

KLK2-comPAS: A Phase 3, Randomized, Double-blind, Placebo-controlled Study of Pasritamig, JNJ-9401, and Best Supportive Care in Metastatic Castration-resistant Prostate Cancer

Kim N. Chi¹, Craig Gedye², Laurence Belkoff³, Pedro Barata⁴, Alice Bernard-Tessier⁵, Julie N. Graff⁶, Nobuaki Matsubara⁷, Shahneen Sandhu⁸, Michael T. Schweizer⁹, Mark Stein¹⁰, Gunhild von Amsberg¹¹, Bilal A. Siddiqui¹², Xiao X. Wei¹³, Mina Hosseini¹⁴, Katie Dacey¹⁵, Fouad Moussa¹⁶, Sherry C. Wang¹⁷, Shiva Dibaj¹⁸, Sarah Nagle¹⁴, Elena Castro¹⁹

¹BC Cancer Agency, Vancouver, BC, Canada; ²Icon Cancer Centre Adelaide, SA, Australia; ³Midlantic Urology, Bala Cynwyd, PA, USA; ⁴University Hospitals, Seidman Cancer Center, Cleveland, Ohio, USA; ⁵Institut Gustave Roussy, University of Paris-Saclay, Villejuif, France; ⁶Oregon Health & Science University, Portland, OR; ⁷National Cancer Center Hospital East, Chiba, Japan; ⁸Peter MacCallum Department of Oncology, University of Melbourne, Melbourne, Australia; ⁹Fred Hutch Cancer Center, University of Washington, Seattle, WA; ¹⁰Columbia University Medical Center, New York, NY, USA; ¹¹University Cancer Center Hamburg and Martini-Klinik, Hamburg, Germany; ¹²Department of Genitourinary Medical Oncology, The University of Texas MD Anderson Cancer Center, Houston, TX, USA; ¹³Division of Medical Oncology, Dana-Farber Cancer Institute, Boston, MA, USA; ¹⁴Johnson & Johnson, Raritan, NJ, USA; ¹⁵Johnson & Johnson, Horsham, PA, USA; ¹⁶Johnson & Johnson, Spring House, PA, USA; ¹⁷Johnson & Johnson, San Francisco, CA, USA; ¹⁸Johnson & Johnson, San Diego, CA, USA; ¹⁹Hospital Universitario 12 de Octubre, Madrid, Spain

Key Takeaways

-  Pasritamig (PAS) is a first-in class, KLK2xCD3 T cell engager targeting human kallikrein 2 (KLK2), a novel, highly prostate-specific target that exists in secreted and cell surface forms.¹
-  PAS demonstrated a favorable and differentiated safety profile that supports Q6W outpatient dosing (<10% CRS, all grade 1 [fever only]).^{2,3}
-  JNJ-9401 is a PSMAxCD28 costimulatory TCE that synergizes with PAS.
-  The comPAS study Part 1 is evaluating PAS + BSC (best supportive care) vs placebo + BSC (NCT07164443). In Part 2, PAS + BSC, PAS + JNJ-9401 + BSC, and placebo + BSC are being evaluated.
-  The trial is active and currently enrolling participants.



Poster Please scan QR code

Copies of this poster obtained through QR, AR and/or text key codes are for personal use only and may not be reproduced without written permission of the authors.



kchi@bccancer.bc.ca

Acknowledgments

We are grateful to the patients, their families/caregivers, and the clinical trial teams for their contribution to this study. The study was sponsored by Johnson & Johnson.

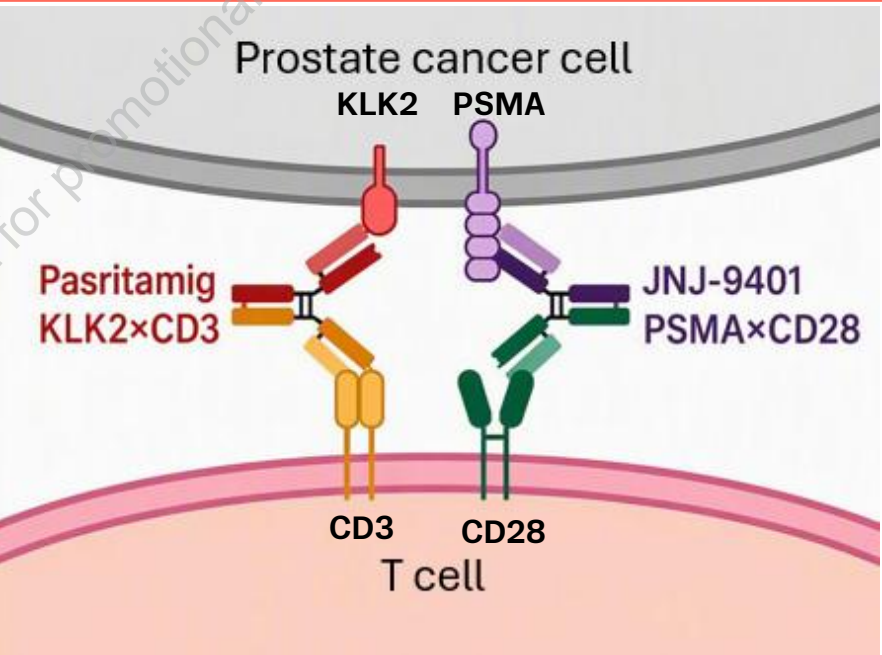
Disclosures

Honoraria/Consulting/Funding: Amgen, Astellas Pharma, AstraZeneca, Bayer, Bristol Myers Squibb, Janssen, Merck, Novartis, Sanofi, Pfizer, and Eli Lilly Research Funding: Research funding has been received from organizations including AstraZeneca, Bayer, Janssen, and Prostate Cancer Canada.

Introduction

- Metastatic castration-resistant prostate cancer (mCRPC), also termed androgen pathway modulation-resistant (APMR) in PCWG4⁴, remains an incurable disease with high morbidity and a median overall survival of approximately two years.⁵
- Human kallikrein 2 (KLK2) is highly specific to normal and malignant prostate tissue, including in late-stage mCRPC.¹ It exists in secreted and cell surface forms.
- Pasritamig** (PAS) is a first-in-class KLK2xCD3 T cell engager (TCE). PAS is a humanized, IgG1-based bispecific antibody that targets KLK2-expressing cells via CD3 engagement, inducing T cell-mediated targeted cytotoxicity.¹
- JNJ-9401** is a human IgG1 bispecific antibody that binds to PSMA on tumor cells and CD28 on T cells. Addition of a selective costimulatory signal (Signal 2) via CD28 agonism to antigen specific CD3-induced T-cell activation (Signal 1) leads to long-lasting antitumor responses by potentiating T-cell activation, increasing T-cell infiltration into tumors, improving T-cell-mediated tumor killing, enhancing T-cell memory phenotype, and optimizing tumor specificity while limiting on-target/off-tumor toxicity.
 - Phase 1 data for Pas+JNJ-9401 will be presented at a forthcoming medical meeting
- KLK2-comPAS** is a double-blind, randomized, global phase 3 study to evaluate the safety and efficacy of pasritamig, JNJ-9401, and BSC in participants with late-line mCRPC.

Figure 1: Antitumor activity with pasritamig + JNJ9401 T-cell activation requiring both T-cell receptor engagement and co-stimulation evaluated in Part 2



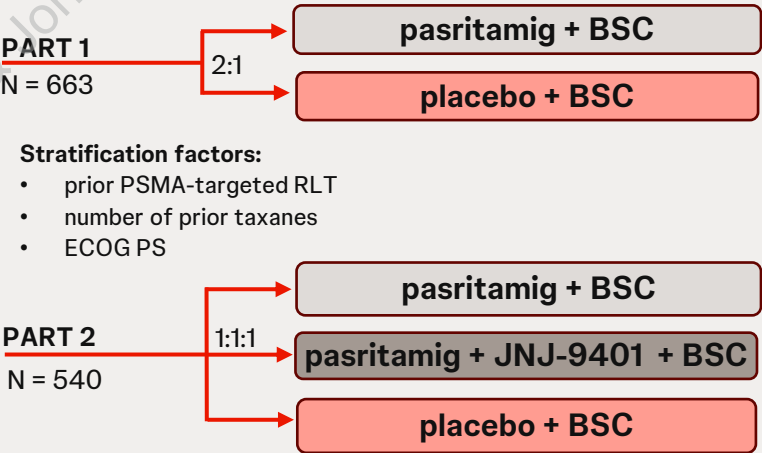
Methods

Key Eligibility

- Received all available life-prolonging therapies (ARPI, taxanes, RLT, PARPI for BRCAm)
- ECOG 0-2
- PSA ≥2 ng/mL
- Adequate organ function
- Ongoing ADT or prior orchiectomy
- No visceral metastases
- No prior KLK2 or CD3 directed therapies
- No active autoimmune disease requiring immunosuppression

Study design

Figure 2: A global, randomized, double-blinded, placebo-controlled, phase 3 study evaluates the efficacy and safety of PAS + best supportive care (BSC), pasritamig + JNJ-6140, + BSC or placebo plus BSC (NCT07164443)



Primary endpoint: OS

Key secondary endpoints:

- rPFS (investigator-assessed per RECIST v1.1 and PCWG3)
- Time to symptomatic progression
- Time to skeletal-related event
- PFS (including clinical and/or unconfirmed bone progression)

Figure 3: PAS is administered in the outpatient setting. Treatment begins with two weekly step-up doses (3.5 mg IV on Day 1 and 18 mg IV on Day 8) followed by the target dose of 300 mg IV every 6 weeks until progression. JNJ-6401 at a dose of 200 mg SC will be administered every 6 weeks until progression.

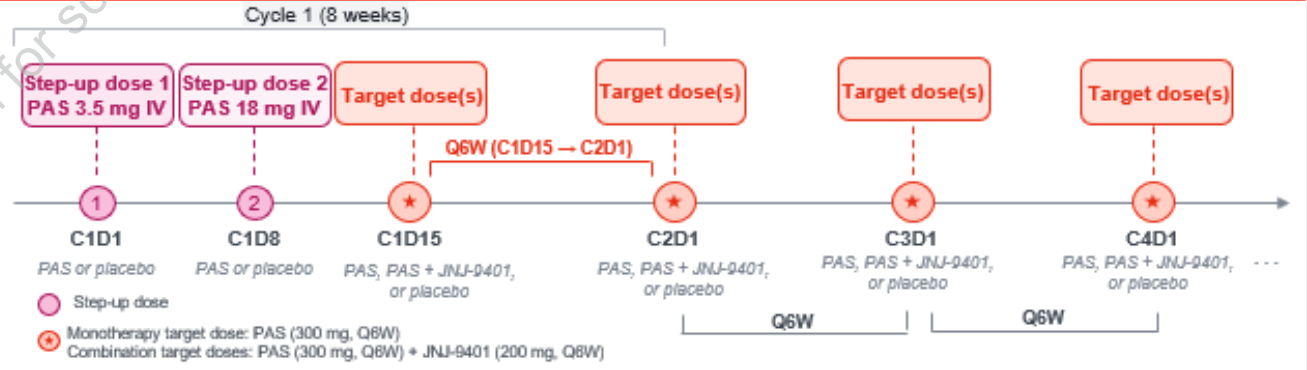


Figure 4: Countries participating in the comPAS trial



References

- Shen F et al. Clin Can Res. 2025; doi:10.1158/1078-0432.CCR-25-0950.
- Baldini C et al. ASCO Annual Meeting May 30–June 3, 2025; Chicago, IL, USA
- Stein MN et al. J Clin Oncol. 2025; doi:10.1200/JCO-25-00678
- Armstrong AJ et al. J Clin Oncol. 2026; doi:10.1200/JCO-25-02834.
- Freedland SJ et al. Prostate Cancer Prostatic Dis. doi: 10.1038/s41391-023-00725-8

