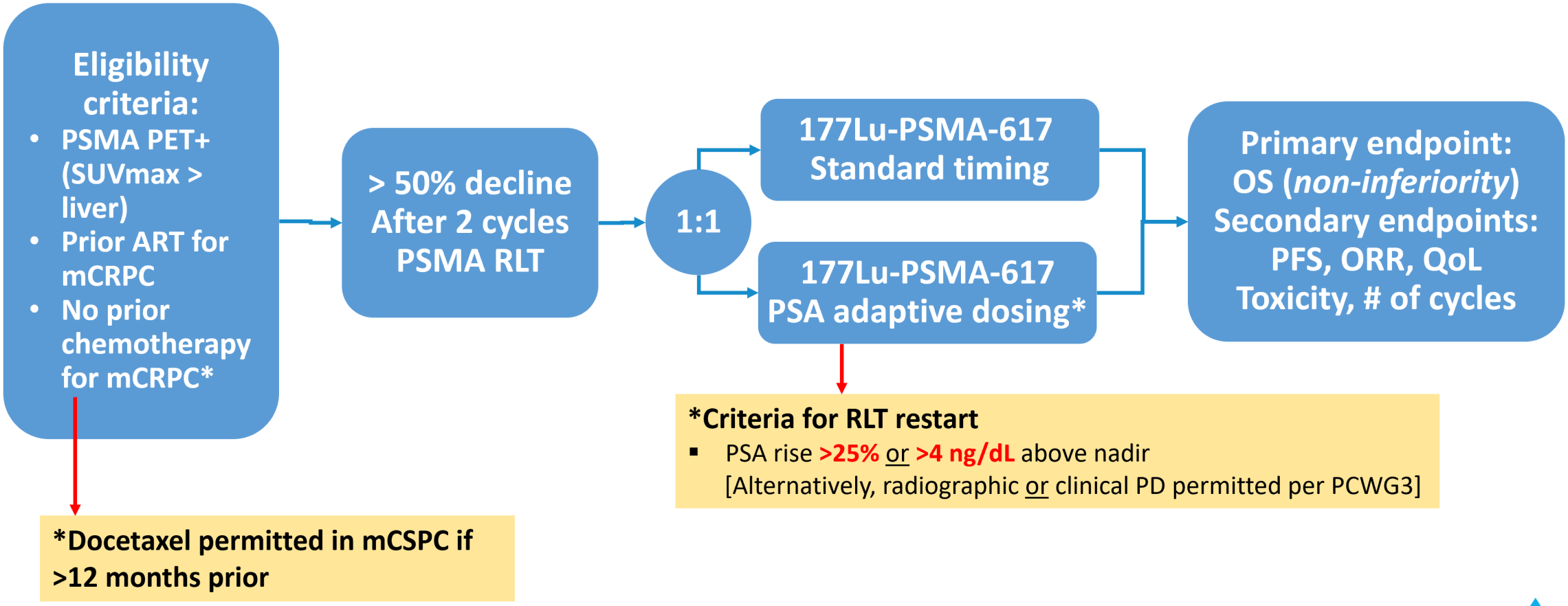
All participants will be monitored and assessed for efficacy response, disease progression and adverse events.

mCRPC = metastatic castration-resistant prostate cancer; PSMA = prostate-specific membrane antigen; RLT = radioligand therapy

https://clinicaltrials.gov/study/NCT06402331; https://www.urologytimes.com/view/dosing-begins-in-study-of-psma-targeted-therapy-in-mcrpc

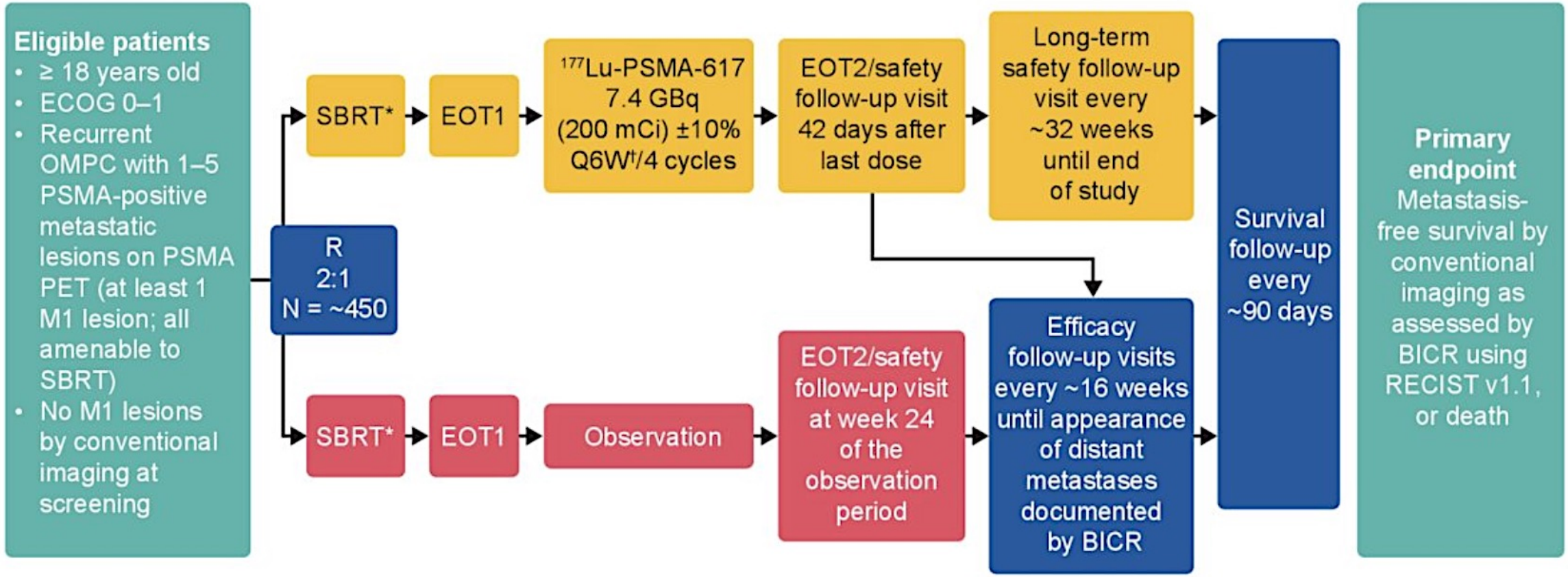
UC San Diego Health

RECIPROCAL – Currently Accruing



https://clinicaltrials.gov/study/NCT07200830

PSMA DC – SBRT +/- ^{177}Lu-PSMA-617 without ADT



Sartor O et al. ASCO 2025

AcTFirst – ^{225}Ac-PSMA-617 +/- ARPI vs. SOC in mCRPC

TRIAL SCHEMA

AcTFirst — 225Ac-PSMA-617 (AAA817) ± ARPI vs SoC in PSMA+ mCRPC

NCT06855277 • Phase 3, open-label, randomized • N = 940 *Primary completion: Sep 2028*

KEY ELIGIBILITY

- Adult men, ECOG 0–2; adenocarcinoma (no neuroendocrine)
- PSMA-PET positive on approved PSMA imaging agent
- mCRPC with progression on ARPI in mHSPC/earlier (last therapy)
- No prior taxane in mCRPC; no prior PSMA-RLT, Ra-223, or PARPi for mCRPC
- Taxane in mHSPC allowed if next-line therapy clinically appropriate
- HRR-mutated patients eligible only if prior PARPi

RANDOMIZE 1 : 1 : 1

INVESTIGATIONAL A
AAA817 + ARPI

- 225Ac-PSMA-617 10 MBq ± 10% IV
- Up to 6 cycles • every 8 weeks
- + enzalutamide or abiraterone (oral)

INVESTIGATIONAL B
AAA817 monotherapy

- 225Ac-PSMA-617 10 MBq ± 10% IV
- Up to 6 cycles • every 8 weeks
- No ARPI

CONTROL
Investigator's choice SoC

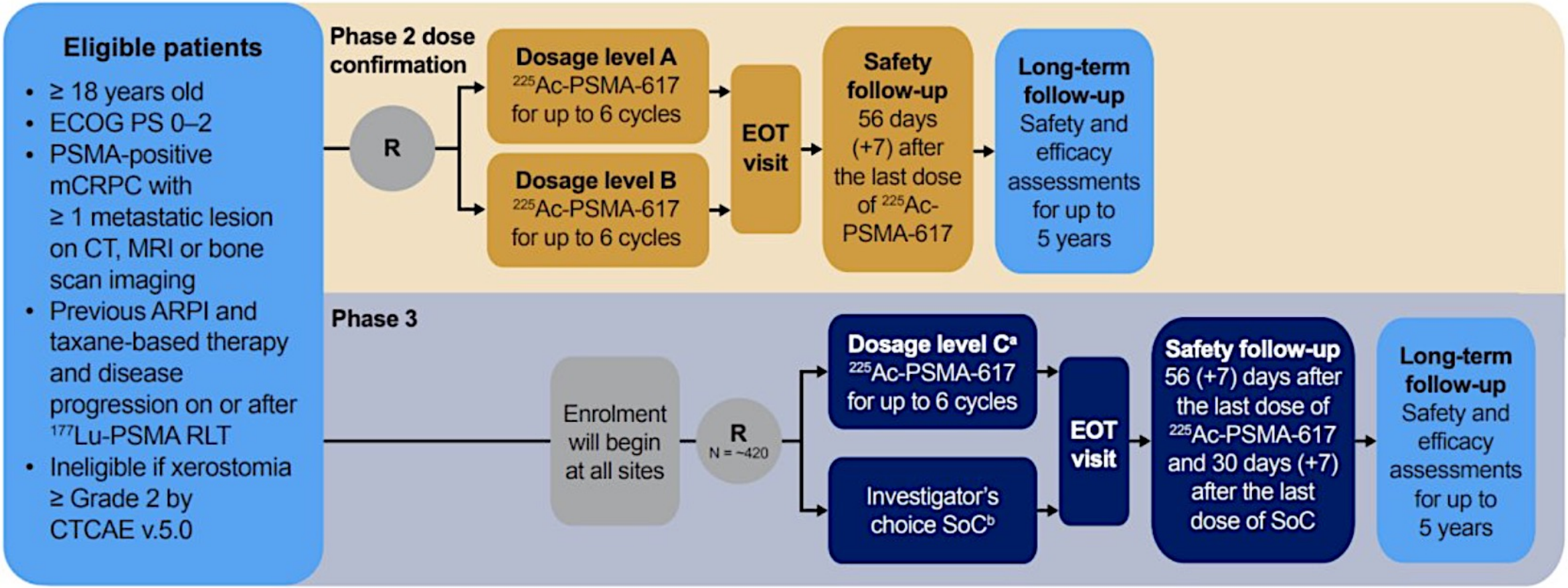
- ARPI change (enza or abi), or
- Docetaxel / cabazitaxel, or
- 177Lu-PSMA-617 (AAA617)
- No crossover permitted

PRIMARY ENDPOINT Radiographic progression-free survival (rPFS) — investigator-assessed

KEY SECONDARY **Overall survival** • rPFS by BICR (PCWG3-RECIST v1.1) • PFS, PFS2 • ORR, DCR, DoR • TTSSE • PSA50/PSA90 • PK • FACT-P, BPI-SF • Safety

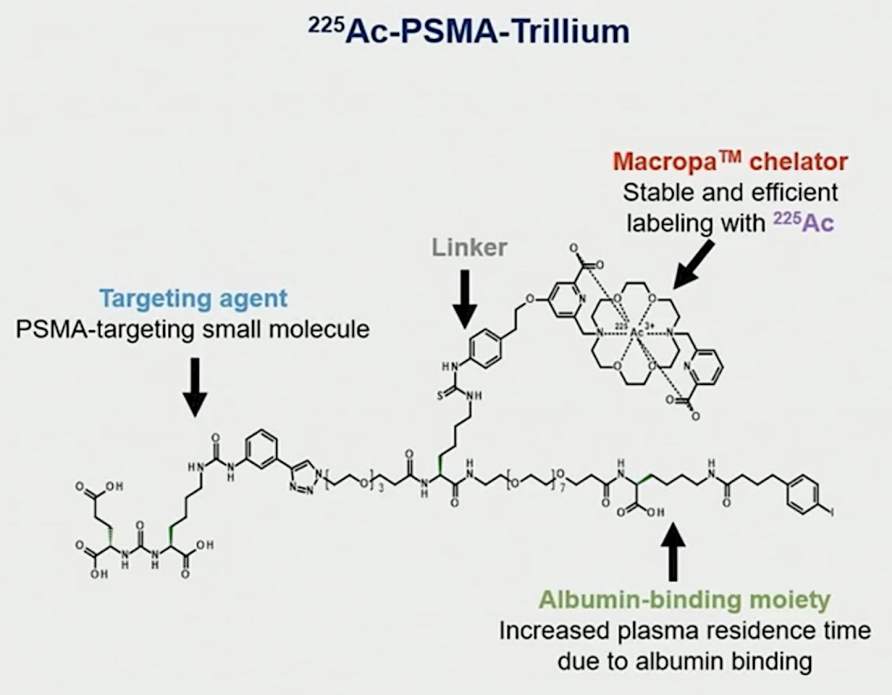
https://clinicaltrials.gov/study/NCT06855277

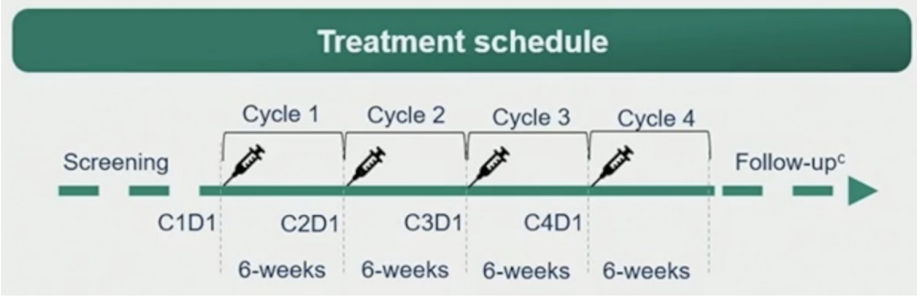
PSMAcTION – ^{225}Ac-PSMA-617 vs. SOC



Emmett L et al. ESMO 2025

Phase 1 PAnTHA – 225Ac-PSMA-Trillium (BAY 3563254)

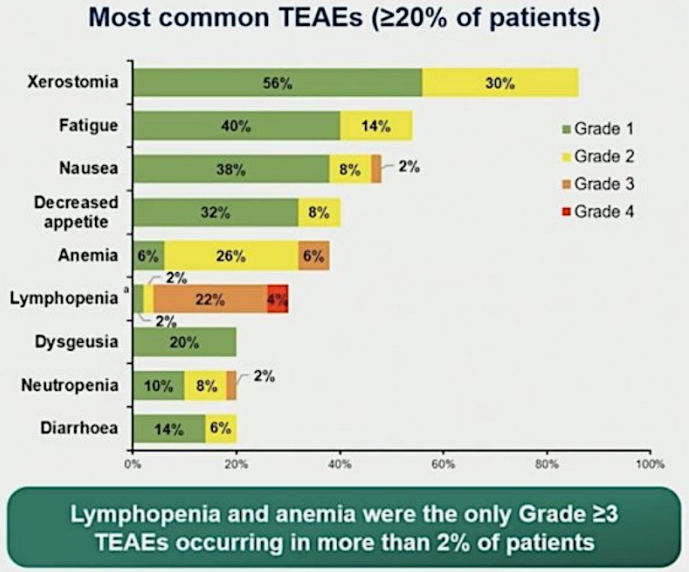




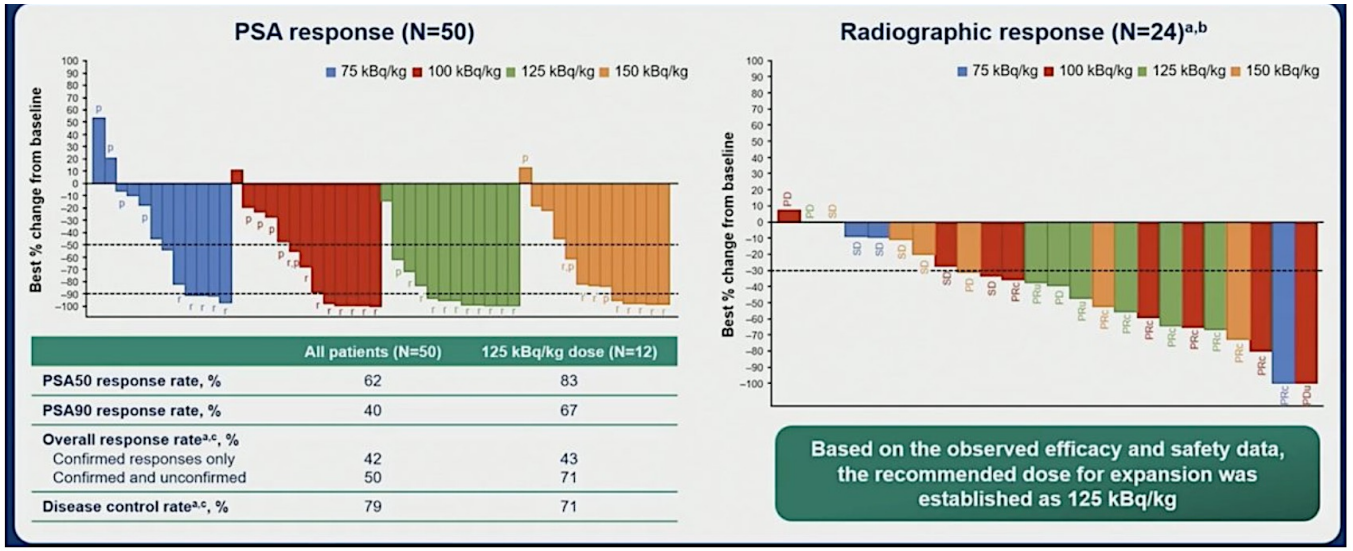
Treatment-emergent adverse events (TEAEs)	75 kBq/kg (N=12)	100 kBq/kg (N=13)	125 kBq/kg (N=12)	150 kBq/kg (N=13)	Total (N=50)
Any TEAE, n (%)	12 (100)	13 (100)	12 (100)	13 (100)	50 (100)
Grade 3-4	5 (42)	2 (15)	6 (50)	9 (69)	22 (44)
Leading to dose modification	0 (0)	1 (8)	1 (8)	3 (23)	5 (10)
Leading to permanent discontinuation	1 (8)	1 (8)	1 (8)	0 (0)	3 (6)
Leading to death	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
DLTs	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)

TEAEs leading to permanent discontinuation were:

- Grade 3 lymphopenia (worsened from Grade 2; related to study drug)
- Grade 3 glomerular filtration rate decreased (unrelated to study drug)
- Grade 2 renal impairment (related to study drug)



Lymphopenia and anemia were the only Grade ≥3 TEAEs occurring in more than 2% of patients



	All patients (N=50)	125 kBq/kg dose (N=12)
PSA50 response rate, %	62	83
PSA90 response rate, %	40	67
Overall response rate[a,c], %		
Confirmed responses only	42	43
Confirmed and unconfirmed	50	71
Disease control rate[a,c], %	79	71

Based on the observed efficacy and safety data, the recommended dose for expansion was established as 125 kBq/kg

Conclusions

- Radium-223 established the first survival benefit for a radiopharmaceutical in mCRPC – bone-metastatic, symptomatic disease, post-ARPI/post-chemotherapy
- Radium-223 + enzalutamide now extends that benefit into the first-line mCRPC setting for bone-dominant disease, with OS and rPFS gains and a manageable safety profile when bone-protective agents are used
- 177Lu-PSMA-617 is established in PSMA-positive mCRPC across both the post-taxane (VISION) and taxane-naïve / post-ARPI (PSMAfore) settings
- The field is now evolving into mHSPC (PSMAddition) and toward earlier-line use, with patient selection and OS maturation as the key open questions
- Next-generation agents – particularly PSMA-targeted alpha emitters (225Ac-PSMA-617) – are the most important near-term frontier and other non-PSMA-targeted agents being explored

Discussion Questions

67 y/o M with de novo metastatic PC, high volume, Gleason 9. Received ARASENS regimen with PSA response but relapsed after 1 year.
Has extensive skeletal mets with myelophthisic anemia. Because of anemia, he was not considered a candidate for lutetium by radiation oncology.

Can lutetium be safely administered in a patient with cytopenias from prostate cancer — myelophthisis?

RTP
RESEARCH
TO PRACTICE

Discussion Questions

65-year-old man with de novo metastatic prostate cancer but also with end-stage renal disease. Disease progression on triplet therapy.

Can lutetium be used in a patient with ESRD?

In which situations, if any, would you include lutetium in the management of hormone-sensitive metastatic disease?

RTP
RESEARCH
TO PRACTICE

Discussion Questions

71 yo man with hormone-sensitive metastatic disease; osseous and pulmonary metastases.
Declined docetaxel chemotherapy. Progression on ADT/ARPI.
Receives lutetium with stable disease. Completed tx 10 months ago.
Now presents with slowly rising PSA, but asymptomatic.

How are you generally sequencing lutetium relative to taxane-based chemotherapy for your patients with mCRPC? When do you prefer to give lutetium first?

When would you initiate next-line therapy for this patient versus continue observation?

RTP
RESEARCH
TO PRACTICE

Discussion Questions (Continued)

71 yo man with hormone-sensitive metastatic disease; osseous and pulmonary metastases.
Declined docetaxel chemotherapy. Progression on ADT/ARPI.
Receives lutetium with stable disease. Completed tx 10 months ago.
Now presents with slowly rising PSA, but asymptomatic.

Would you consider repeat treatment with lutetium?

What about new radioligands? What are potential advantages and disadvantages of actinium compared to lutetium?

In which situations, if any, will you use fewer than 6 cycles of lutetium?

How do you prevent and manage xerostomia in patients on lutetium?

RTP
RESEARCH
TO PRACTICE

Discussion Questions

83-year-old male with mCRPC heavily pretreated with ARPI, docetaxel and lutetium. Progressive symptomatic bone disease, ECOG 2.

Would you offer radium-223?

In what situations are you using radium-223 as monotherapy? What about in combination with enzalutamide?

RTP
RESEARCH
TO PRACTICE

What Clinicians Want to Know: Addressing Community Oncologists' Questions About the Care of Patients with Prostate Cancer

Saturday, May 30, 2026

7:00 PM – 9:00 PM CT (8:00 PM – 10:00 PM ET)

Faculty

Wassim Abida, MD, PhD

Rahul Aggarwal, MD

Emmanuel S Antonarakis, MD

Karim Fizazi, MD, PhD

Moderator

Rana R McKay, MD, FASCO

RTP RESEARCH TO PRACTICE

Consensus or Controversy? Documenting and Discussing Investigators' Approaches to the Use of Oral SERDs and Agents Targeting the PI3K/AKT/mTOR Pathway in Breast Cancer

A CME Symposium Held Adjunct with the 2026 ASCO® Annual Meeting

Sunday, May 31, 2026

7:00 PM – 9:00 PM CT (8:00 PM – 10:00 PM ET)

Faculty

Sara A Hurvitz, MD, FACP
Erica Mayer, MD, MPH, FASCO
Joyce O'Shaughnessy, MD
Nicholas Turner, MD, PhD

Moderator

Sara M Tolaney, MD, MPH

RTP RESEARCH TO PRACTICE

What Clinicians Want to Know: Addressing Community Oncologists' Questions About the Current and Future Management of Endometrial Cancer

A CME Symposium Held Adjunct with the 2026 ASCO® Annual Meeting

Sunday, May 31, 2026

7:00 PM – 8:30 PM CT (8:00 PM – 9:30 PM ET)

Faculty

Floor J Backes, MD

Brian M Slomovitz, MD

Moderator

Shannon N Westin, MD, MPH, FASCO, FACOG

RTP RESEARCH TO PRACTICE

What Clinicians Want to Know: Addressing Community Oncologists' Questions About the Roles of CAR T-Cell Therapy and Bispecific Antibodies in the Management of Non-Hodgkin Lymphoma

A CME Symposium Held Adjunct with the 2026 ASCO® Annual Meeting

Sunday, May 31, 2026

7:00 PM – 9:00 PM CT (8:00 PM – 10:00 PM ET)

Faculty

Joshua Brody, MD

Manali Kamdar, MD, MBBS

Tycel Phillips, MD, FASCO

Jason Westin, MD, MS, FACP, FASCO

Moderator

Jeremy S Abramson, MD, MMSc

RTP RESEARCH TO PRACTICE

Thank you for joining us!
Your feedback is very important to us.

Please complete the survey currently up on the iPads for attendees in the room and on Zoom for those attending virtually. The survey will remain open up to 5 minutes after the meeting ends.

How to Obtain CME Credit

In-person attendees: Please refer to the program syllabus for the CME credit link or QR code. Online/Zoom attendees: The CME credit link is posted in the chat room.

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