ENA 2026
EORTC NCI AACR
38th Symposium

Efficacy, molecular activity and safety from the pivotal PYNNACLE Phase 2 trial assessing rezatapopt, a p53 reactivator, in *TP53* Y220C-positive advanced solid tumors

Alison M. Schram,[1] Jean-Sebastian Frenel,[2] Andrew L. Coveler,[3] Melissa Johnson,[4] Veronique Debien,[5] Isabelle Ray-Coquard,[6] Marcel Wiesweg,[7] Yohann Loriot,[8] Lorena Fariñas-Madrid,[9] Giuseppe Curigliano,[10] Jung-Yun Lee,[11] David Shao Peng Tan,[12] Geoffrey I. Shapiro,[13] Shivaani Kummar,[14] Alastair Greystoke,[15] Peter Grimison,[16] Anna Passarelli,[17] Allison Nelson,[18] Anita Schmid,[18] Deepika Jalota,[18] Marc Fellous,[18] Ecaterina Dumbrava[19]

[1]Memorial Sloan Kettering Cancer Center, Department of Medicine, New York City, USA; [2]Institut de Cancérologie de l'Ouest, Department of Medical Oncology, Saint-Herblain, France; [3]University of Washington, Fred Hutch Cancer Center, Seattle, USA; [4]Sarah Cannon and HCA Research Institute, Lung Cancer Research, Nashville, USA; [5]Institut Bergonie, Medical Oncology Department, Bordeaux, France; [6]Centre Léon Bérard, Centre Régional de Lutte Contre Le Cancer de Lyon, Lyon, France; [7]University Hospital Essen, West German Cancer Center, Essen, Germany; [8]Institut Gustave Roussy, Department of Therapeutic Innovation and Early Trials, Villejuif, France; [9]Instituto de Investigacion Oncologica Vall d'Hebron (VHIO), Medical Oncology Department, Barcelona, Spain; [10]Istituto Europeo Di Oncologia, IRCCS, and Department of Oncology and Hemato-Oncology, University of Milano, Milano, Italy; [11]Yonsei Cancer Center and Severance Hospital, Yonsei University College of Medicine, Seoul, Republic of Korea; [12]NUS Centre for Cancer Research (N2CR), Yong Loo Lin School of Medicine, National University of Singapore, and Department of Haematology-Oncology, National University Cancer Institute, National University Hospital, Singapore; [13]Dana-Farber Cancer Institute, Department of Medical Oncology, Boston, USA; [14]Oregon Health & Science University (OHSU), Knight Cancer Institute, Portland, USA; [15]Royal Victoria Infirmary, Department of Medical Oncology, Newcastle Upon Tyne, UK; [16]Chris O'Brien Lifehouse Hospital, Department of Medical Oncology, Camperdown, Australia; [17]IRCCS Istituto Nazionale Tumori Fondazione G. Pascale, Naples, Italy; [18]PMV Pharmaceuticals, Inc., Princeton, USA; [19]The University of Texas MD Anderson Cancer Center, Department of Investigational Cancer Therapeutics, Houston, USA.

To view the presentation please scan the QR code

EORTC European Organisation for Research and Treatment of Cancer
NIH NATIONAL CANCER INSTITUTE
AACR American Association for Cancer Research

Disclosure information

Alison M. Schram

I have the following relevant financial relationships to disclose:

- Advisory board: Relay Therapeutics, Mersana, Merus, Partner Therapeutics, PMV Pharma, Schrödinger, Repare Therapeutics, Revolution Medicines, Endeavor Biomedicines, Day One Biopharmaceuticals, Transcode Therapeutics, Guardant, Boehringer Ingelheim
- Advisory role: Merus, Pfizer, PMV Pharmaceuticals, Inc., Schrödinger, Repare Therapeutics, Relay Therapeutics
- Research funding to institution: AstraZeneca, ArQule, BeiGene/SpringWorks, Black Diamond Therapeutics, Boehringer Ingelheim, Debiopharm, Elevar Therapeutics, Elevation Oncology, Kura, Lilly, Merus, Northern Biologics, Partner Therapeutics, Pfizer, Pheon Therapeutics, PMV Pharmaceuticals, Inc., Relay Therapeutics, Repare Therapeutics, Revolution Medicines, Surface Oncology

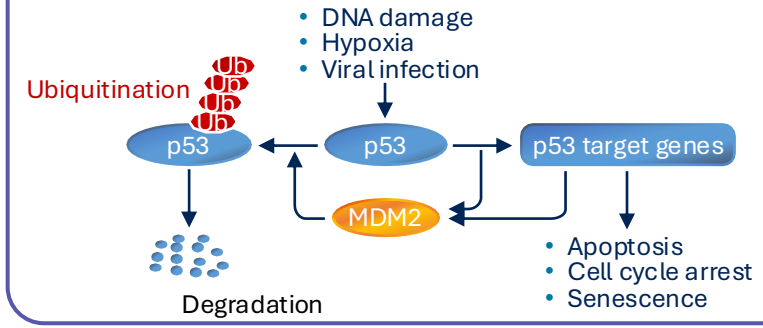
Disclosures (related to PMV Pharmaceuticals, Inc. only)

- **ALC** received institutional research funding. **MJ** received research funding. **DSPT** received personal fees for advisory board membership and institutional funding as a local Principal Investigator. **SK** received institutional research funding. **AN**, **AS, DJ** and **MF** are employees (with stock options). **EED** received research or grant funding, attended advisory boards, and provided a speaker role.

Full author disclosures can be found at the QR code.

Targeting p53 with rezatapopt, a selective p53 Y220C reactivator

p53 is a key player in the body's defense against cancer



- *TP53*: most frequently mutated gene across all cancers[1–3]
- p53: binds DNA; key role in cell cycle arrest, DNA repair and apoptosis[2–5]
- *TP53* mutations: inactivation of p53, contributing to oncogenesis[2–5]
- *TP53* Y220C: in ~1.2% of solid tumors, ~3.2% of ovarian cancer and ~3.7% of HGSOC[6]
- *TP53* Y220C: destabilizes p53, resulting in loss of tumor suppressor function[1,7,8]

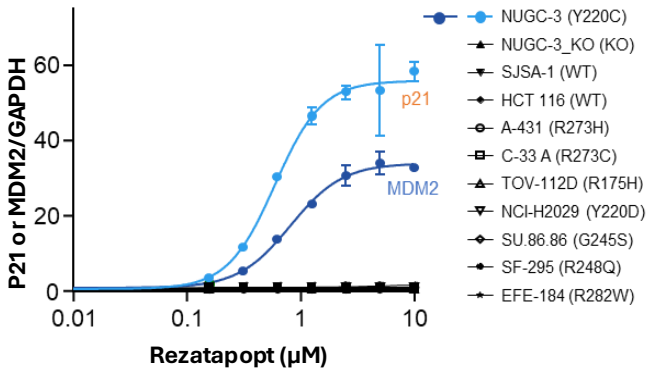
Rezatapopt is a selective p53 Y220C reactivator

- Rezatapopt: investigational, first-in-class, selective p53 reactivator specific to the *TP53* Y220C mutation[7–9]
- Rezatapopt: stabilizes mutated Y220C-p53 in wild-type p53 conformation by binding to the pocket created in the protein, restoring p53 tumor suppressor activity[7–9]

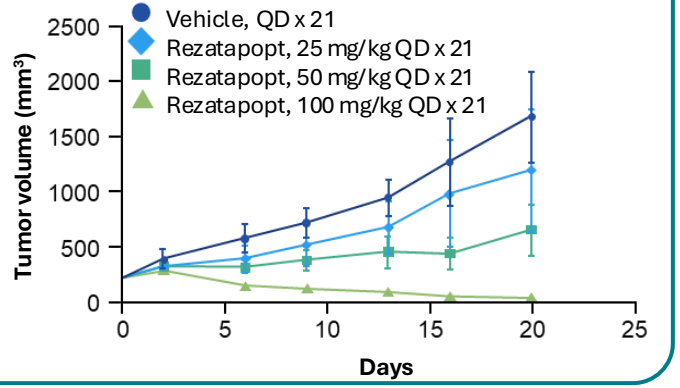


Pocket created near the site of the mutation[7–9] → Rezatapopt binds to the pocket and stabilizes the p53 protein[7–9] → p53 function restored[7–9]

p53 transcriptional activity restored[10]



Tumor regression in vivo[10]



HGSOC, high-grade serous ovarian cancer; KO, knockout; MDM2, mouse double minute 2 homolog; QD, once daily; UB, ubiquitin; WT, wild-type.

1. Baugh EH, et al. *Cell Death Differ*. 2018;25:154–160; 2. Kastenhuber ER, et al. *Cell*. 2017;170:1062–1078; 3. Levine AJ. *Nat Rev Cancer*. 2020;20:471–480; 4. Hassin O, et al. *Nat Rev Drug Discov*. 2023;22:127–144; 5. Chillemi G, et al. *Cold Spring Harb Perspect Med*. 2017;7:a028308; 6. FoundationInsights™. A proprietary database (FoundationCore® Q1-2026) used under license with review and approval from Foundation Medicine®. Available at: Biopharma Services Overview | Foundation Medicine. Deduplicated data accessed May 2026; 7. Vu BT, et al. *ACS Med Chem Lett*. 2025;16:34–39; 8. Puzio-Kuter AM, et al. *Cancer Discov*. 2025;15:1159–1179; 9. PYNNACLE Study. Available at: https://pynnaclestudy.com/. Accessed November 2026; 10. Dumble M, et al. *Cancer Res*. 2021;81(13_Suppl):Abstract LB006.

PYNNACLE: Pivotal Phase 2 study design assessing rezatapopt

PYNNACLE (NCT04585750): Pivotal Phase 2, global, multi-cohort clinical trial assessing rezatapopt in patients with solid tumors with a *TP53* Y220C mutation and *KRAS* wild-type[1,2]

Key eligibility criteria

- Aged ≥12 years[a,b]
- Locally advanced or metastatic solid tumors, excluding primary CNS tumors
- Documented somatic *TP53* Y220C mutation and *KRAS* wild-type[c]
- Prior standard therapy or ineligible for SoC

Basket N≈200

Patient cohorts defined by tumor type

Rezatapopt 2000 mg QD with food

- Cohort 1: Ovarian cancer (platinum-resistant/refractory)
- Cohort 2: Lung cancer
- Cohort 3: Breast cancer
- Cohort 4: Endometrial cancer
- Cohort 5: All other solid tumors

Endpoints

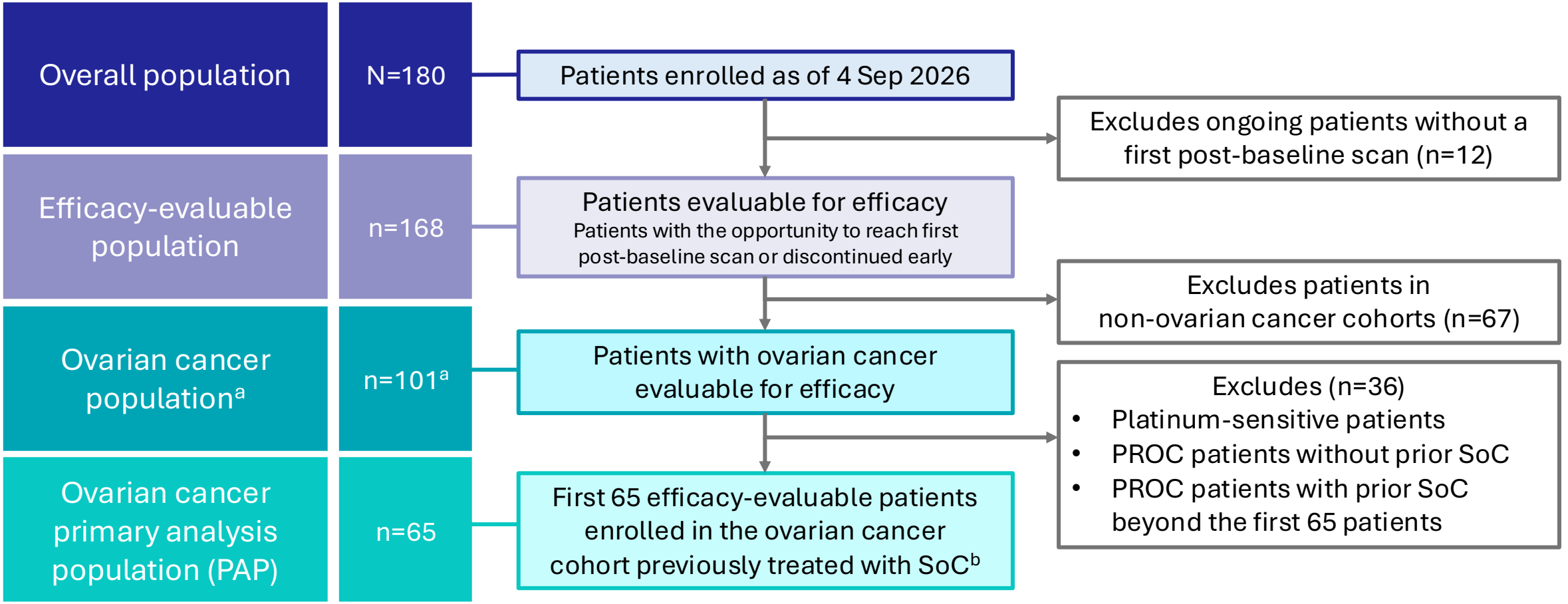
Primary: ORR (RECIST v1.1) per BICR across cohorts and in the ovarian cancer cohort

Key secondary: ORR per investigator, TTR, DoR, DCR, PFS per BICR and investigator, OS, safety

Exploratory: Changes in ctDNA dynamics and mutational/genomic profile (e.g., *TP53* Y220C, other *TP53* mutations, non-*TP53* mutations, known oncogenic drivers) before and after treatment

[a] Adults aged ≥18 years for all global sites except Singapore (must be ≥21 years of age) and the Republic of Korea (must be ≥19years of age). [b] Adolescents aged 12–17 years if weighing ≥40 kg (in Australia, the Republic of Korea [12–18 years of age] and the USA only). [c] Test results confirmed from tissue analysis; exception: ovarian cancer patients with a known *KRAS* single nucleotide variant mutation are permitted. BICR, blinded independent central review; CNS, central nervous system; ctDNA, circulating tumor DNA; DCR, disease control rate; DoR, duration of response; ORR, overall response rate; OS, overall survival; PFS, progression-free survival; QD, once daily; RECIST, Response Evaluation Criteria in Solid Tumors; SoC, standard of care; TTR, time to response. 1. ClinicalTrials.gov. Available at: https://clinicaltrials.gov/study/NCT04585750. Accessed November 2026; 2. PYNNACLE Study. Available at: https://pynnaclestudy.com/. Accessed November 2026.

Analysis populations

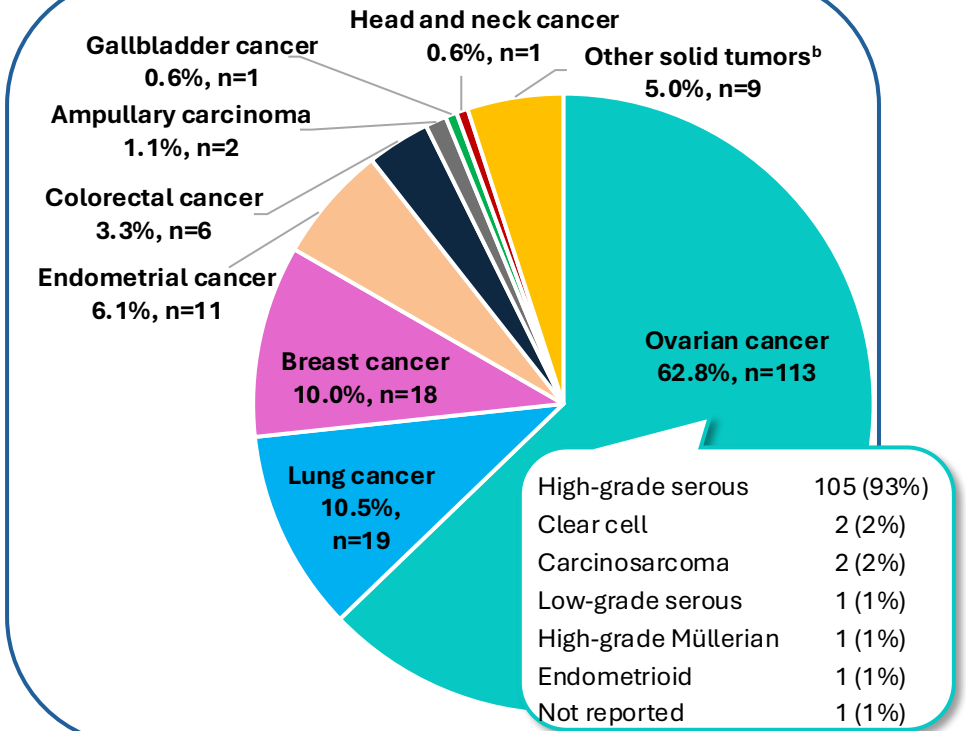


[a] In total, 113 patients with ovarian cancer were enrolled; 101 patients with ovarian cancer were efficacy evaluable (includes patients with first post-baseline tumor assessment and patients who discontinued early). [b] As defined by the protocol. Seamless adaptive clinical trial design: patient enrollment continues beyond the initial 65 patients with ovarian cancer for the primary analysis to collect supportive data. PROC, platinum-resistant/refractory ovarian cancer; SoC, standard of care.

Patient demographics and baseline characteristics

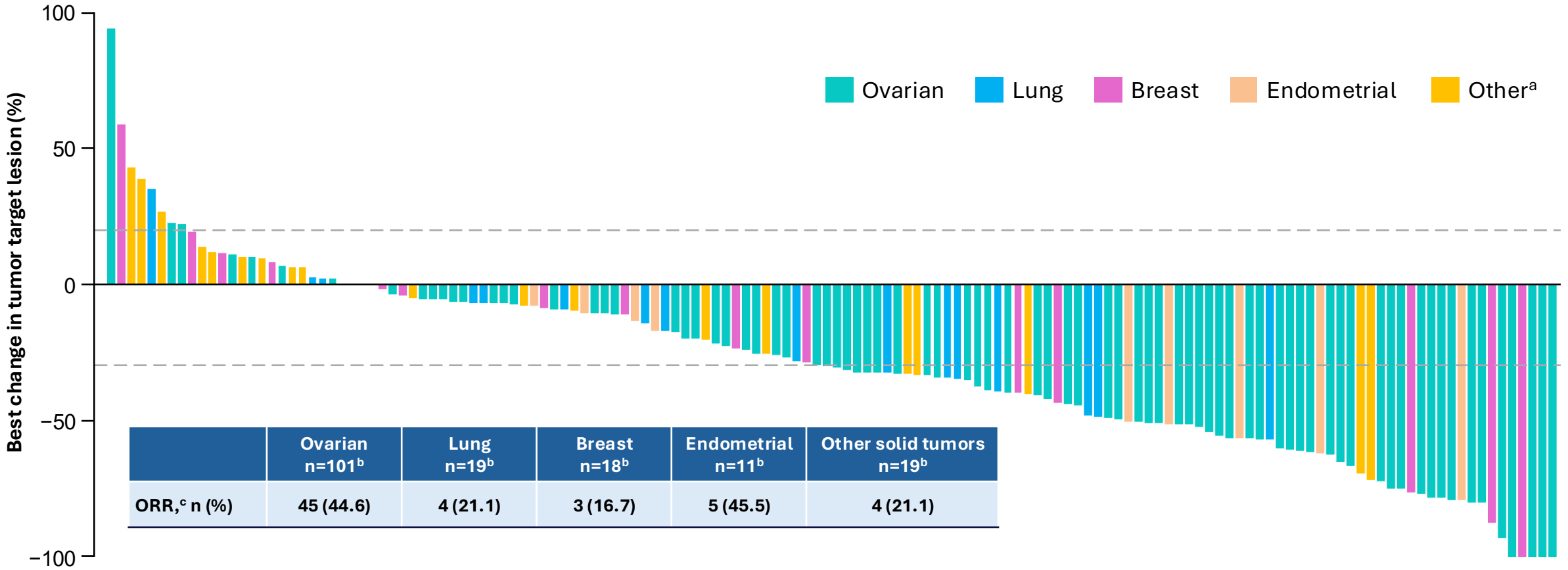
	Overall population N=180	Ovarian cancer n=113	Ovarian cancer PAP n=65
Median age, years (min–max)	65 (33–91)	66 (33–91)	65 (46–84)
ECOG status,[a] n (%)	n=179		
0/1	76 (42)/103 (58)	50 (44)/63 (56)	33 (51)/32 (49)
Platinum status, n (%)			
Sensitive	N/A	3 (3)	N/A
Resistant	N/A	71 (63)	44 (68)
Refractory	N/A	39 (35)	21 (32)
Primary platinum refractory	N/A	9 (8)	5 (8)
Prior lines of systemic therapy, n (%)	n=168		
1/2	24 (14)/37 (22)	9 (8)/21 (19)	1 (2)/6 (9)
≥3	102 (61)	83 (73)	58 (89)
Median (min–max)	3 (0–9)	4 (1–9)	4 (1–9)
Prior therapies, n (%)			
Bevacizumab	N/A	93 (82)	59 (91)
PARPi	N/A	59 (52)	43 (66)
Mirvetuximab soravtansine	N/A	26 (23)	17 (26)
HRD+, n (%)	N/A	n=105 41 (39)	n=63 27 (43)
Somatic *BRCA* 1/2 mutation,[a] n (%)	N/A	13 (12)	5 (8)
FOLR1+,[a] n (%)	N/A	n=95 46 (48)	n=56 25 (45)
PD-L1 CPS+,[a] n (%)	N/A	n=72 28 (39)	n=49 18 (37)
***CCNE1* amplification,[a] n (%)**	N/A	n=90 17 (19)	n=57 12 (21)

Patients were heavily pretreated across a broad spectrum of tumor types



Data cutoff: 4 Sep 2026. [a] Status was reported post data cutoff. [b] Includes gastric cancer (n=2), sarcoma (n=2), small intestine cancer (n=1), hepatocellular carcinoma (n=1), pancreatic cancer (n=1), thymic carcinoma (n=1) and esophageal carcinoma (n=1). *BRCA*, breast cancer gene; *CCNE1*, cyclin E1; ECOG, Eastern Cooperative Oncology Group; FOLR1, folate receptor 1; HRD, homologous recombination deficiency; PAP, primary analysis population; PARPi, poly (ADP-ribose) polymerase inhibitor; PD-L1 CPS, programmed death-ligand 1 combined positive score.

Deep tumor shrinkage observed across tumor types following treatment with rezatapopt – overall population



	Ovarian n=101[b]	Lung n=19[b]	Breast n=18[b]	Endometrial n=11[b]	Other solid tumors n=19[b]
ORR,[c] n (%)	45 (44.6)	4 (21.1)	3 (16.7)	5 (45.5)	4 (21.1)

Data cutoff: 4 Sep 2026. n=144 patients with a post-baseline assessment. [a] Responses observed in head and neck cancer, colorectal cancer, gallbladder cancer and ampullary carcinoma. [b] Efficacy-evaluable population (n=168) includes patients with first post-baseline tumor assessment and who discontinued early. [c] ORR is calculated using CR, PR and uPR. CR, complete response; ORR, overall response rate; PR, partial response; uPR, unconfirmed partial response.

Clinically meaningful response rates observed across tumor types and ovarian cancer populations

Investigator-assessed ORR per RECIST v1.1

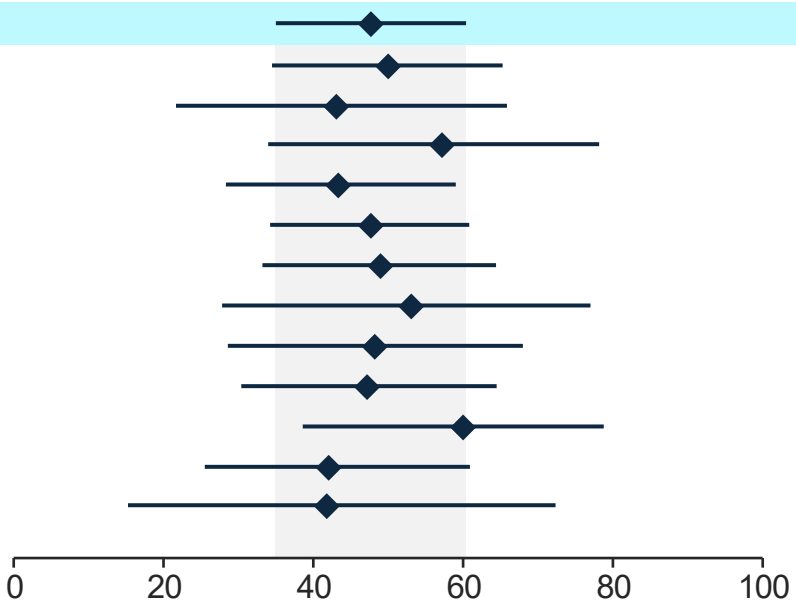
Patients, n (%)	Overall population n=168[a]	Ovarian cancer n=101[a]	Ovarian cancer PAP n=65[a]
ORR,[b]	**61 (36.3)**	**45 (44.6)**	**31 (47.7)**
(95% CI)	29.0-44.1	34.7-54.8	35.1-60.5
CR	4 (2.4)	4 (4.0)	1 (1.5)
PR	53 (31.5)	37[c] (36.6)	30 (46.2)
uPR	4 (2.4)	4 (4.0)	–
SD	69 (41.1)	36 (35.6)	23 (35.4)
Disease control rate[d]	**130 (77.4)**	**81 (80.2)**	**54 (83.1)**

Data cutoff: 4 Sep 2026. [a] Efficacy-evaluable population includes patients with first post-baseline tumor assessment and patients who discontinued early. [b] ORR is calculated using CR, PR and uPR. [c] Post data cutoff 1 patient with a PR subsequently converted to a uCR. [d] Disease control rate is calculated using CR, PR, uPR and SD. CI, confidence interval; CR, complete response; ORR, overall response rate; PAP, primary analysis population; PR, partial response; SD, stable disease; uCR, unconfirmed complete response; uPR, unconfirmed partial response.

Consistent response rates observed across subgroups in the ovarian cancer PAP

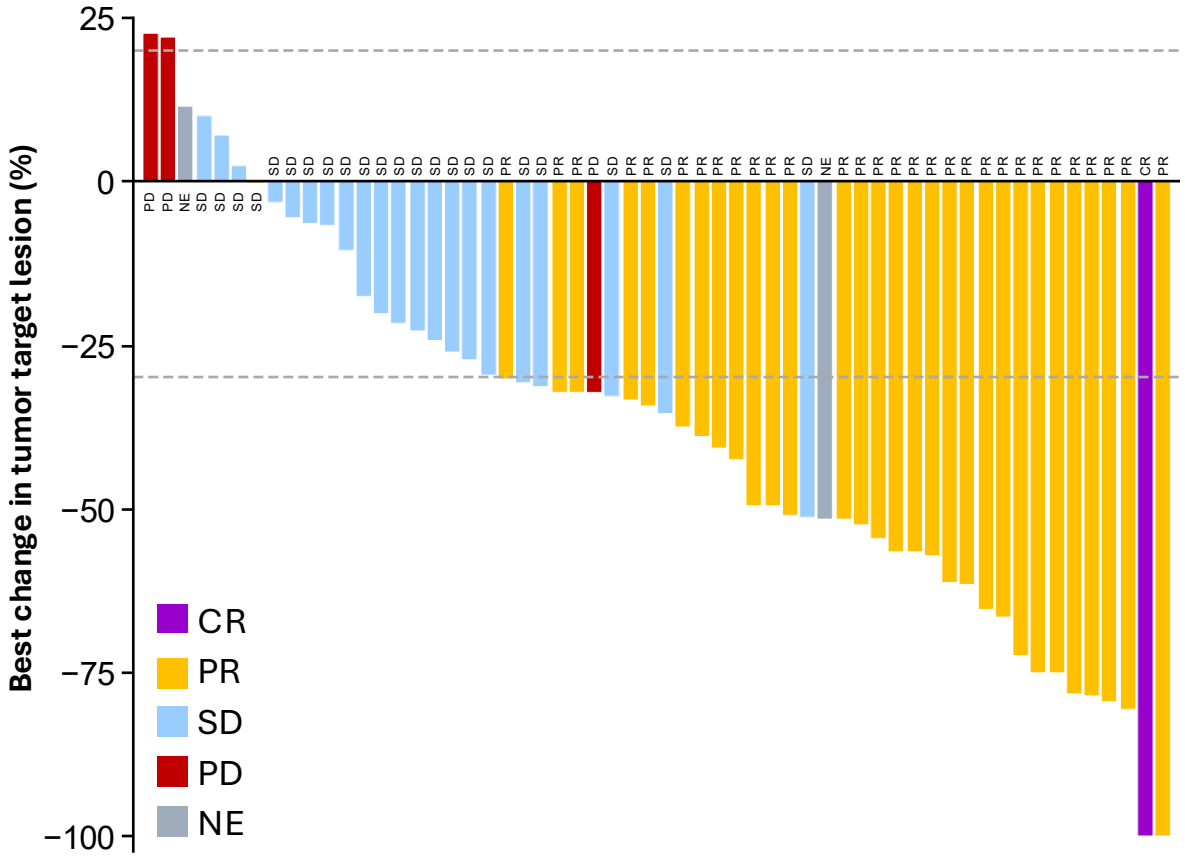
Investigator-assessed ORR[a] per RECIST v1.1 by subgroup

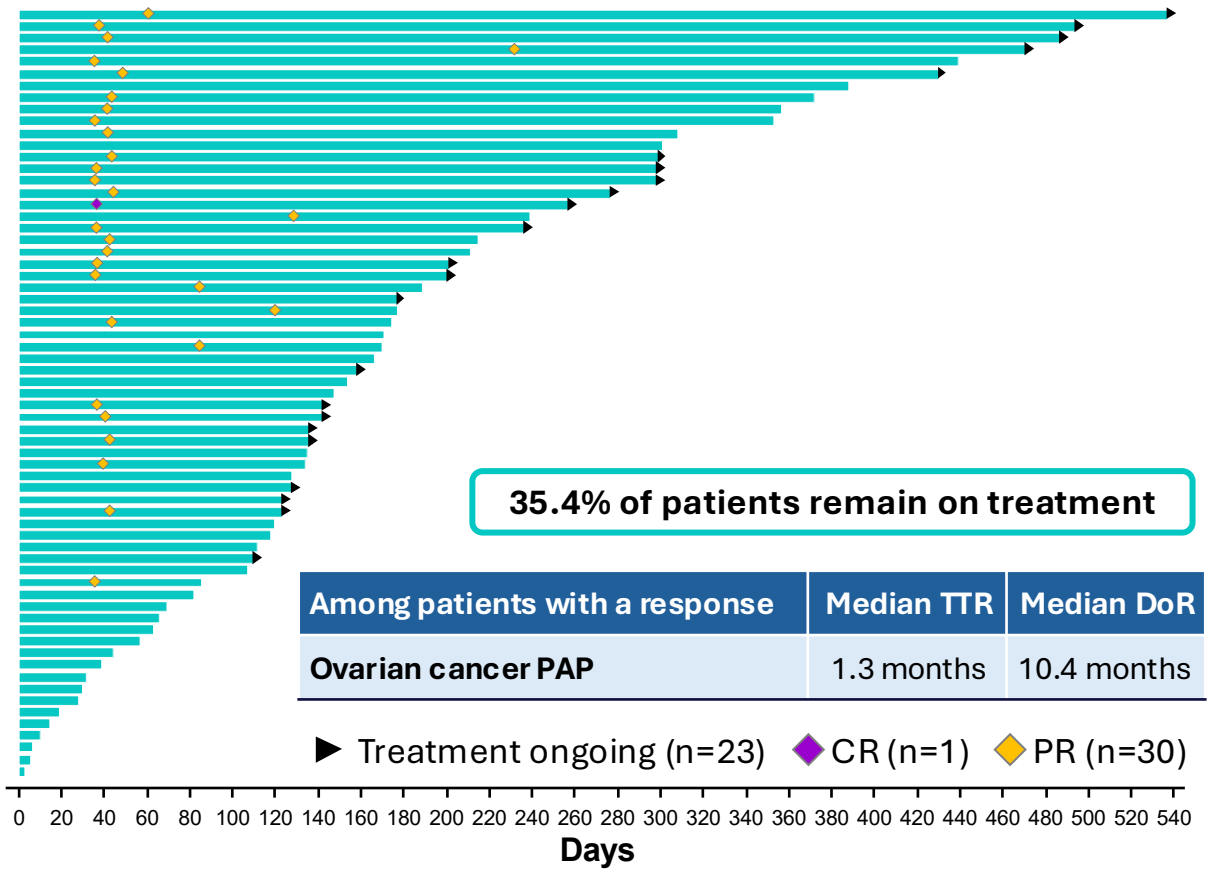
Subgroup	Responders/n	ORR, % (95% CI)
Ovarian cancer PAP[b]	**31/65**	**47.7 (35.1–60.5)**
Platinum resistant	22/44	50.0 (34.6–65.4)
Platinum refractory	9/21	42.9 (21.8–66.0)
1–3 prior lines of therapy	12/21	57.1 (34.0–78.2)
>3 prior lines of therapy	19/44	43.2 (28.3–59.0)
Prior bevacizumab	28/59	47.5 (34.3–60.9)
Prior PARPi	21/43	48.8 (33.3–64.5)
Prior mirvetuximab soravtansine	9/17	52.9 (27.8–77.0)
HRD+	13/27	48.1 (28.7–68.1)
HRD−	17/36	47.2 (30.4–64.5)
FOLR1+	15/25	60.0 (38.7–78.9)
FOLR1−	13/31	41.9 (25.5–60.9)
CCNE1 amplification	5/12	41.7 (15.2–72.3)



Exploratory subgroup analyses – data cutoff: 4 Sep 2026. [a] ORR is calculated using CR, PR and uPR. [b] Same efficacy-evaluable population as the previous slide – efficacy-evaluable population includes patients with first post-baseline tumor assessment and patients who discontinued early. *CCNE1*, cyclin E1; CI, confidence interval; CR, complete response; FOLR1, folate receptor 1; HRD, homologous recombination deficiency; ORR, overall response rate; PAP, primary analysis population; PARPi, poly (ADP-ribose) polymerase inhibitor; PR, partial response; uPR, unconfirmed partial response.

Deep tumor shrinkage with rapid and durable responses in the ovarian cancer PAP





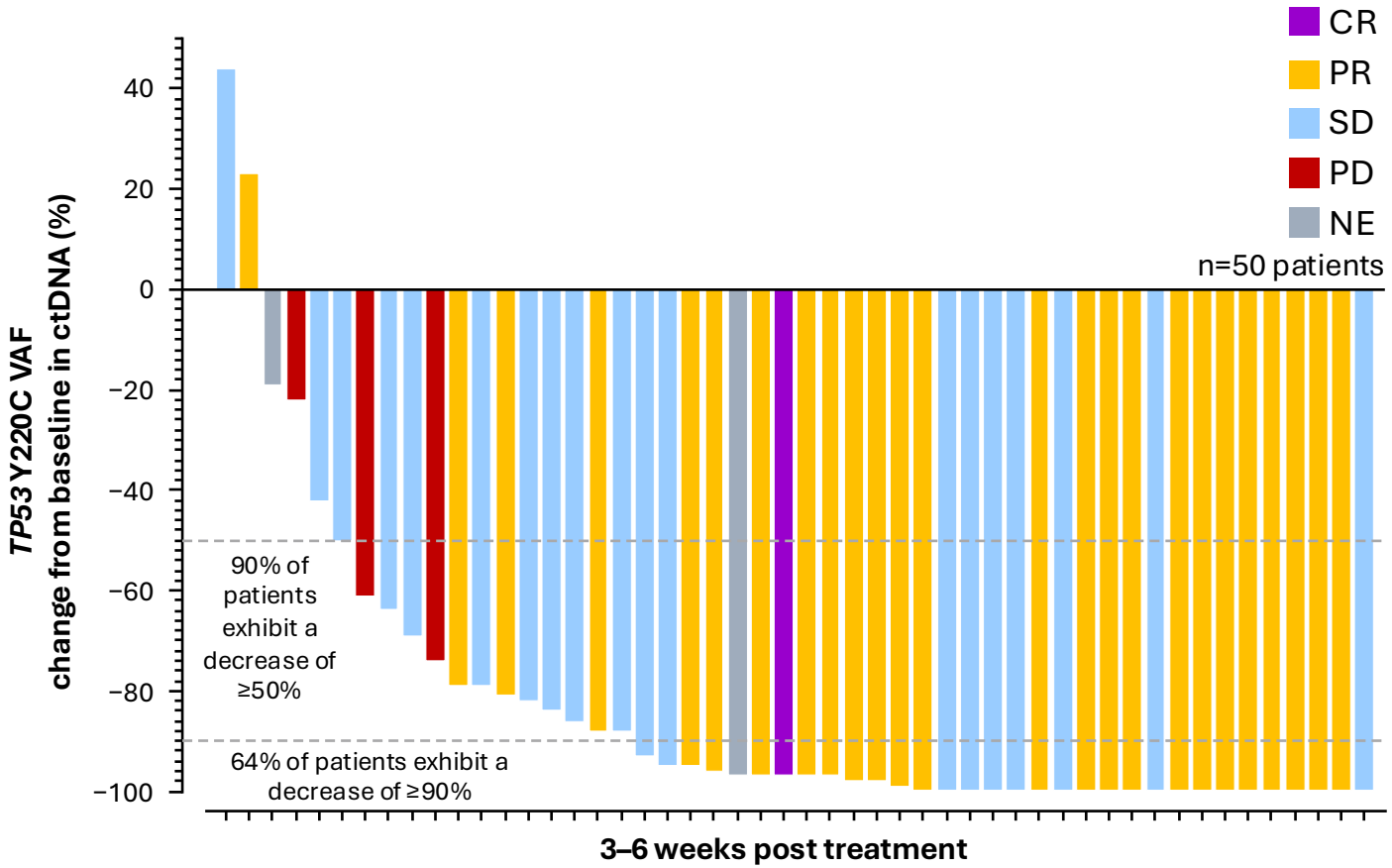
35.4% of patients remain on treatment

Among patients with a response	Median TTR	Median DoR
Ovarian cancer PAP	1.3 months	10.4 months

▶ Treatment ongoing (n=23) ◆ CR (n=1) ◆ PR (n=30)

Data cutoff: 4 Sep 2026. [a] n=58 patients with a post-baseline assessment.
CR, complete response; DoR, duration of response; NE, non-evaluable; PAP, primary analysis population; PD, progressive disease; PR, partial response; SD, stable disease; TTR, time to response.

Rezatapopt on-target activity in the ovarian cancer PAP was supported by decreases in ctDNA *TP53* Y220C VAF

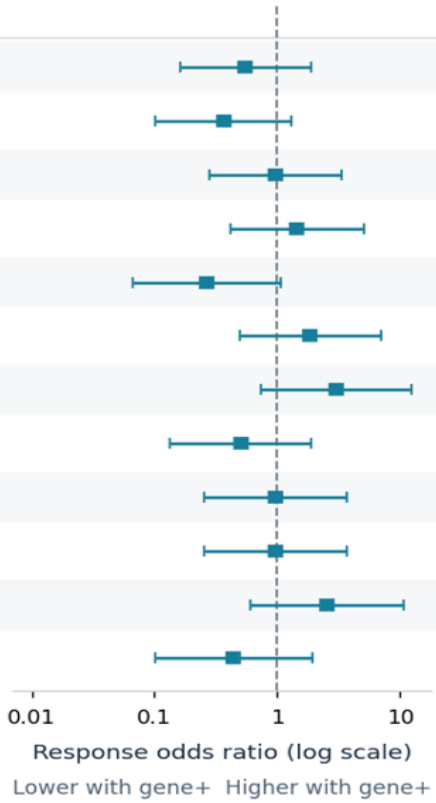


- 50 patients in the ovarian cancer PAP had available ctDNA *TP53* Y220C VAF data at baseline and following treatment (3–6 weeks)
- All patients except one experiencing a response had a reduction in *TP53* Y220C VAF
- 90% of patients achieved ≥50% reduction in ctDNA *TP53* Y220C VAF
- 64% of patients achieved ≥90% reduction in ctDNA *TP53* Y220C VAF

Data cutoff: 4 Sep 2026. CR, complete response; ctDNA, circulating tumor DNA; NE, non-evaluable; PAP, primary analysis population; PD, progressive disease; PR, partial response; SD, stable disease; VAF, variant allele frequency.

In ovarian cancer, responses occurred regardless of co-occurring genomic alterations at baseline

Gene[a]	Response[a]	OR (95% CI)[b]	P value[c]	q[d]
PRKCI	5/13	0.55 (0.16–1.87)	0.375	0.643
TERC	4/13	0.37 (0.10–1.32)	0.137	0.643
CCNE1	6/12	0.97 (0.28–3.33)	1.000	1.000
MYC	7/12	1.45 (0.41–5.04)	0.754	1.000
NF1	3/12	0.27 (0.07–1.08)	0.065	0.643
MAP3K1	7/11	1.86 (0.50–6.99)	0.516	0.774
NOTCH3	8/11	3.02 (0.73–12.44)	0.191	0.643
ATR	4/11	0.50 (0.13–1.89)	0.346	0.643
ARID1A	5/10	0.97 (0.26–3.67)	1.000	1.000
BCL2L1	5/10	0.97 (0.26–3.67)	1.000	1.000
ROS1	7/10	2.56 (0.61–10.77)	0.309	0.643
PIK3CA	3/9	0.44 (0.10–1.92)	0.309	0.643

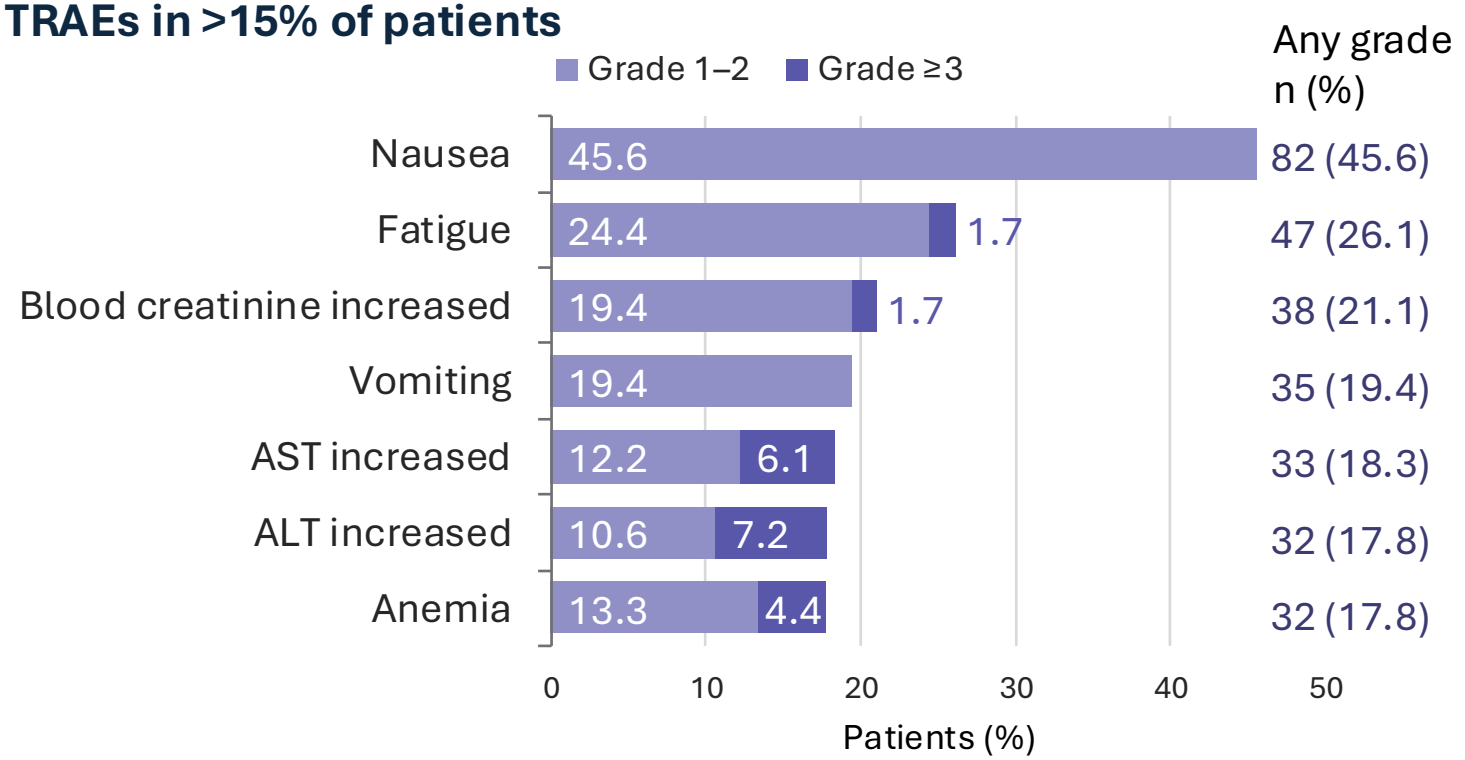


- 75 patients with ovarian cancer had tumor genomic profiling and response data available
- No association between co-occurring alterations at baseline and response
- Four patients had additional co-occurring *TP53* mutations
  - CR (n=1; two additional *TP53* mutations)
  - PR (n=1); SD (n=1); PD (n=1)

Data cutoff: 4 Sep 2026. [a] Responders/gene-positive patients; gene presence counted once per patient. [b] Bars: unadjusted approximate log-Wald 95% CIs. [c] Two-sided Fisher exact P. [d] Benjamini–Hochberg false discovery rate-adjusted P value (q) across 12 tests. *ARID1A,* AT-rich interaction domain 1A; *ATR*, ATR checkpoint kinase; *BCL2L1*, BCL2 like 1; *CCNE1*, cyclin E1; CI, confidence interval; CR, complete response; *MAP3K1*, mitogen-activated protein kinase kinase kinase 1; *MYC*, MYC proto-oncogene, bHLH transcription factor; *NF1*, neurofibromin 1; *NOTCH3,* Notch receptor 3; OR, odds ratio; PD, progressive disease; *PIK3CA*, phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha; PR, partial response; *PRKCI,* protein kinase C iota; *ROS1*, ROS proto-oncogene 1, receptor tyrosine kinase; SD, stable disease; *TERC*, telomerase RNA component.

Rezatapopt displays a favorable safety and tolerability profile in the overall population

Total safety population (N=180)
TRAEs in >15% of patients



- TRAEs were mostly Grade 1/2
- Laboratory abnormalities were manageable/monitorable, with most cases being transient and reversible
- No Hy's law cases were observed
- Similar safety profile in patients with ovarian cancer
- 11 patients (6.1%) discontinued treatment due to TRAEs, including 7 (6.2%) with ovarian cancer

Data cutoff: 4 Sep 2026. Bars show the maximum CTCAE grade per patient; one patient experienceda Grade 4 TRAE of AST increased; no Grade 5 TRAEs were observed.
ALT, alanine aminotransferase; AST, aspartate aminotransferase; CTCAE, Common Terminology Criteria for Adverse Events; TRAE,treatment-related adverse event.

Case study: A heavily pretreated patient with primary platinum-refractory HGSOC achieved a partial response by Week 6

73-year-old white woman with primary platinum-refractory HGSOC

- HRD+, FOLR1−
- *CCNE1* and *FGFR1* amplification

Three lines of prior therapy

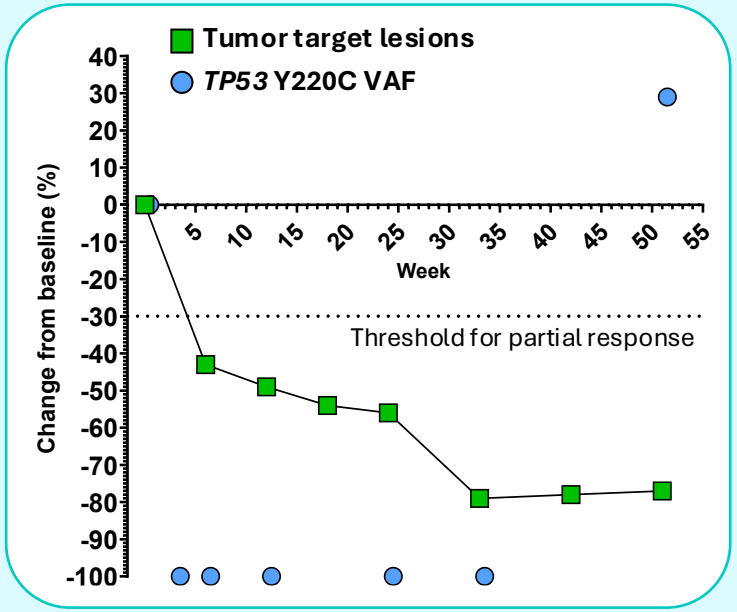
- Carboplatin/paclitaxel
- Pegylated liposomal doxorubicin
- Letrozole

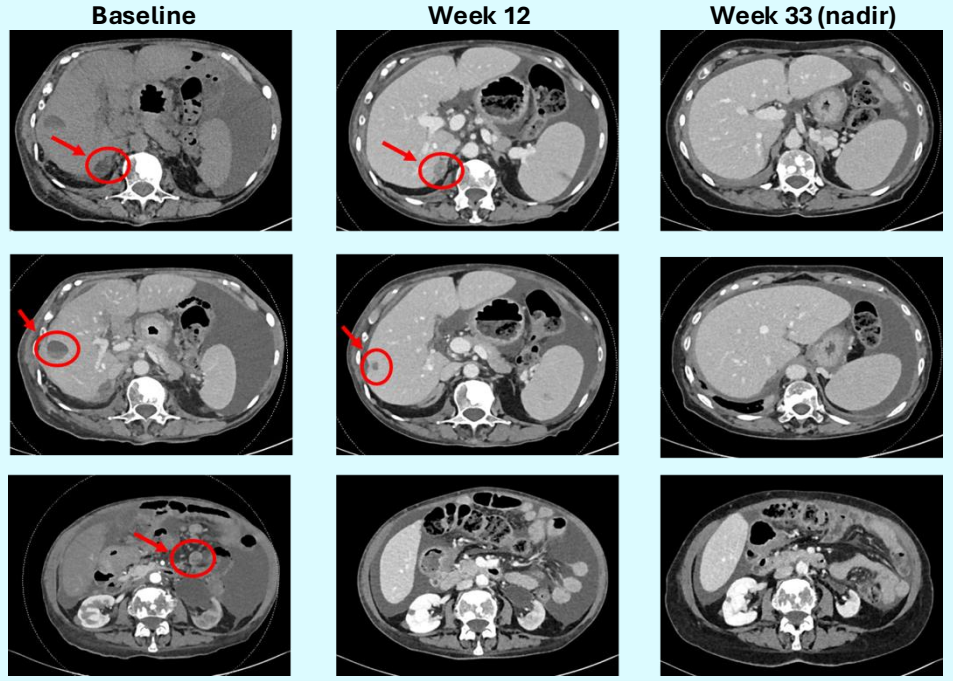
Rezatapopt 2000 mg QD

- No SAEs or AESIs
- One dose reduction to 1500 mg QD at Cycle 3 due to Grade 2 nausea
- Treatment duration: 14 months
- EOT: due to clinical progression

Response to rezatapopt

- Target tumor reduction (baseline: 107 mm) – Week 6: 61 mm (43%); Week 33 (nadir): 23 mm (78.5%)
- CA125 levels (baseline: 944 U/mL) – Week 6: 270 U/mL; Week 33: 108 U/mL; EOT: 379 U/mL
- DoR: 10.4 months; TTR: 1.6 months





Data cutoff: 4 Sep 2026. AESI, adverse event of special interest; *CCNE1*, cyclin E1; DoR, duration of response; EOT, end of treatment; *FGFR1*, fibroblast growth factor receptor 1; FOLR1, folate receptor 1; HGSOC, high-grade serous ovarian cancer; HRD, homologous recombination deficiency; QD, once daily; SAE, serious adverse event; TTR, time to response; VAF, variant allele frequency.

Case study provided courtesy of Anna Passarelli

PYNNACLE
NCT04585750

Conclusions

- Rezatapopt, a first-in-class, selective p53 Y220C reactivator, demonstrated efficacy and a manageable safety profile as an oral, chemotherapy-free monotherapy in heavily pretreated patients with *TP53* Y220C-positive solid tumors, including PROC
  - Overall population: ORR 36%
  - Ovarian cancer primary analysis population: ORR 48%
  - Mostly Grade 1/2 TRAEs; 6.1% discontinuation rate due to TRAEs
- Consistent response rates were seen across all ovarian cancer subgroups, regardless of co-occurring genomic alterations at baseline

To view the presentation please scan the QR code

ORR, overall response rate; PROC, platinum-resistant/refractory ovarian cancer; TRAE, treatment-related adverse event. https://clinicaltrials.gov/study/NCT04585750.

Acknowledgments

We would like to thank:

- All the patients, their families and caregivers who have participated, and continue to participate, in this clinical trial
- Investigators and research staff
- PPD, part of Thermo Fisher Scientific
- Foundation Medicine

This clinical trial is sponsored by PMV Pharmaceuticals, Inc.

Medical writing was provided by Gabrielle Audsley, Lucretia Ramnath and Danielle Lindley of Nucleus Global, funded by PMV Pharmaceuticals, Inc.

PYNNACLE Phase 2 clinical trial: Sites and Principal Investigators

USA
- Ikechukwu Akunyili: WellSpan York Cancer Center
- Allen Cohn: Rocky Mountain Cancer Center
- Andrew L. Coveler: University of Washington, Fred Hutch Cancer Center
- Gilberto de Lima Lopes: University of Miami - Sylvester Comprehensive Cancer Center
- Ecaterina E. Dumbrava: The University of Texas MD Anderson Cancer Center
- Anthony El-Khoueiry: USC Norris Comprehensive Cancer Center
- Robert Holloway: AdventHealth Florida
- June Hou: Columbia University Medical Center
- Melissa Johnson: Sarah Cannon and HCA Research Institute
- Shumei Kato: UC San Diego Moores Cancer Center
- Gottfried Konecny: UCLA Jonsson Comprehensive Cancer Center
- Shivaani Kummar: Oregon Health & Science University (OHSU) Knight Cancer Institute
- Patricia LoRusso: Yale Cancer Center
- Lainie Martin: Abramson Cancer Center of The University of Pennsylvania
- Niharika Mettu: Duke University
- David Miller: UT Southwestern Simmons Cancer Center
- Jamal Misleh: Medical Oncology Hematology Consultants
- Aparna R. Parikh: Massachusetts General Hospital Cancer Center
- Ivor Percent: Florida Cancer Specialists South
- Debra Richardson: University of Oklahoma Peggy and Charles Stephenson Cancer Center
- Alison M. Schram: Memorial Sloan Kettering Cancer Center
- Geoffrey Shapiro: Dana-Farber Cancer Institute
- Dale Shepard: The Cleveland Clinic Taussig Cancer Center
- David Sommerhalder: New Experimental Therapeutics - NEXT Oncology
- Alexander Spira: Virginia Cancer Specialists (Fairfax) – USOR
- Sarah Taylor: University of Pittsburgh Medical Center
- Nataliya Uboha: University of Wisconsin Cancer Center
- Terrence Wong: Texas Oncology

France
- Veronique Debien: EDOG – Institut Bergonié – PPDS
- Xavier Durando: Centre Jean Perrin
- Lauriane Eberst: ICANS – Institut de cancérologie Strasbourg Europe
- Frédéric Fiteni: CHU de Nîmes
- Jean-Sébastien Frenel: Institut de cancérologie de l'Ouest
- Carlos-Alberto Gomez-Roca: Institut Claudius Regaud
- Aurélien Lambert: Institut de cancérologie de Lorraine
- Yohann Loriot: Institut Gustave Roussy
- Isabelle Ray-Coquard: Centre Léon Bérard Centre Régional de Lutte Contre Le Cancer

Italy
- Giuseppe Curigliano: Istituto Europeo di Oncologia, IRCCS
- Massimo Di Nicola: Fondazione IRCCS Istituto Nazionale dei Tumori
- Anna Fagotti: Fondazione Policlinico Universitario Agostino Gemelli IRCCS
- Lorenza Landi: Istituto Fisioterapici Ospitalieri - Istituto Nazionale Tumori Regina Elena
- Chiara Lazzari: Fondazione del Piemonte per l'Oncologia (IRCCS)
- Domenica Lorusso: Humanitas San Pio X
- Anna Passarelli: Istituto Nazionale Tumori IRCCS Fondazione G. Pascale
- Armando Santoro: Istituto Clinico Humanitas
- Salvatore Siena: ASST Grande Ospedale Metropolitano Niguarda

Spain
- Lorena Farinas: Instituto de Investigación Oncológica Vall d'Hebron (VHIO)
- José Alejandro Pérez Fidalgo: Hospital Clínico Universitario de Valencia
- Elena Garralda: NEXT Oncology-Hospital Universitari Quirónsalud Barcelona
- Irene Moreno: START MADRID, Hospital Universitario HM Sanchinarro
- Victor Moreno García: START MADRID, Hospital Universitario Fundación Jiménez Díaz
- Jorge Luis Ramon Patino: START Rioja
- Santiago Ponce Aix: Hospital Universitario 12 de Octubre

Australia
- Sophia Frentzas: Monash Health, Monash Medical Centre
- Peter Grimison: Chris O'Brien Lifehouse Hospital
- Michael Millward: Linear Clinical Research Ltd
- Catherine Shannon: Mater Cancer Care Centre
- Shawgi Sukumaran: Flinders Medical Centre

Republic of Korea
- Seock-Ah Im: Seoul National University Hospital
- Myong Cheol Lim: National Cancer Center
- Chel Hun Choi: Samsung Medical Center
- Dae Ho Lee: Asan Medical Center
- Jung-Yun Lee: Severance Hospital Yonsei University

Germany
- Dirk Arnold: Asklepios Klinik Altona
- Rainer Claus: Universitätsklinikum Augsburg
- Martin Haag: Nationales Centrum für Tumorerkrankungen (NCT) Heidelberg
- Marcel Wiesweg: West German Cancer Center, University Hospital Essen

Singapore
- David Shao Peng Tan: National University of Singapore (NUS), National University Hospital
- Tira J. Tan: National Cancer Centre

UK
- Elisa Fontana: Sarah Cannon Research Institute UK
- Alastair Greystoke: Freeman Hospital