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Reversing Chemoresistance in Platinum-Resistant Ovarian Cancer: The Role of Glucocorticoid-Receptor Modulation and other Emerging Therapeutic Pathways

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Disclosures

- **Thomas C Krivak, MD:** Speaker's bureau – AbbVie, Astra Zeneca, Daiichi Sankyo, Genmab, GSK, Merck, Pfizer; consultant – AbbVie, Astra Zeneca, Daiichi Sankyo, Genmab, GSK, Merck, Pfizer; research/grant support – AbbVie, GOG, GSK, Merck, Myriad

- I will be discussing unlabeled and investigational use of medications to treat ovarian cancer

Learning Objectives

- Describe the biologic mechanisms driving chemoresistance in PROC, including the role of GR signaling and other tumor-intrinsic resistance pathways
- Assess the latest clinical efficacy and safety data on emerging therapies such as GR modulation, FRα-directed ADCs, and other novel pathways in patient populations with varied biomarker profiles, prior treatments, and comorbidity considerations
- Implement toxicity-monitoring and supportive-care strategies to maintain dose intensity and optimize outcomes with therapies used in PROC

PROC = platinum-resistant ovarian cancer; GR = glucocorticoid receptor; FRα = folate receptor alpha; ADC = antibody-drug conjugate.

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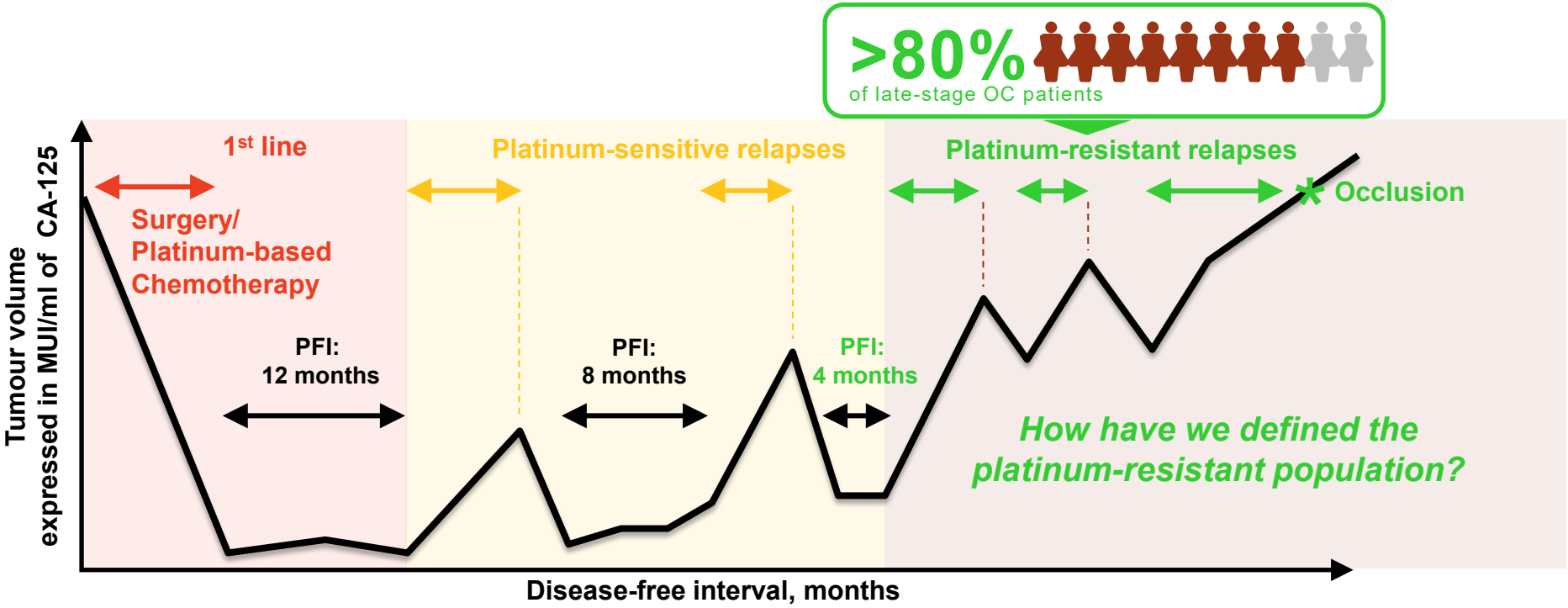
Platinum Resistant Ovarian Cancer

Defining and Treating Platinum Resistance in Pts with Advanced OC Remains an Unresolved Clinical Dilemma



CA-125 = cancer antigen 125; OC = ovarian cancer; PFI = platinum-free interval.

González-Martín A, et al. *Ann Oncol*. 2023;34(10):833-848. Salani R, et al. *Am J Obstet Gynecol*. 2011;204(6):466-478. St Laurent J, Liu JF. *J Clin Oncol*. 2024;42(2):127-133. Adapted from Giornelli GH. *Springerplus*. 2016;5(1):1197.

Platinum-Resistant Ovarian Cancer

Journal of Clinical Oncology
The Official Journal of the American Society of Clinical Oncology
Vol 10, No 4 April 1992

EDITORIAL

Responses to Salvage Chemotherapy in Ovarian Cancer: A Critical Need for Precise Definitions of the Treated Population

GYNECOLOGIC ONCOLOGY **69,** 91–92 (1998)
ARTICLE NO. GO984997

EDITORIAL

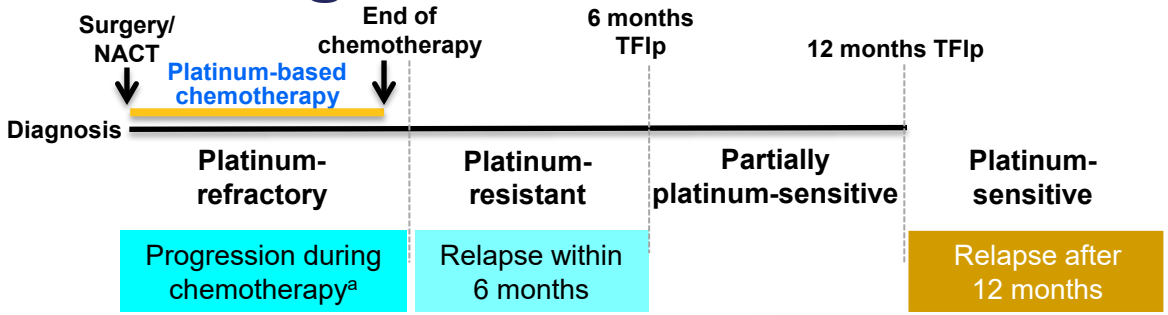
"Recurrence within 6 Months of Platinum Therapy": An Adequate Definition of "Platinum-Refractory" Ovarian Cancer?

Maurie Markman, M.D.
Cleveland Clinic Cancer Center, and Department of Hematology/Medical Oncology, Cleveland Clinic Foundation, Cleveland, Ohio 44195

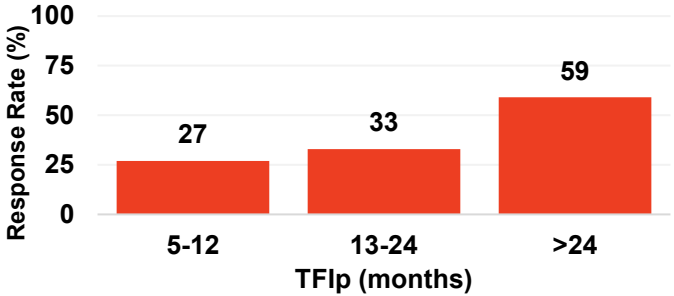
- Primary platinum-resistant
  - Patients who progress on initial therapy, who have less than a partial response, or who initially respond and then progress while on therapy

Markman M, Hoskins W. *J Clin Oncol*. 1992;10(4):513-514. Markman M. *Gynecol Oncol*. 1998;69(2):91-92.

Defining Platinum Resistance



2L response rate by treatment-free interval for platinum-based chemotherapy (TFIp) (n=82; 1991)



Response rates correlated with TFIp irrespective of platinum-containing compound

*"In reporting results from clinical trials with **strictly defined definitions of prior platinum response**, it will be far easier to identify agents that possess antineoplastic activity against a tumor-cell population different from that of the platinum compounds."*

Maurie Markman
William Hoskins, 1992

*As PROC definitions are evolving, differences may exist across local guidelines. [a]Or relapse within 1 month of completing platinum-based ctx.
NACT = neoadjuvant ctx; TFIp = treatment-free interval for platinum-based ctx.

Luvero D, et al. *Ther Adv Med Oncol*. 2014;6(5):229-239. Markman M, et al. *J Clin Oncol*. 1991;9(3):389-393. Gore ME, et al. *J Clin Oncol*. 1990;36(2):207-211. Markman M, Hoskins W. *J Clin Oncol*. 1992;19(4):513-514. Friedlander M, et al. *Int J Gynecol Cancer*. 2011;21(4):771-775.

Platinum Resistant Ovarian Cancer

- Upfront/Primary Therapy
  - Goal for prolonged remission
  - Aggressive debulking surgery &chemotherapy
  - Maintenance (treatment/maintenance) therapy
  - Potential chance of “cure”
  - Accepting of potential toxicity

- Platinum Resistant
  - Poor prognosis
  - Limited role for surgery
  - Quality of life and longevity

Surveillance/Detection Ovarian Cancer

Clinical Practice Statement

Updates to post-treatment surveillance after curative intent treatment for patients with gynecologic cancers: A Society of Gynecologic Oncology clinical practice statement

Ritu Salani [a,*], David Atallah [b], Amanda N. Fader [c], Marina Frimer [d], Andreas Obermair [e], Rene Pareja [f], Marilyn Huang [g]

HIGHLIGHTS

- Surveillance practices are designed to improve cancer related outcomes.
- Vaginal cytology should be eliminated in the surveillance of ovarian and uterine cancers.
- Emerging strategies, such as ctDNA and groin ultrasounds, may enhance surveillance practices but require additional study.

2025 SGO Epithelial Ovarian Cancer Surveillance Recommendations.

Time from completion of primary therapy	Years 0–2	Years 3–5	>5 years[b]
Symptom review	3–4 months	6 months	Yearly
Physical exam	3–6 months	6 months	Yearly
Pap test	Not recommended		
CA125, if initially elevated	3–6 months	6 months	As indicated
Radiographic imaging	Reserve for clinical indications; insufficient data to support routine use[a,b]		
ctDNA	May be useful in select cases		

[a] Consider imaging if CA125 is not initially elevated.
[b] May be followed by a gynecologic oncologist or generalist.

SGO = Society of Gynecological Oncology.
Salani R, et al. *Gynecol Oncol*. 2026;204:109-117.

Surgical and Blood-Based Minimal Residual Disease in Patients with Ovarian Cancer after First-line Therapy: Clinical Outcomes and Translational Opportunities

ABSTRACT

Purpose: Minimal residual disease (MRD) after first-line treatment of advanced-stage ovarian cancer remains a long-standing barrier to cure. We investigated the prognostic and translational value of MRD detection by second-look laparoscopy (SLL) and ctDNA at the completion of first-line therapy.

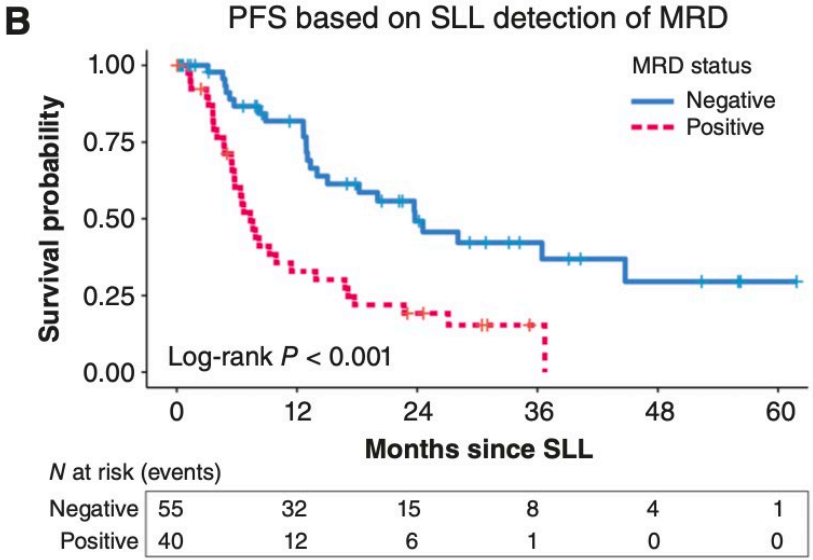
Experimental Design: Patients with high-grade epithelial ovarian cancer who had a complete clinical response to first-line therapy and underwent SLL and plasma collection for ctDNA were included. Progression-free survival (PFS) and overall survival (OS) were estimated based on MRD and clinicopathologic status. Spatial transcriptomics (GeoMx and Visium) and proteomics (CODEX) profiling were performed on serial samples from select patients.

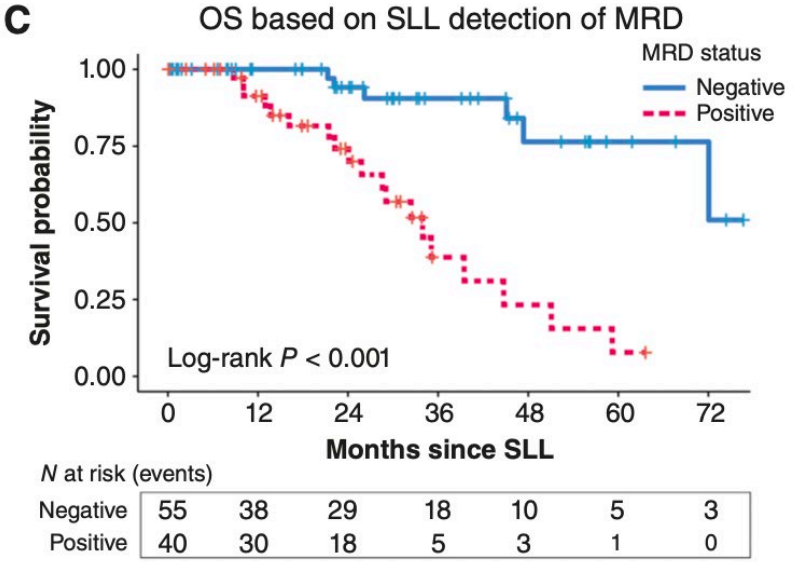
Results: Forty of 95 (42.1%) patients had surgically detected MRD, which was associated with worse PFS (median PFS 7.4 vs. 23.8 months; $P < 0.001$) and OS (median OS 33.9 vs. not reached; $P < 0.001$). SLL positivity was an independent negative prognostic factor for OS (HR, 4.40; 95% confidence interval, 1.37–14.21; $P = 0.013$) in multivariable analysis. Among 44 patients who underwent SLL and had ctDNA testing, 34% (15/44) were ctDNA-positive, which was associated with worse PFS (6.4 vs. 28.1 months; $P < 0.001$) and OS (32.4 months vs. not reached; $P = 0.008$). We demonstrated the feasibility of spatial multiomics in studying MRD and their ability to provide hypothesis-generating observations, implicating the upregulation of the hypoxia signaling pathway, expression of multiple druggable targets (*CDK6*, *GLS*, *MSLN*, *ERBB2*), and immune exclusion in MRD lesions.

Conclusions: Approximately half of patients in clinical remission after first-line therapy have assessable MRD, which can inform prognosis, therapeutic target discovery, and clinical trials.

Knisely A, et al. *Clin Cancer Res*. 2025;31(19):4122-4135.

Patient Outcomes: Second Look Laparoscopy

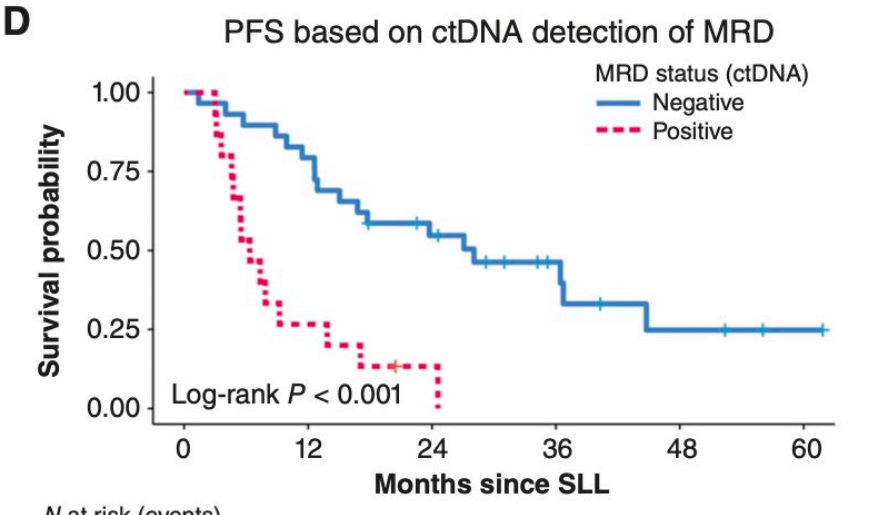


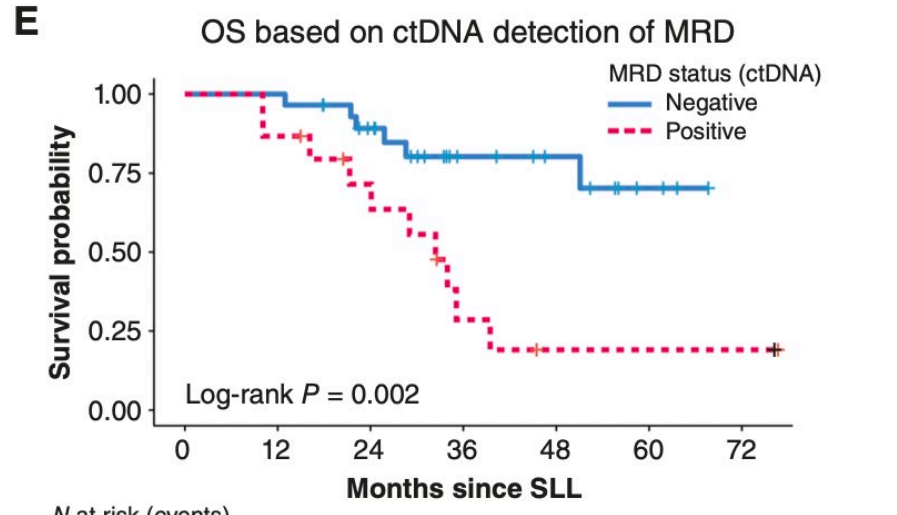


PFS = progression-free survival; OS = overall survival; SLL = small lymphocytic lymphoma.

Knisely A, et al. *Clin Cancer Res*. 2025;31(19):4122-4135.

Patient Outcomes: ctDNA





Knisely A, et al. *Clin Cancer Res*. 2025;31(19):4122-4135.

Second Look & ctDNA- Summary

- Positive SLL & ctDNA
  - Poor PFS
  - ctCDA worse PFS
- Small study
- SLL pos- approximately 50%ctDNA pos
- Serial ctDNA- show improved detection
- High positive ctDNA & SLL
  - Need better upfront therapy
  - Maintenance strategies

Platinum Resistant Ovarian Cancer Treatment Options

PRINCIPLES OF SYSTEMIC THERAPY

Acceptable Recurrence Therapies for Epithelial Ovarian (including LCOC)[o]/Fallopian Tube/Primary Peritoneal Cancer

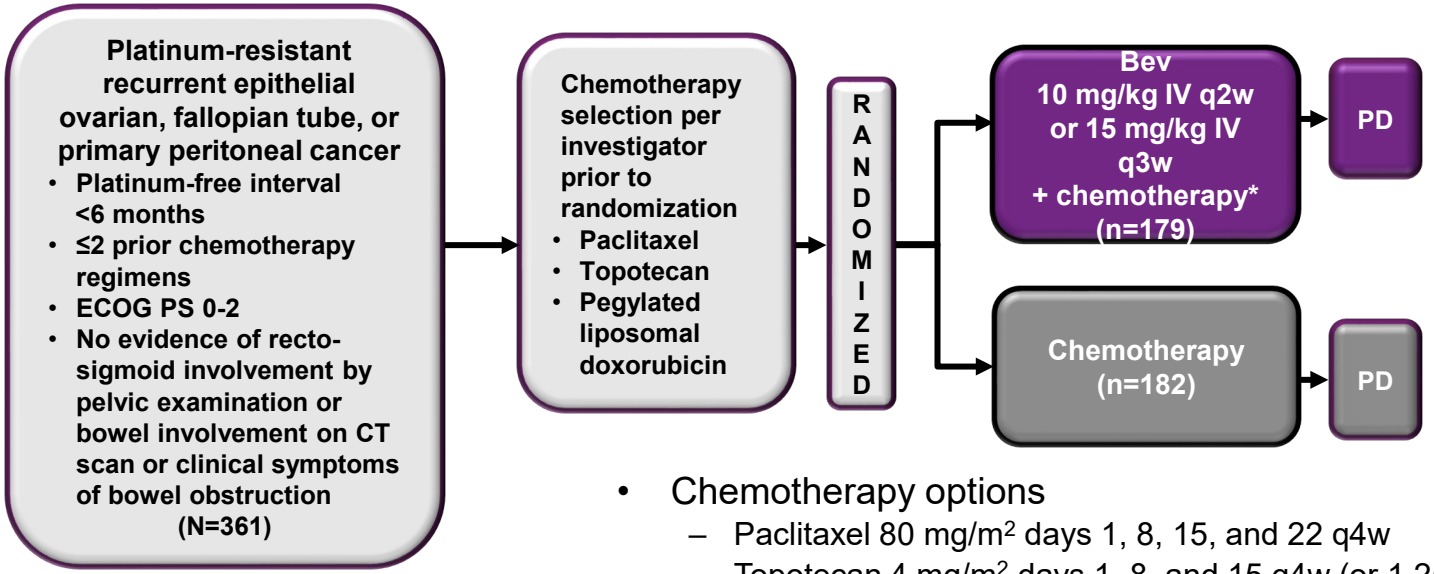
Recurrence Therapy for Platinum-Resistant Disease (alphabetical order)

Preferred Regimens	Other Recommended Regimens	Useful in Certain Circumstances
Cytotoxic Therapy Cyclophosphamide (oral)/ bevacizumab[q,46] Docetaxel[47] Etoposide (oral)[48] Gemcitabine[49,50] Liposomal doxorubicin[49,50] Liposomal doxorubicin/ bevacizumab[q,51] Paclitaxel (weekly)[g,52] Paclitaxel (weekly)/ bevacizumab[g,q,51] Topotecan[53,54] Topotecan/bevacizumab[q,51] Targeted Therapy (single agents) Bevacizumab[q,21,22] Mirvetuximab soravtansine-gynx (for FRα-expressing tumors [≥75% positive tumor cells])(category 1)[x,55,56]	Cytotoxic Therapy[s] Capecitabine Carboplatin[*] Carboplatin/docetaxel[*] Carboplatin/paclitaxel (weekly)[g,*] Carboplatin/gemcitabine[14] ± bevacizumab[q,r,15,*] Carboplatin/liposomal doxorubicin[16] ± bevacizumab[q,17,*] Carboplatin/paclitaxel[g,18] ± bevacizumab[q,r,19,*] Cyclophosphamide Cyclophosphamide (oral)/pembrolizumab/bevacizumab[58,59] Doxorubicin Gemcitabine/bevacizumab[60] Gemcitabine/cisplatin[20,*] Ifosfamide Irinotecan Ixabepilone/bevacizumab (category 2B)[z,61] Melphalan Oxaliplatin Paclitaxel Paclitaxel, albumin bound Pemetrexed Sorafenib/topotecan[57] Vinorelbine Targeted Therapy (single agents) Niraparib (category 3)[t,27] Olaparib (category 3)[u,28] Pazopanib (category 2B)[29] Rucaparib (category 3)[v,30] Hormone Therapy Aromatase inhibitors (anastrozole, exemestane, letrozole) Goserelin acetate Leuprolide acetate Megestrol acetate Tamoxifen[j]	Carboplatin/paclitaxel (for age >70)[g,w,*] Carboplatin/paclitaxel, albumin bound (for confirmed taxane hypersensitivity)[*] Immunotherapy[x] Dostarlimab-gxly (for dMMR/MSI-H recurrent or advanced tumors)[43] Pembrolizumab (for patients with MSI-H or dMMR solid tumors, or TMB-H tumors ≥10 mutations/megabase)[44] Hormone Therapy Fulvestrant (for low-grade serous carcinoma) Targeted Therapy[x] Dabrafenib + trametinib (for *BRAF* V600E-positive tumors)[32] Entrectinib[33] or larotrectinib[34] or repotrectinib[35] (for *NTRK* gene fusion-positive tumors) Fam-trastuzumab deruxtecan-nxki (for HER2-positive tumors [IHC 3+ or 2+])[36] Mirvetuximab soravtansine-gynx/bevacizumab (for FRα-expressing tumors [≥25% positive tumor cells])[q,38,62,63] Selpercatinib (for *RET* gene fusion-positive tumors)[39] For low-grade serous carcinoma: • Avutometinib/defactinib (for KRAS-mutated tumors)[40] • Trametinib[41] • Binimetinib (category 2B)[42,43] For mucinous carcinoma: • FOLFIRI ± bevacizumab (category 2B)[64-67]

* Platinum agents have limited activity when the disease has demonstrated growth through a platinum-based regimen, and platinum rechallenge is generally not recommended in this setting.

Footnotes on OV-C 9A of 12

Aurelia: Randomized Phase 3 Trial: Bev + Ctx: PROC

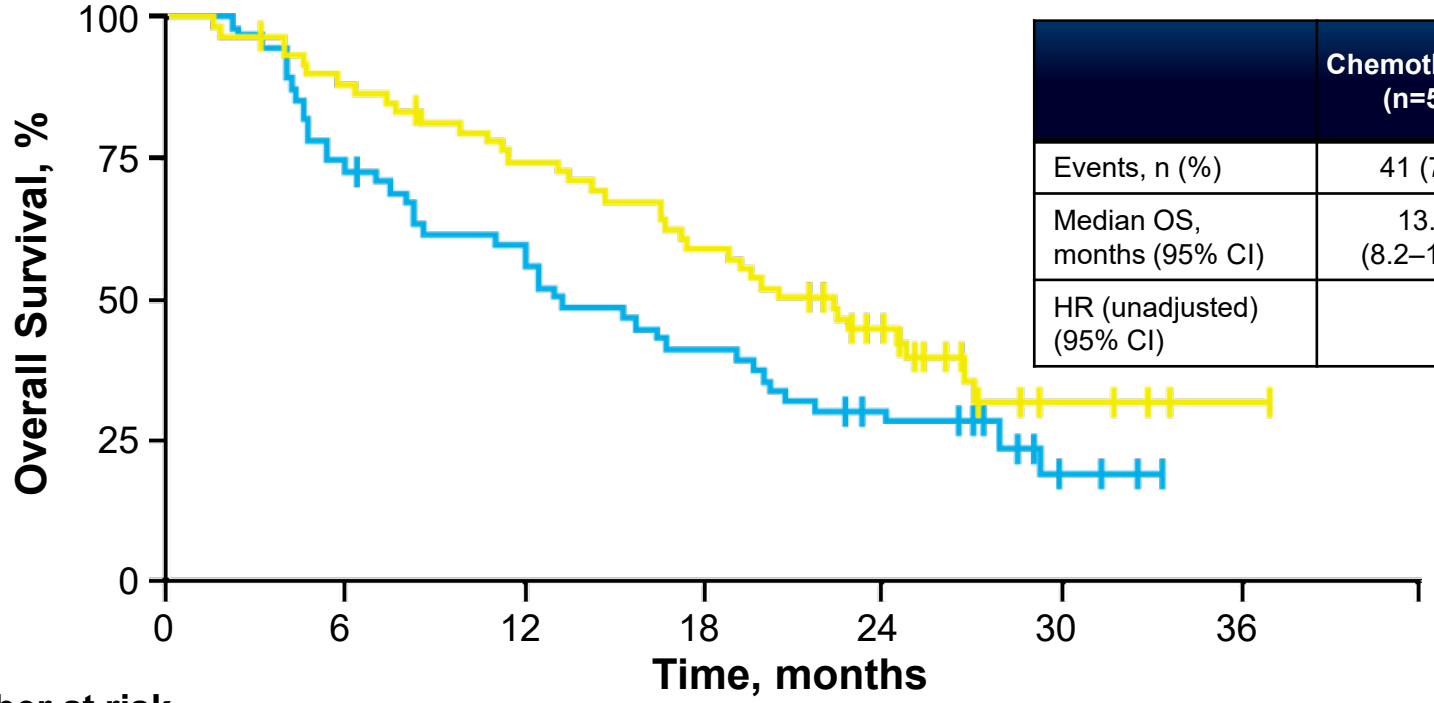


- Chemotherapy options
  - Paclitaxel 80 mg/m^2 days 1, 8, 15, and 22 q4w
  - Topotecan 4 mg/m^2 days 1, 8, and 15 q4w (or 1.25 mg/m^2 days 1-5 q3w)
  - Pegylated liposomal doxorubicin 40 mg/m^2 day 1 q4w
- **Main outcome measure:** PFS
- **Secondary outcome measures:** OS, ORR

ORR = overall response rate; ECOG PS = Eastern Cooperative Oncology Group performance status; PD = progressive disease.

Pujade-Lauraine E, et al. *J Clin Oncol*. 2014;32(13):1302-8.

Paclitaxel Cohort



	Chemotherapy (n=55)	Bev + Chemotherapy (n=60)
Events, n (%)	41 (75)	36 (60)
Median OS, months (95% CI)	13.2 (8.2–19.7)	22.4 (16.7–26.7)
HR (unadjusted) (95% CI)	**0.65** (0.42–1.02)	

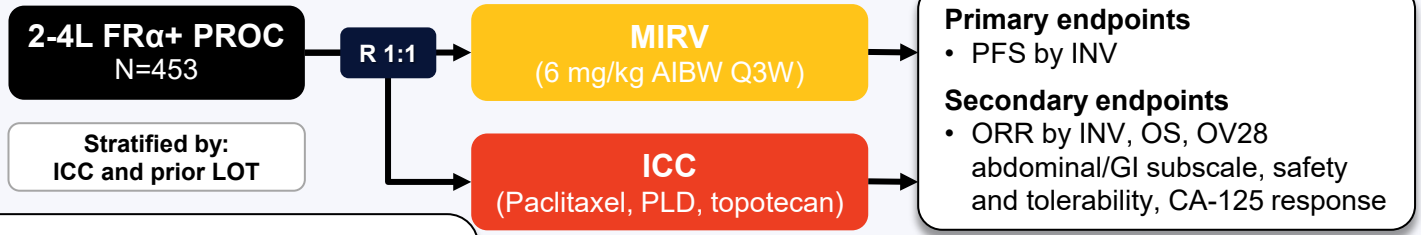
Number at risk

CT	55	40	32	22	13	3	0
Bev + CT	60	52	43	34	19	4	1

Witteveen P, et al. *Eur J Cancer*. 2013;49(Suppl. 3):S3.

MIRV: MIRASOL Trial Overview

MIRASOL: Phase III, global, randomized, open-label trial of MIRV vs IC chemotherapy in patients with FRα-high PROC



Key inclusion criteria:
- HGS FRα+ (≥75% TC; ≥2+ staining) PROC
- 1-3 prior LOT
- Prior BEV and PARPi allowed

Key exclusion criteria:
- Active or chronic ocular conditions
- Prior MIRV or other FRα-targeting agents

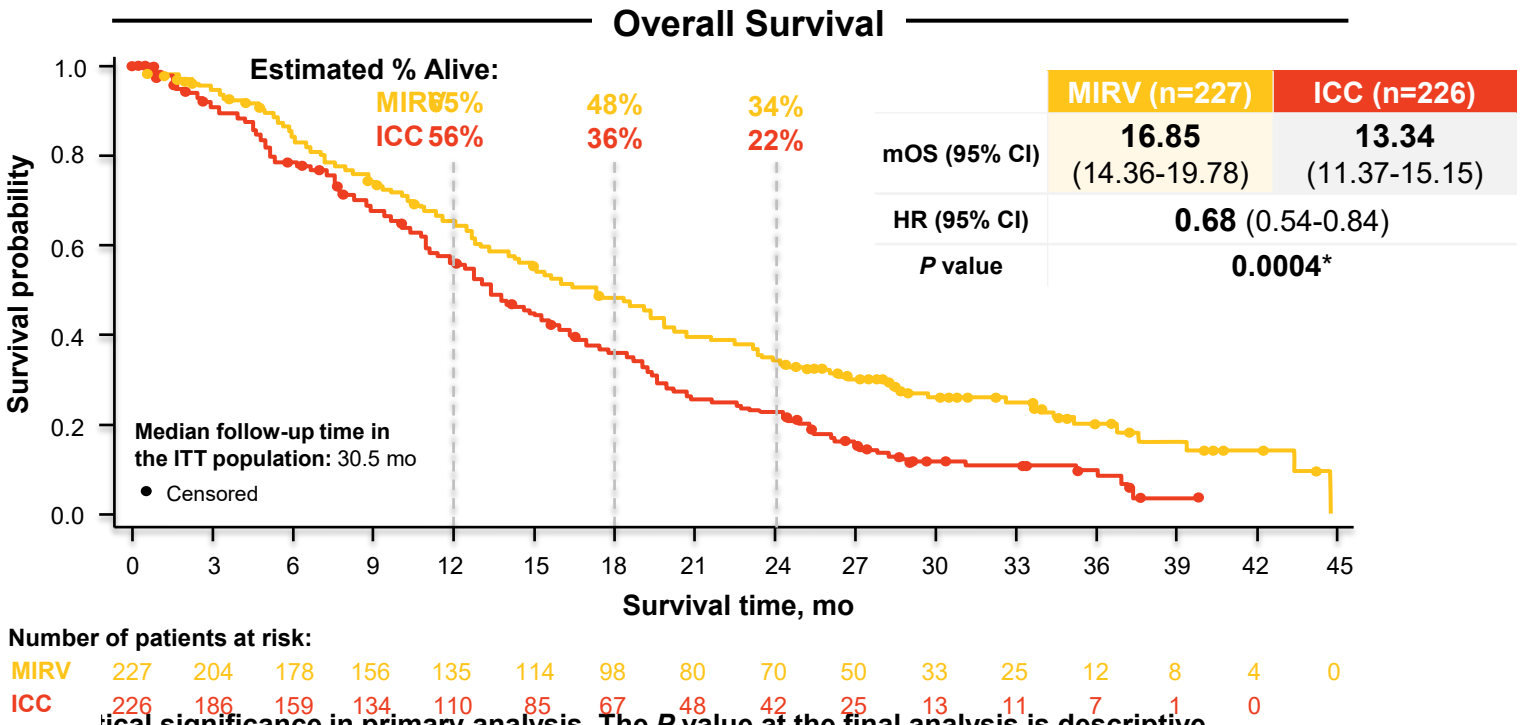
214 sites
21 countries

Lead Investigators: Dr. Kathleen N. Moore, University of Oklahoma College of Medicine, USA; Dr. Toon Van Gorp, University of Leuven, Belgium.

2-4L = on second-fourth line; AIBW = adjusted ideal body weight; GI, gastrointestinal; HGS = high-grade serous (histology); ICC = investigator's choice chemotherapy; INV = investigator; LoT = line of therapy; MIRV = mirvetuximab soravtansine; OV28 = European Organisation for Research and Treatment of Cancer Quality of Life Questionnaire, 28-item Ovarian Cancer Module; PLD = pegylated liposomal doxorubicin; TC = tumor cells.

Van Gorp T, et al. Presented at: 2025 SGO Annual Meeting on Women's Cancer; March 14-17, 2025; Seattle, WA. 939696.

MIRASOL Trial: Efficacy



	MIRV (n=227)	ICC (n=226)
mOS (95% CI)	**16.85** (14.36-19.78)	**13.34** (11.37-15.15)
HR (95% CI)	**0.68** (0.54-0.84)	
P value	**0.0004***	

***OS reached statistical significance in primary analysis. The *P* value at the final analysis is descriptive.**

HR = hazard ratio; ICC = investigator's choice chemotherapy; ITT = intend-to-treat; mOS = median OS.

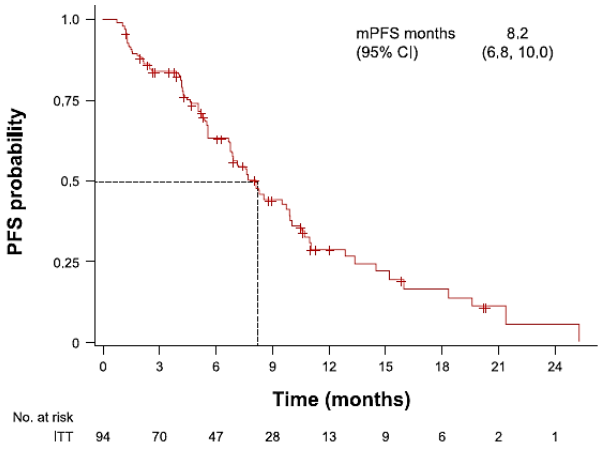
Van Gorp T, et al. Presented at: 2025 SGO Annual Meeting on Women's Cancer; March 14-17, 2025; Seattle, WA. 939696.

MIRVETUXIMAB SORAVTANSINE AND BEVACIZUMAB IN FOLATE RECEPTOR αLPHA-POSITIVE OVARIAN CANCER: EFFICACY IN PATIENTS WITH AND WITHOUT PRIOR BEVACIZUMAB

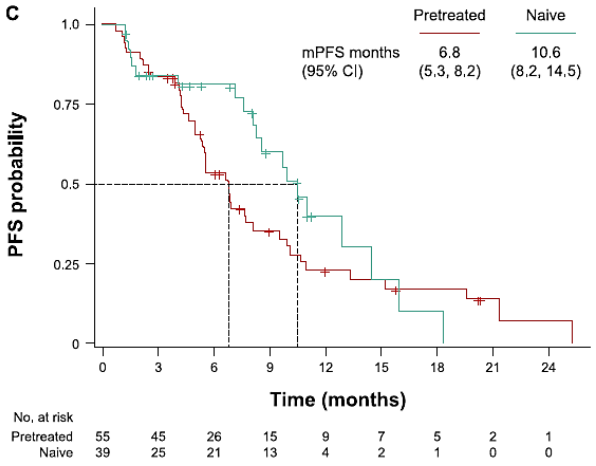
O'Malley D, et al. *Int J Gynecol Cancer.* 2022;32(Suppl 3A):A7-A8.

Phase 1b/2 FORWARD II Trial Subgroup Analysis of Mirv + Bev in Recurrent FRα-Expressing PROC

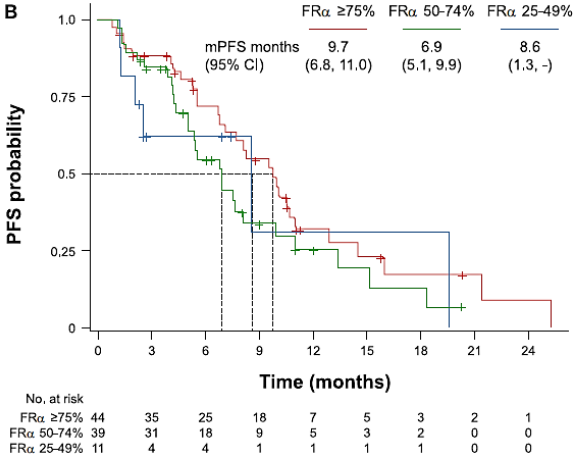
PFS in ITT



PFS by bev status



PFS by FRα expression



Data cutoff: June 21, 2021.

Gilbert L, et al. *Gynecol Oncol*. 2023;170:241-247.

Safety and efficacy of mirvetuximab soravtansine, a folate receptor alpha (FRα)–targeting antibody-drug conjugate (ADC), in combination with pembrolizumab in patients with platinum-resistant ovarian cancer

Table 2
Summary of efficacy measures.

Endpoint	All Participants[a] $N = 55$	Low FRα $n = 14$	Medium FRα $n = 14$	High FRα $n = 26$
Confirmed ORR, *n* (%)	17 (31)	3 (21)	4 (29)	9 (35)
95 % CI	19–45	5–51	8–58	17–56
Best overall response, *n* (%)				
Complete response	0	0	0	0
Partial response	17 (31)	3 (21)	4 (29)	9 (35)
Stable disease	27 (49)	7 (50)	6 (43)	14 (54)
Progressive disease	9 (16)	4 (29)	3 (21)	2 (8)
Not evaluable	2 (4)	0	1 (7)	1 (4)
Median PFS, months	4.2	3.25	2.79	5.59
95 % CI	2.8–5.6	1.38–5.52	1.38–5.45	2.79–8.48
Median DOR,[b] months	8.0	4.37	12.44	18.20
95 % CI	4.2-NR	3.02-NR	2.79-NR	2.79-NR

CI, confidence interval; DOR, duration of response; FRα, folate receptor alpha; NR, not reached; ORR, objective response rate; PFS, progression-free survival.
[a] FRα expression data was missing in one subject.
[b] Calculated among participants who achieved a complete or partial response.

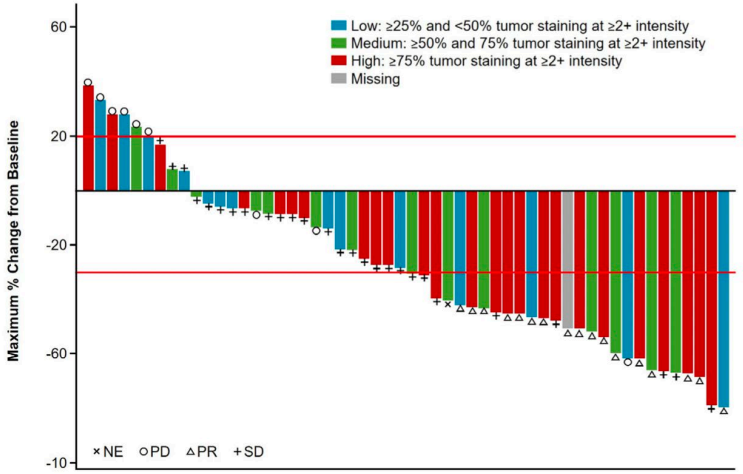


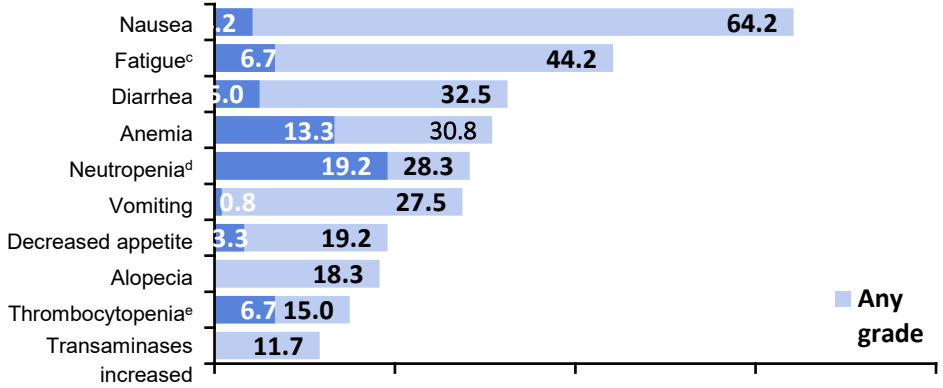
Fig. 2. Maximum percentage change in tumor lesion size from baseline by FRα expression.
Fig. 2 shows the maximum percentage change in tumor lesion size from baseline for individual patients by FRα expression status. The red lines (−30 % and 20 %) indicate the stable disease boundaries according to RECIST version 1.1 guidelines. Low, medium, and high expression was defined as 25 %–49 %, 50 %–74 %, and ≥75 % of tumor cells, respectively, with FRα membrane staining of ≥2+ intensity using PS2+ scoring methodology. Overall, 83 % of participants ($n = 45/54$) experienced tumor shrinkage of their target lesions in response to combination treatment. Confirmed PRs were observed in 16 participants.
FRα, folate receptor alpha; PR, partial response; PS2+, positive staining 2+; RECIST, Response Evaluation Criteria in Solid Tumors.

Matulonis UA, et al. *Gynecol Oncol*. 2025;200:96-104.

Gynecological Cancer Subgroup Analyses from the Phase 2: DESTINY-PanTumor02 Trial of T-DXd in HER2-Expressing Solid Tumors: Safety

Safety summary, n (%)		Gynecologic cohorts (N=120)
Any drug-related TEAEs		106 (**88.3**)
Drug-related TEAEs grade ≥3		54 (**45.0**)
Serious drug-related TEAEs		18 (**15.0**)
Drug-related TEAEs associated with:	Dose discontinuations	7 (**5.8**)
	Dose interruptions	24 (**20.0**)
	Dose reductions	35 (**29.2**)
	Deaths[a]	2 (**1.7**)

Most Common Drug-Related TEAEs (>10%) in Gynecologic Cohorts



ILD/pneumonitis adjudicated as T-DXd related[b], n (%)	Grade 1	Grade 2	Grade 3	Grade 4	Grade 5	Any grade
Gynecological cohorts N=120	4 (**3.3**)	8 (**6.7**)	**0**	**0**	1 (**0.8**)	13 (**10.8**)

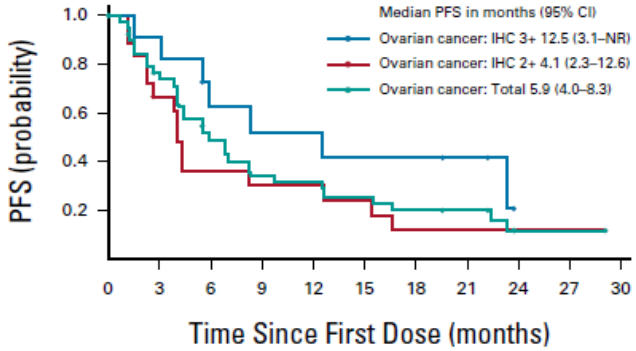
[a]Included pneumonia (n=1) and organizing pneumonia (n=1); [b]All ILD/pneumonitis cases were reviewed by an Adjudication Committee; [c]Category includes the preferred terms fatigue, asthenia, and malaise; [d]Category includes the preferred terms neutrophil count decreased and neutropenia; [e]Category includes the preferred terms platelet count decreased and thrombocytopenia.

TEAE = treatment-emergent adverse event; ILD = interstitial lung disease; T-DXd = trastuzumab deruxtecan; HER = human epidermal growth factor receptor.

Makker V, et al. *Gynecol Oncol.* 2024;190:S7-S8.

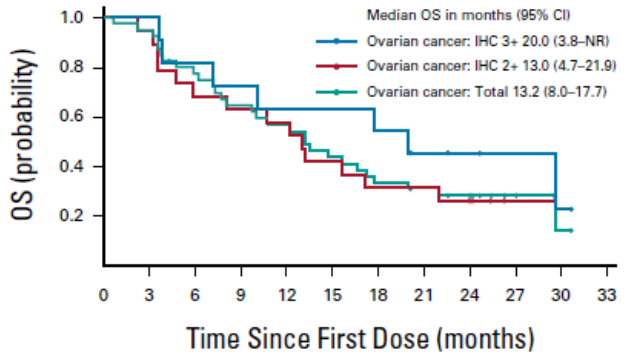
Phase 2 DESTINY-PanTumor02 Trial of T-DXd for HER2-Expressing Locally Advanced or Metastatic Tumors: Ovarian Cancer Subgroup Efficacy

Progression-Free Survival



No. at risk:											
Ovarian cancer: IHC 3+	11	10	6	5	5	4	4	3	0		
Ovarian cancer: IHC 2+	19	11	6	5	5	4	2	2	1	1	0
Ovarian cancer: Total	40	28	17	12	11	9	7	6	1	1	0

Overall Survival



No. at risk:											
Ovarian cancer: IHC 3+	11	11	9	8	7	7	6	4	3	2	1	0
Ovarian cancer: IHC 2+	19	18	13	12	11	8	6	6	4	1	0	
Ovarian cancer: Total	40	38	30	25	22	17	13	11	8	3	1	0

	All Ovarian N=40	IHC 3+ n=11	
ORR, % (95% CI)	45.0 (29.3-61.5)	63.6 (30.8-89.1)	36.8 (NA)

IHC = immunohistochemistry.

Meric-Bernstam F, et al. *J Clin Oncol*. 2024;42(1):47-60.

Pembrolizumab vs Placebo Plus Weekly Paclitaxel With or Without Bevacizumab for Platinum-Resistant Recurrent Ovarian Cancer: Results from the Randomized, Double-Blind Phase 3 ENGOT-ov65/ KEYNOTE-B96 Study

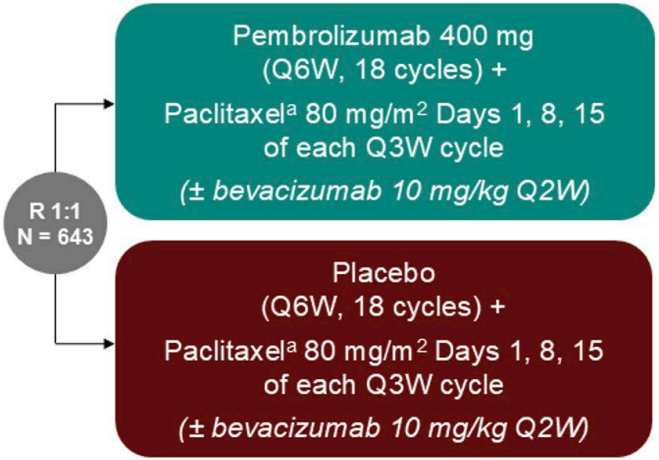
Colombo N, et al. *Ann Oncol.* 2025;36:S1582.

NCT05116189

ENGOT-ov65/KEYNOTE-B96: Study Design

Key Eligibility Criteria

- Histologically confirmed epithelial ovarian, fallopian tube, or primary peritoneal carcinoma
- 1 or 2 prior lines of therapy; at least 1 platinum-based chemotherapy
  - Prior anti-PD-1 or anti-PD-L1, PARPi and bevacizumab permitted
- Radiographic progression within 6 months after the last dose of platinum-based chemotherapy
- ECOG PS 0 or 1



Stratification Factors

- Planned bevacizumab use (yes vs no)
- Region (US vs EU vs ROW)
- PD-L1 CPS (<1 vs 1 to <10 vs ≥10)[b]

Primary Endpoint: PFS per RECIST v1.1 by investigator

Key Secondary: OS

[a]Docetaxel (75 mg/m² Q3W) may be considered in participants with severe hypersensitivity reaction to paclitaxel or an adverse event requiring discontinuation of paclitaxel after consultation with the sponsor; [b]The combined positive score (CPS) was assessed at a central laboratory using PD-L1 IHC 22C3 pharmDx and defined as the number of PD-L1 CPS ≥1 cells (tumor cells, lymphocytes, macrophages) divided by the total number of tumor cells × 100.

Colombo N, et al. *Ann Oncol.* 2025;36:S1582.

Baseline Characteristics

	Pembro Arm (N = 322)	Placebo Arm (N = 321)
Age, median (range)	62 y (37-85)	61 y (37-82)
Race[a]		
White	207 (64.3%)	217 (67.6%)
Asian	72 (22.4%)	58 (18.1%)
Multiple	12 (3.7%)	17 (5.3%)
Black or African American	8 (2.5%)	6 (1.9%)
Hawaiian/Pacific Islander	1 (0.3%)	1 (0.3%)
PD-L1 CPS		
<1	88 (27.3%)	89 (27.7%)
1 to <10	133 (41.3%)	132 (41.1%)
≥10	101 (31.4%)	100 (31.2%)
Stage at diagnosis (FIGO 2014 criteria)		
IA-IIB	25 (7.8%)	26 (8.1%)
III-IIIC	183 (56.8%)	189 (58.9%)
IVA-IVB	114 (35.4%)	106 (33.0%)

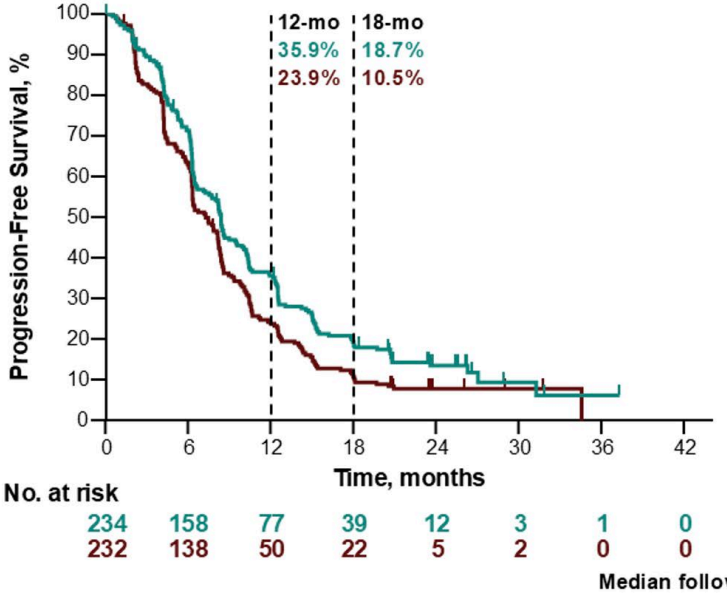
	Pembro Arm (N = 322)	Placebo Arm (N = 321)
ECOG PS 1	142 (44.1%)	144 (44.9%)
High-grade serous histology[b]	278 (86.3%)	275 (85.7%)
Bevacizumab use	235 (73.0%)	236 (73.5%)
Prior lines of therapy[c]		
1 line	121 (37.6%)	113 (35.2%)
2 lines	200 (62.1%)	207 (64.5%)
Prior anticancer therapy		
Anti-PD-1 or PD-L1	7 (2.2%)	7 (2.2%)
Bevacizumab	149 (46.3%)	146 (45.5%)
PARP inhibitor	112 (34.8%)	123 (38.3%)
Platinum-free interval[d]		
<3 mo	137 (42.5%)	162 (50.5%)
≥3 to ≤6 mo	183 (56.8%)	154 (48.0%)
>6 mo	2 (0.6%)	4 (1.2%)

[a]44 participants had missing information for race, 22 (6.8%) in the pembro arm and 22 (6.9%) in the placebo arm. [b]Other histology subtypes in the pembro and placebo arms, respectively, were clear cell in 24 (7.5%) and 26 (8.1%), endometrioid in 9 (2.8%) and 4 (1.2%), low-grade serous in 6 (1.9%) and 10 (3.1%), carcinosarcoma in 3 (0.9%) and 5 (1.6%), and other carcinoma in 2 (0.6%) and 1 (0.3%). [c]2 participants had 3 prior lines of therapy, 1 (0.3%) in each treatment arm. [d]1 participant in the placebo arm had missing information for platinum-free interval. Data cutoff date: March 5, 2025.

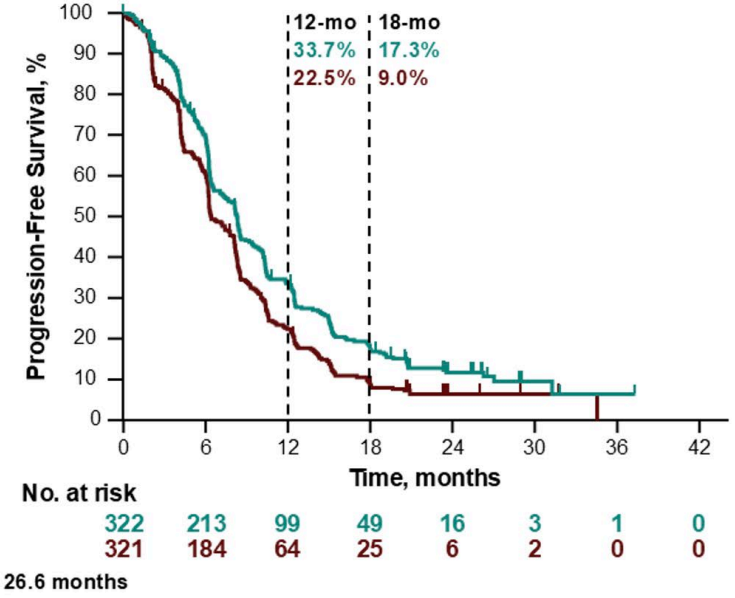
CPS = combined positive score; FIGO = Fédération Internationale de Gynécologie et d'Obstétrique.
Colombo N. Presented at: European Society of Medical Oncology (ESMO) Congress; 2025.

PFS in the CPS ≥1 and ITT Populations at IA2

CPS ≥1 Population	Median, months	Events	HR (95% CI)
Pembro Arm	8.3	81.6%	0.75[a] (0.61-0.91)
Placebo Arm	7.2	86.6%	



ITT Population	Median, months	Events	HR (95% CI)
Pembro Arm	8.3	83.2%	0.73[a] (0.62-0.86)
Placebo Arm	6.4	87.2%	

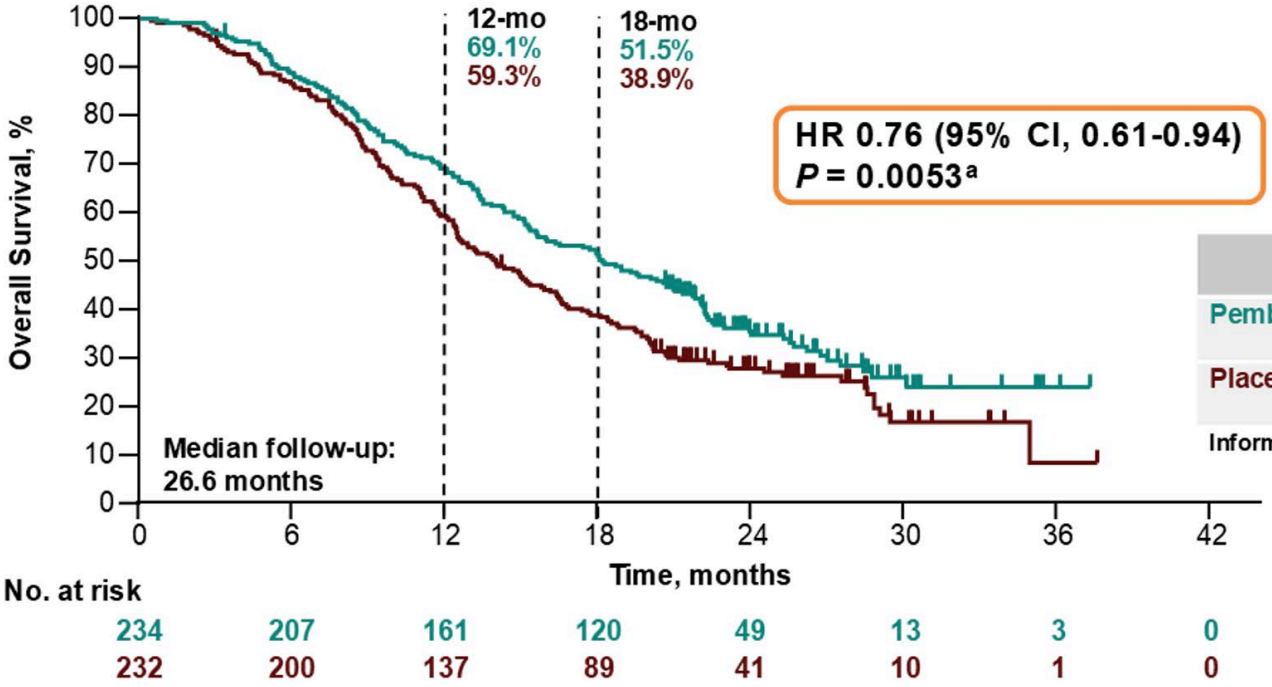


Median follow-up: 26.6 months

Response assessed per RECIST v1.1 by investigator review. [a]Hazard ratio (CI) analyzed based on a Cox regression model with treatment as a covariate stratified by the randomization stratification factors. No statistical testing for PFS was done at this analysis because significance was achieved at IA1. Data cutoff date: March 5, 2025.

Colombo N, et al. *Ann Oncol.* 2025;36:S1582.

Key Secondary Endpoint: OS in the CPS ≥1 Population at IA2



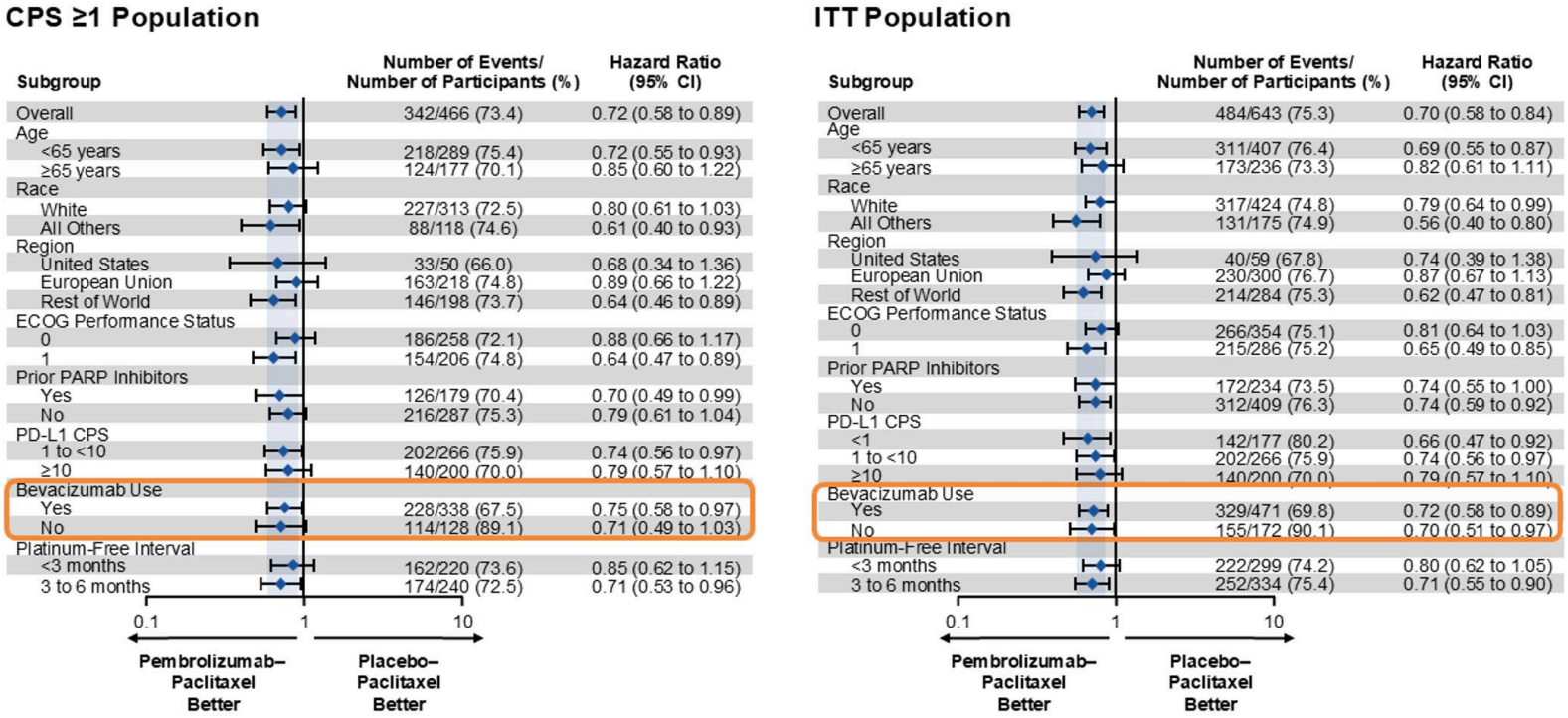
	Pts w/ Event	Median, mo (95% CI)
Pembro Arm	67.1%	18.2 (15.3-21.0)
Placebo Arm	75.4%	14.0 (12.5-16.1)

Information fraction: 90.0%

[a]Hazard ratio (CI) analyzed based on a Cox regression model with treatment as a covariate stratified by the randomization stratification factors. The observed *p*-value crossed the prespecified nominal boundary of 0.0083 at this planned second interim analysis. Data cutoff date: March 5, 2025.

Colombo N, et al. *Ann Oncol.* 2025;36:S1582.

PFS in Subgroups in the CPS ≥1 and ITT Populations at IA1



Response assessed per RECIST v1.1 by investigator review. The subgroup results shown in the forest plot were based on an unstratified Cox model, so the results for CPS ≥1 may differ slightly compared with those of the primary analysis, which were based on a stratified Cox model. Data cutoff date: April 3, 2024.

Colombo N, et al. *Ann Oncol.* 2025;36:S1582.

FDA approves pembrolizumab with paclitaxel for platinum-resistant epithelial ovarian, fallopian tube, or primary peritoneal carcinoma

On February 10, 2026, the Food and Drug Administration approved pembrolizumab as well as pembrolizumab and berahyaluronidase alfa-pmph in combination with paclitaxel, with or without bevacizumab, for adult patients with platinum-resistant epithelial ovarian, fallopian tube, or primary peritoneal carcinoma whose tumors express PD-L1 (CPS≥1) as determined by an FDA-authorized test, and who have received one or two prior systemic treatment regimens.

FDA also approved the PD-L1 IHC 22C3 assay as a companion diagnostic device to identify patients with epithelial ovarian, fallopian tube, or primary peritoneal carcinoma whose tumors express PD-L1 (CPS≥1) for treatment with pembrolizumab.

FDA = US Food and Drug Administration.

US Food and Drug Administration [www.fda.gov]. Last updated February 10, 2026. https://www.fda.gov/drugs/resources-information-approved-drugs/fda-approves-pembrolizumab-paclitaxel-platinum-resistant-epithelial-ovarian-fallopian-tube-or.

Platinum-Resistant Ovarian Cancer

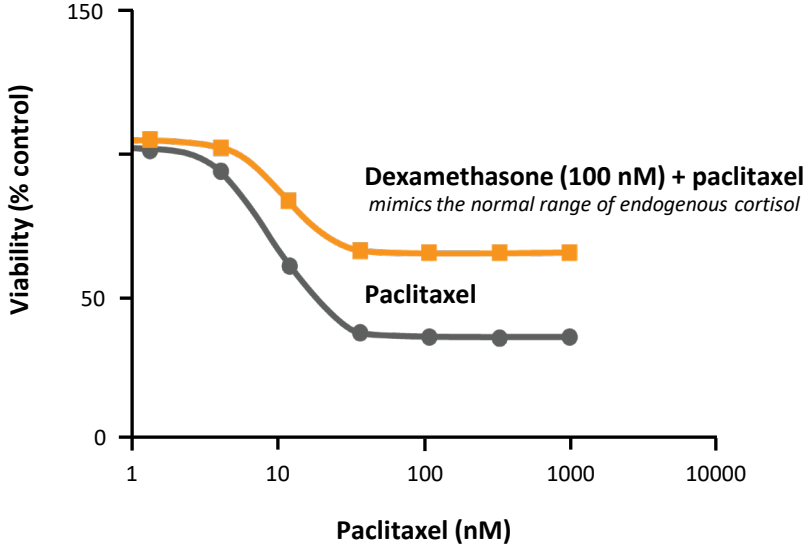
- Sequencing of therapies
  - Not well-defined
  - Single-agent versus combination therapy
- Clinical trials
- NCCN—many options (?good)
- Aurelia data
  - Combination bevacizumab with chemotherapy
  - Bevacizumab after bevacizumab
- Mirvetuximab
  - Folate receptor 75%—mirv
  - Folate receptor 25%—mirv + bev
  - Mirvetuximab + pembro
- Trastuzumab deruxtecan
  - HER2—2+/3+ (1+?)
  - Phase 2 data
- B96
  - Pembro + paclitaxel + /- bev
  - Platinum-resistant/refractory

Glucocorticoid Receptor Pathway

Evaluation of the Effect of GR Pathway Activation on Paclitaxel Activity

In Vitro—Ovarian Cancer Cell Line OVCAR5

- The GR is a ligand-activated transcription factor. Cortisol is an endogenous GR agonist. The normal range for morning serum cortisol, 276–552 nM, is higher than the concentration at which half-maximal GR activation occurs (9.5 nM)
- The effect of GR agonism with dexa at a concentration that mimics the normal physiological range of endogenous cortisol on the activity of paclitaxel was evaluated in an *in vitro* viability assay
  - Residual viability of OVCAR5 cells was quantified after exposure to paclitaxel at various dosages in the absence and presence of the exogenous GR agonist dexamethasone at 100 nM
  - A reduction in paclitaxel activity was observed in the presence of the GR agonist dexa at 100 nM

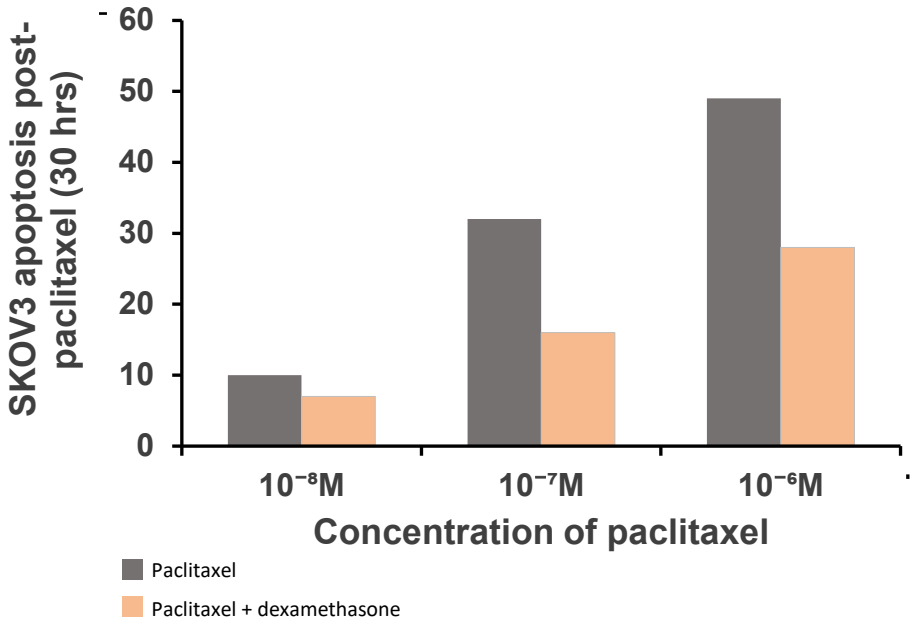


Buonaiuto R, et al. *Biomolecules.* 2023;13(4):653. Greenstein AE, Hunt HJ. *Int Immunopharmacol.* 2023;120:110312. Mulatero P, et al. H*ypertension.* 1997;30(5):1274-1278. Greenstein AE, Hunt HJ. *Oncotarget.* 2021;12(13):1243-1255.

Evaluation of the Effect of GR Pathway Activation on Paclitaxel-Induced Apoptosis

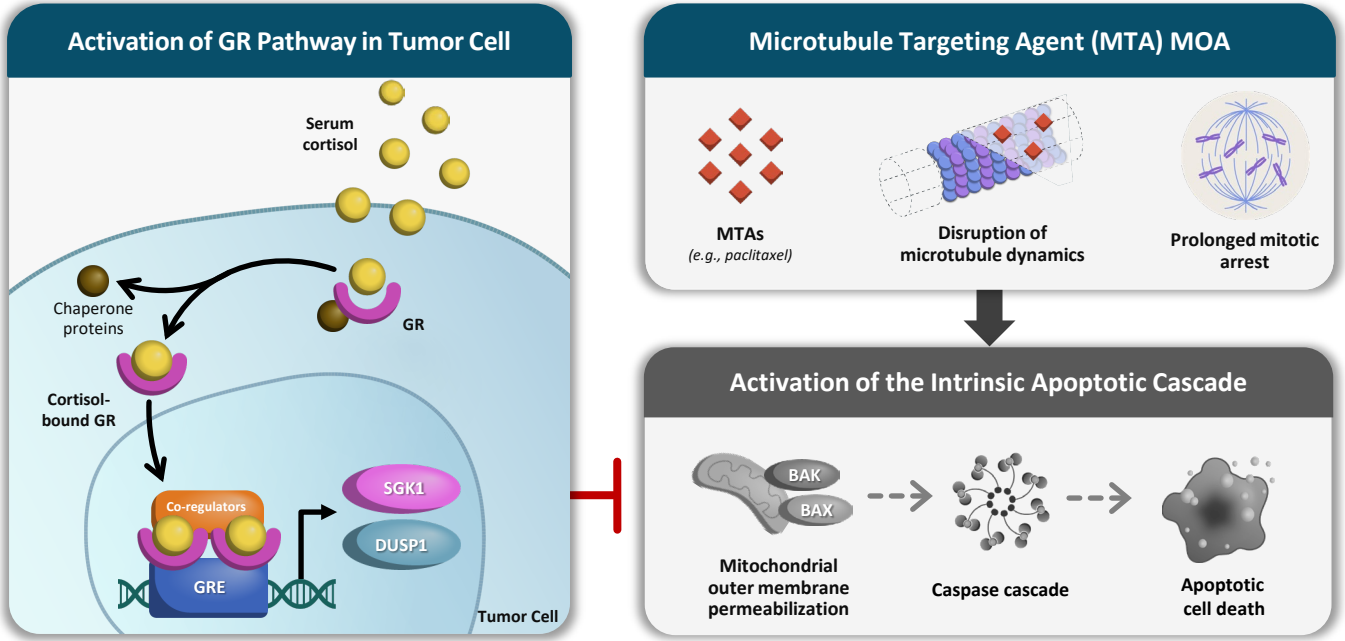
In Vitro – Ovarian Cancer Cell Line SKOV3

- The effect of GR pathway activation on paclitaxel induced apoptosis was evaluated in the ovarian cancer cell line SKOV3
  - Cells were cultured in the absence of growth factors for 24 hours and then treated with either dexa (10^{-6} mol/L) or vehicle for 1-hour before treatment with varying concentrations of paclitaxel
  - A reduction in apoptosis relative to control was observed with dexamethasone pretreatment in the 10^{-7} and 10^{-6} mol/L paclitaxel concentrations



Melhem A, et al. *Clin Cancer Res*. 2009;15(9):3196-204.

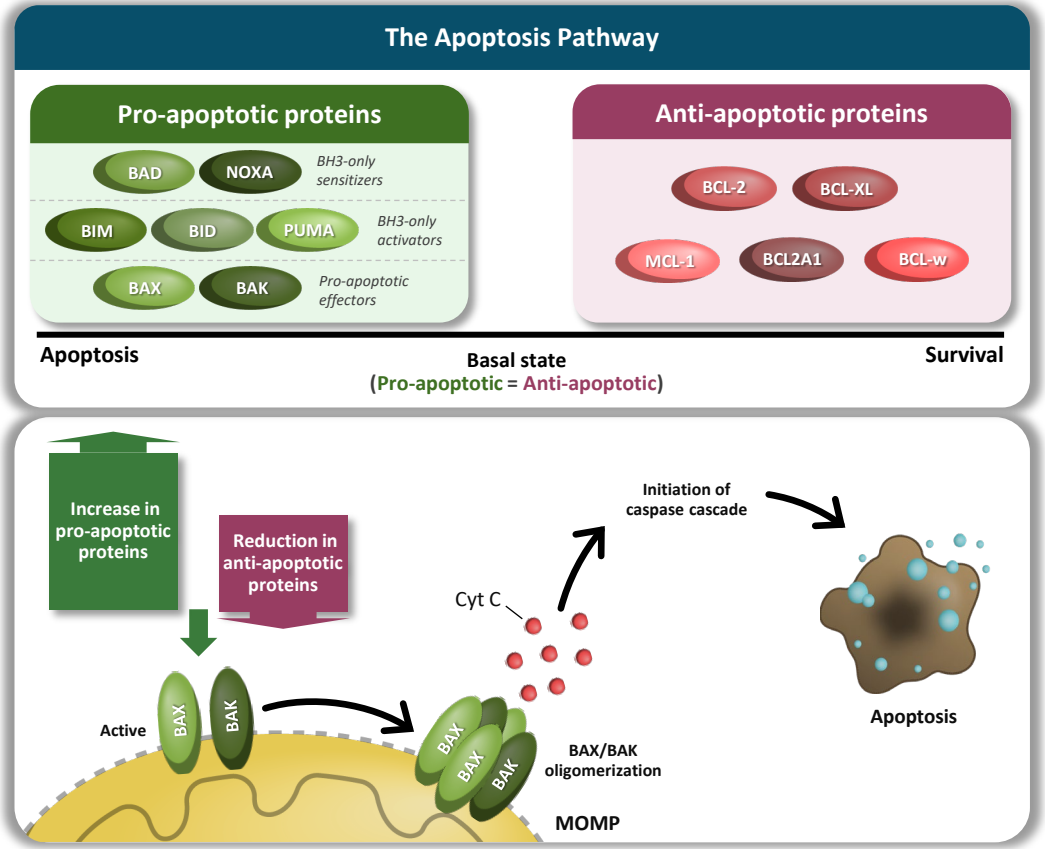
In pre-clinical evaluations a reduction in chemotherapy activity was observed with activation of the glucocorticoid receptor pathway



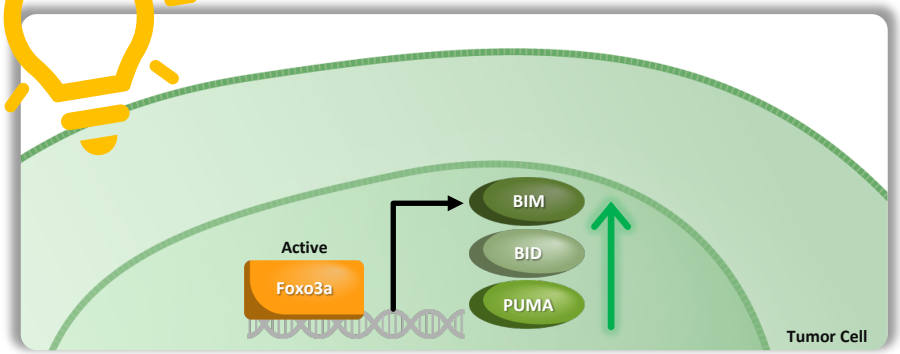
Activation of the GR pathway mediates the expression of genes that encode for the anti-apoptotic proteins SGK1 and DUSP1 resulting in the downstream inhibition of the activation of apoptotic effectors BAX and BAK

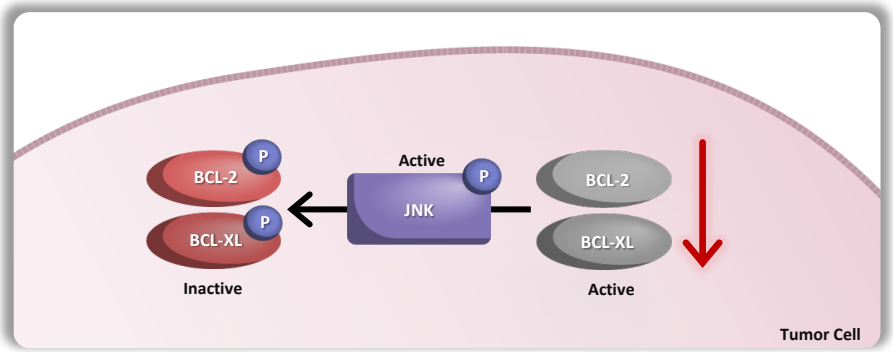
MOA = mechanism of action.

Melhem A, et al. *Clin Cancer Res*. 2009;15(9):3196-204. Stringer-Reasor EM, et al. *Gynecol Oncol*. 2015;138(3):656-62. Buonaiuto R, et al. *Biomolecules*. 2023;13(4):653. Liu Y, et al., *Mol Cancer*. 2018;17(1):104. Whitaker RH, et al. *Cells*. 2019;8(4):346. Pedley R, Gilmore AP. *Biol Chem*. 2016;397(7):595-605.

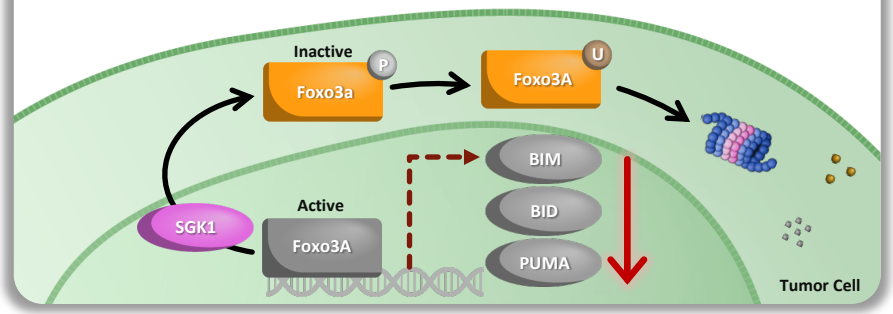


Chipuk JE, Green DR. *Trends Cell Biol.* 2008;18(4):157-164. Whitaker RH, et al. *Cells*. 2019;8(4):346. Pedley R, Gilmore AP. *Biol Chem*. 2016;397(7):595-605.

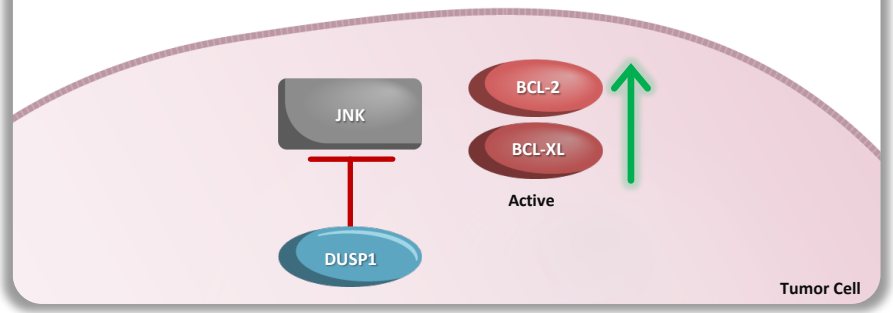




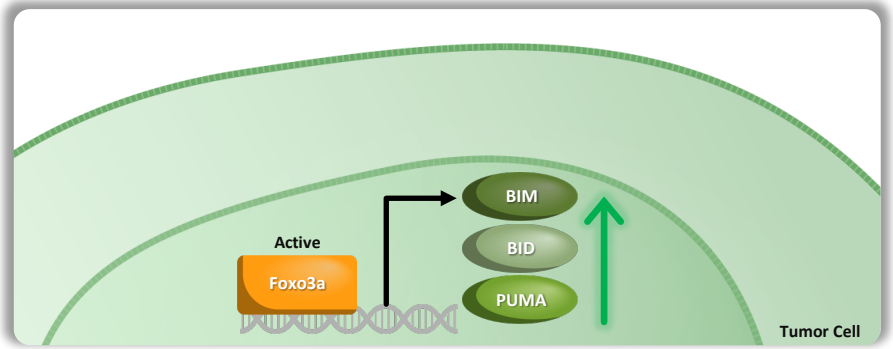
Reduction in Pro-apoptotic Proteins



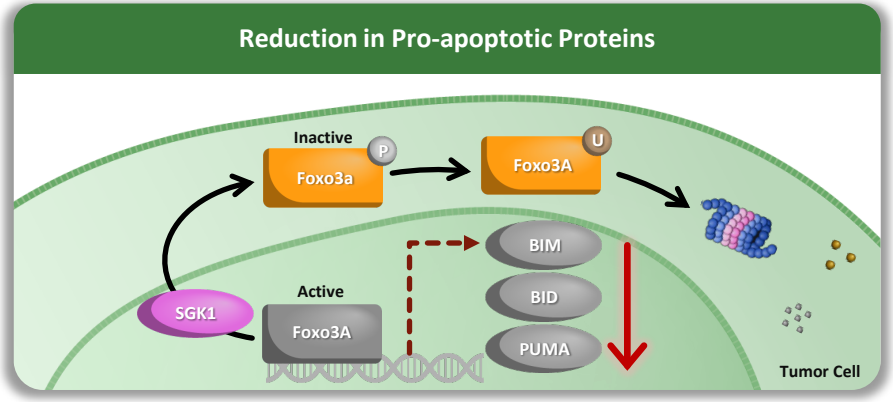
Increase in Active Anti-apoptotic Proteins



Liu Y, et al. *Mol Cancer*. 2018;17(1):104. Buonaiuto R, et al. *Biomolecules*. 2023;13(4):653. Dhanasekaran DN, et al. *Oncogene*. 2008;27(48):6245-6251.

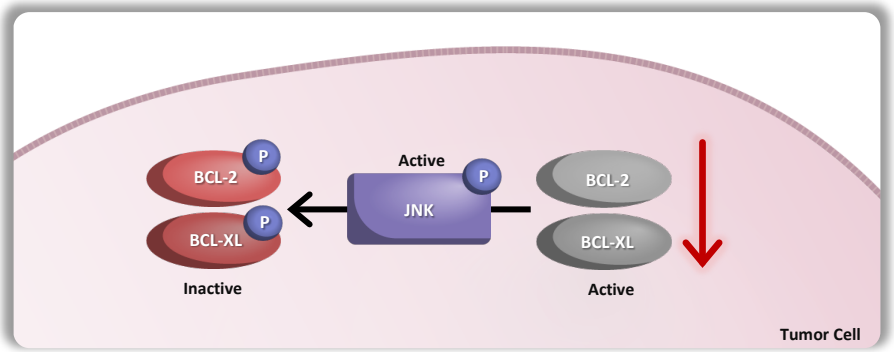


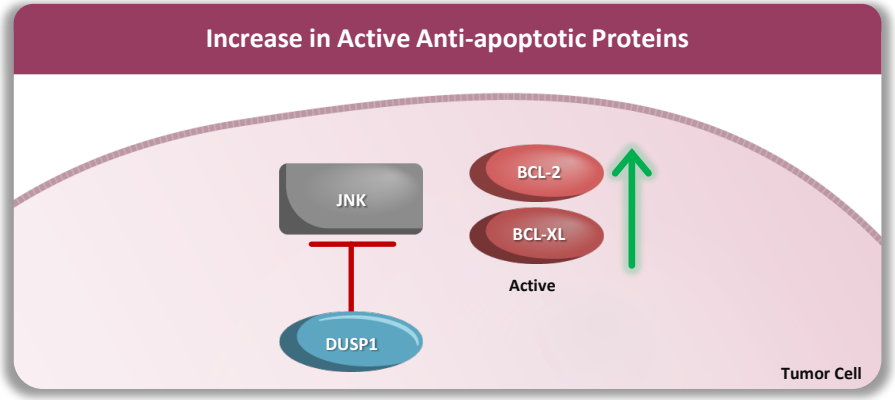
- FOXO3a, a member of the Forkhead family of transcription factors, promotes the expression of pro-apoptotic genes *BIM, BID*, and *PUMA*



- SGK1 regulates apoptosis via its effects on FOXO3a
  - SGK1 phosphorylates FOXO3a, resulting in its exit from the nucleus
  - In the cytoplasm FOXO3a is ubiquitinated and degraded by the proteasome

Liu Y, et al. *Mol Cancer*. 2018;17(1):104. Buonaiuto R, et al. *Biomolecules*. 2023;13(4):653. Dhanasekaran DN, et al. *Oncogene*. 2008;27(48):6245-6251.

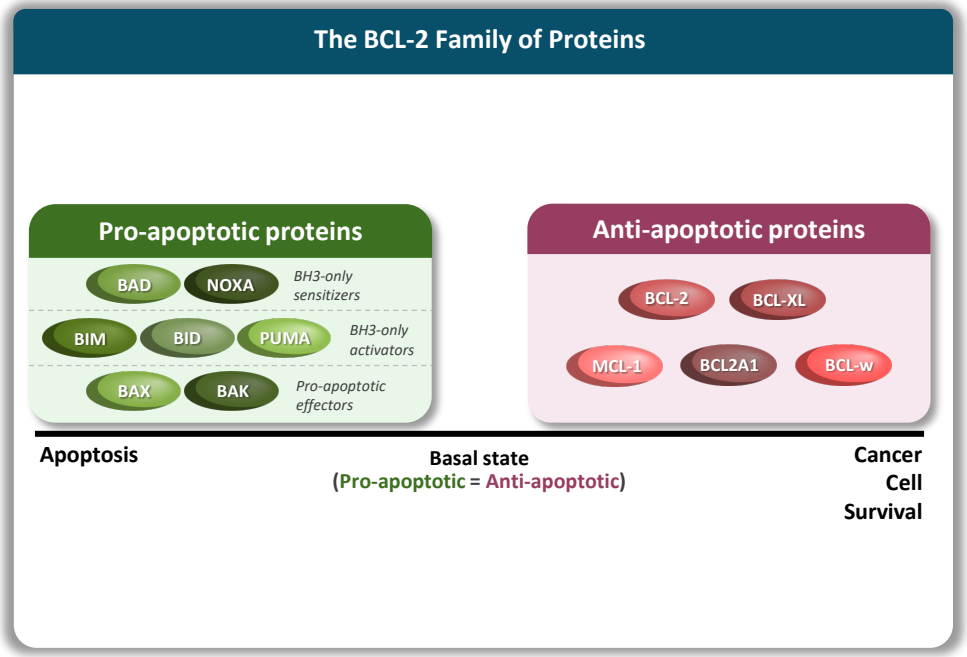




- In the absence of the dual-specificity protein phosphatase DUSP1, active JNK phosphorylates and inactivates the anti-apoptotic proteins BCL-2 and BCL-XL

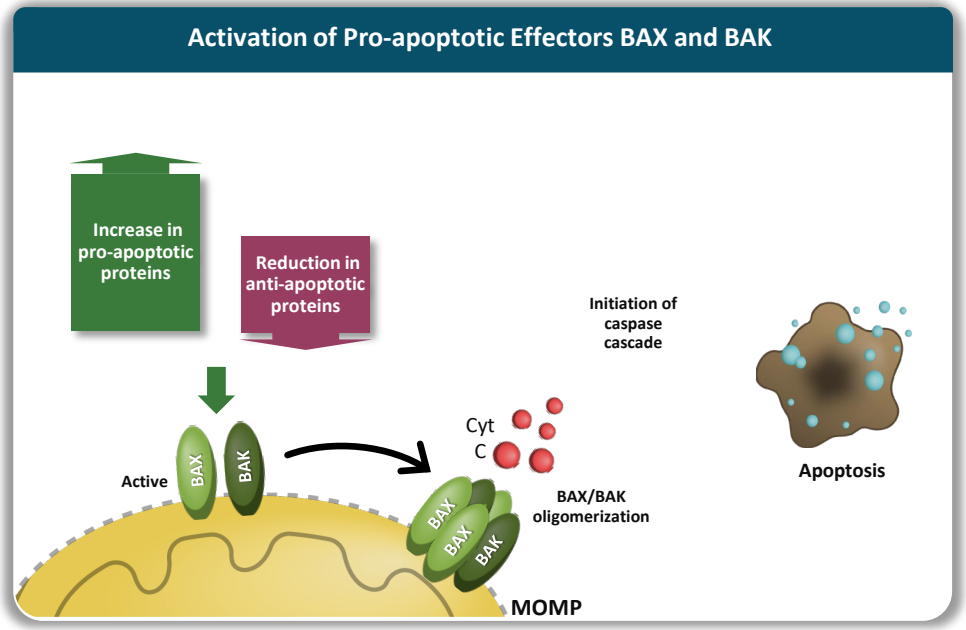
- DUSP1 dephosphorylates JNK, inhibiting the inactivation of the anti-apoptotic proteins BCL-2 and BCL-XL

Buonaiuto R, et al. *Biomolecules.* 2023;13(4):653. Dhanasekaran DN, et al. *Oncogene*. 2008;27(48):6245-6251.



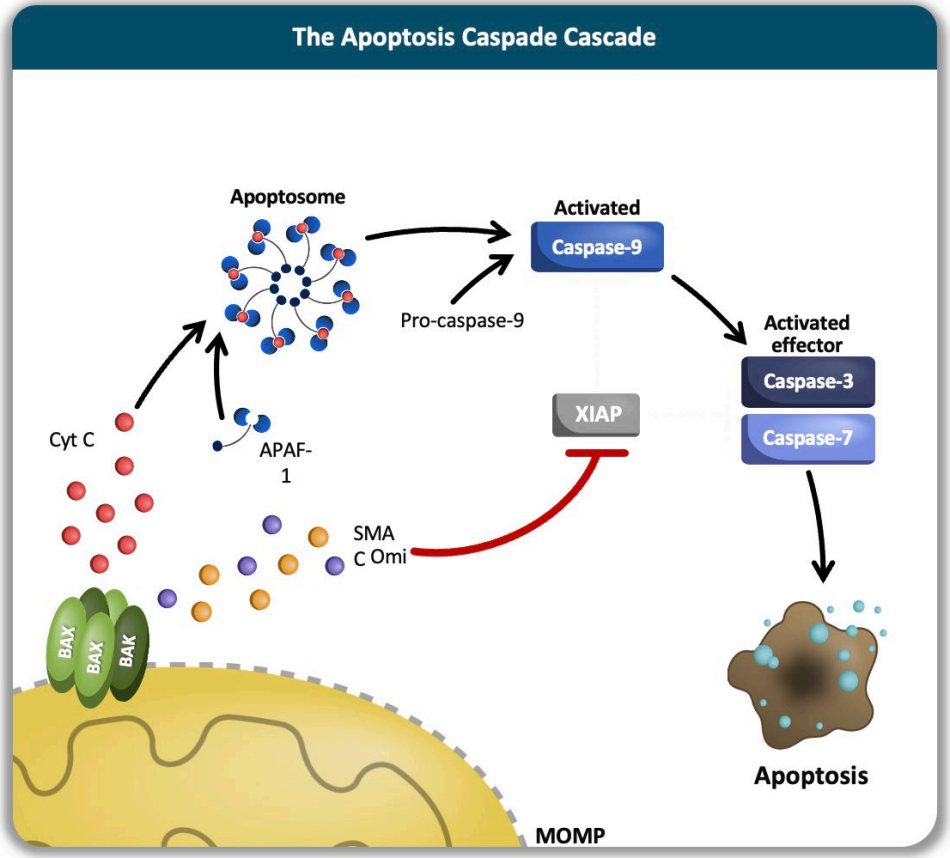
- The BCL-2 family of proteins maintain cellular fate (survival or apoptosis) via balancing pro- and anti-apoptotic proteins
  - Pro-apoptotic members include
    - Sensitizers: Displace activators bound by anti-apoptotic proteins
    - Activators: Induce oligomerization of effector proteins *BAX* and *BAK*
    - Effectors: Induce pore formation in the mitochondrial membrane
- Anti-apoptotic proteins inhibit *BAK/BAX* oligomerization by directly binding to *BAX* and *BAK*, as well as by binding activators sequestering them

Chipuk JE, Green DR. *Trends Cell Biol.* 2008;18(4):157-164. Whitaker RH, et al. *Cells*. 2019;8(4):346. Pedley R, Gilmore AP. *Biol Chem*. 2016;397(7):595-605.



- The sum of interactions between pro- and anti-apoptotic proteins ultimately regulate *BAX* and *BAK* activation resulting in their oligomerization and mitochondrial outer membrane permeabilization (MOMP)
  - *BAX* and *BAK* activation results from an increase in pro-apototic proteins and a reduction in anti-apoptotic proteins
- Induction of MOMP results in the release of cytochrome C, which eventually leads to apoptotic cell death via caspase cascade activation

Chipuk JE, Green DR. *Trends Cell Biol.* 2008;18(4):157-164. Whitaker RH, et al. *Cells*. 2019;8(4):346. Pedley R, Gilmore AP. *Biol Chem*. 2016;397(7):595-605.

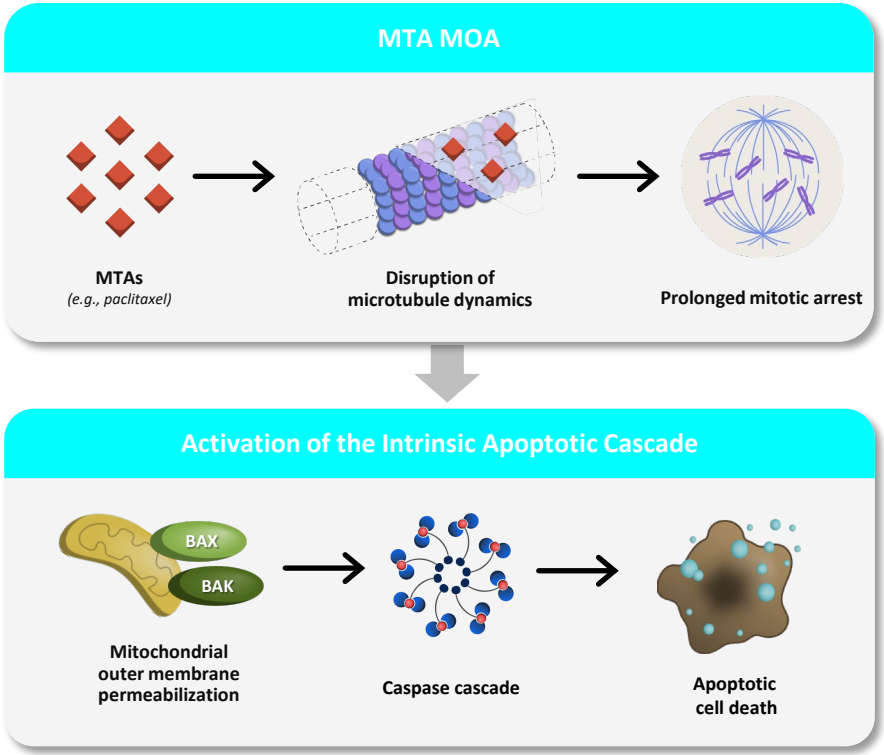


- MOMP results in the passive release of internal mitochondrial proteins, such as cytochrome C, second mitochondria-derived activator of caspases (SMAC) and Omi, into the cytosol
- Cytochrome C binds with cytosolic apoptotic protease activating factor (APAF-1) and oligomerizes to form apoptosome, a heptameric complex that recruits and activates procaspase-92
- Activated dimeric caspase-9 directly cleaves and activate the downstream effectors caspase-3 and caspase-7 and eventually leads to apoptotic cell death
- SMAC and Omi bind to and inhibit X-linked inhibitor of apoptosis protein (XIAP, an inhibitor of caspase activation) in the cytosol, counteracting its anti-apoptotic function and enabling caspase activation
- Caspases are a family of cysteine proteases that cleave cytoplasmic and nuclear substrates

Carneiro B, El-Deiry WS. *Nat Rev Clin Oncol*. 2020;17(7):395-417. Elkholi R, et al. *Cancer Metab*. 2014:2:16. Fulda S. *Int J Cancer*. 2009;124(3):511-515.

Mechanism of Action of MTAs

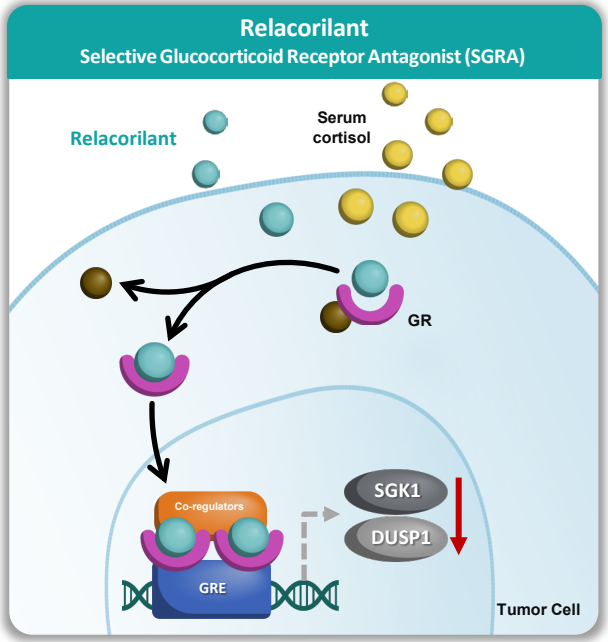
- Microtubule targeting agents induce cell death by activating the intrinsic apoptotic cascade which is tightly controlled by interactions between members of the BCL-2 family of proteins

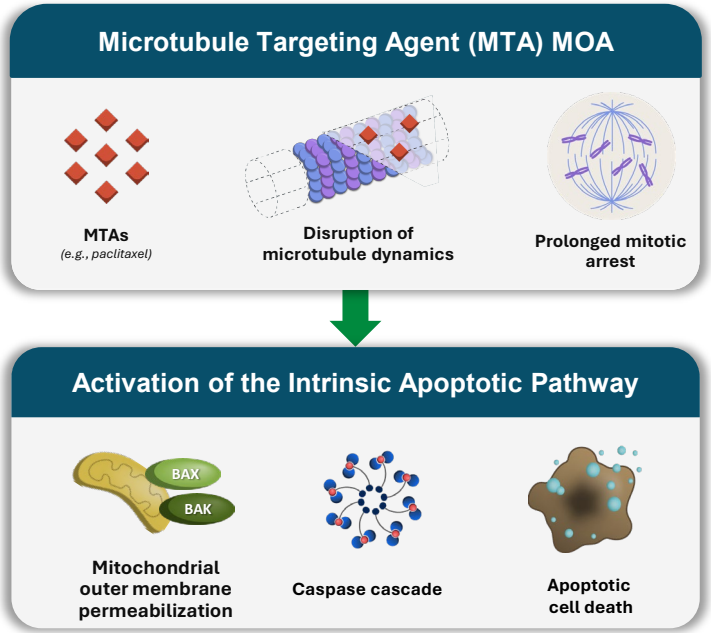


Whitaker RH, et al. *Cells*. 2019;8(4):346. Pedley R, Gilmore AP. *Biol Chem*. 2016;397(7):595-605.

Proposed MOA

Results from *in vitro* studies and xenograft models suggest that relacorilant may improve tumor sensitivity to paclitaxel increasing its efficacy





- Relacorilant (CORT125134) is an investigational selective antagonist of the glucocorticoid receptor. GR binding of relacorilant was observed with a Ki of 0.15 nM, while no measurable binding was detected with the progesterone, estrogen, or androgen receptors
- In pre-clinical evaluations relacorilant suppressed the expression of SGK1 and DUSP1 genes

Greenstein AE, Hunt HJ. *Oncotarget*. 2021;12(13):1243-1255. Melhem A, et al. *Clin Cancer Res*. 2009;15(9):3196-204. Stringer-Reasor EM, et al. *Gynecol Oncol*. 2015;138(3):656-62. Buonaiuto R, et al. *Biomolecules*. 2023;13(4):653. Liu Y, et al., *Mol Cancer*. 2018;17(1):104. Whitaker RH, et al. *Cells*. 2019;8(4):346. Pedley R, Gilmore AP. *Biol Chem*. 2016;397(7):595-605.

Summary of Glucocorticoid Inhibition

- Activation of GR pathway
  - Increases anti-apoptotic proteins
  - Decreases pro-apoptotic proteins
  - Decreases in-vivo effectiveness of chemotherapy in cell lines

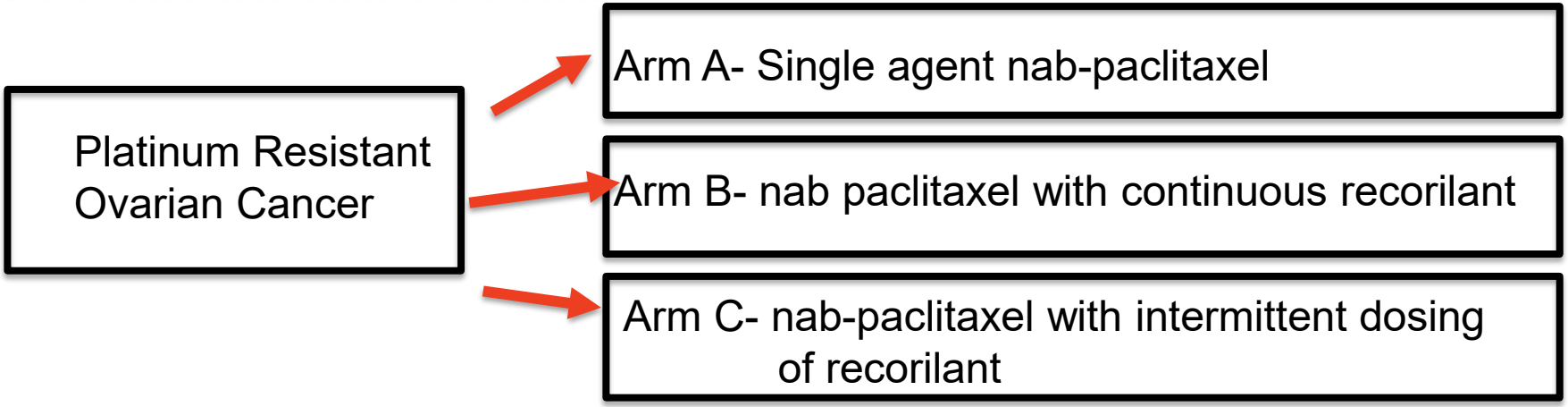
- Blockade of GR pathway
  - Opposite effect on proteins
  - Potential to allow for more effective chemotherapy induction of apopotosis on cancer cells

Original Reports | Gynecologic Cancer

Relacorilant + Nab-Paclitaxel in Patients With Recurrent, Platinum-Resistant Ovarian Cancer: A Three-Arm, Randomized, Controlled, Open-Label Phase II Study

Nicoletta Colombo, MD[1,2]; Toon Van Gorp, MD, PhD[3]; Ursula A. Matulonis, MD[4]; Ana Oaknin, MD, PhD[5]; Rachel N. Grisham, MD[6]; Gini F. Fleming, MD[7]; Alexander B. Olawaiye, MD[8]; Dorothy D. Nguyen, MD[9]; Andrew E. Greenstein, PhD[9]; Joseph M. Custodio, PhD[9]; Hristina I. Pashova, PhD[9]; Iulia C. Tudor, PhD[9]; and Domenica Lorusso, MD, PhD[10]

DOI https://doi.org/10.1200/JCO.22.02624



Colombo N, et al. *J Clin Oncol*. 2023;41(30):4779-4789.

Randomized Phase 2 Trial Nab-Paclitaxel with Recorilant

TABLE 1. Baseline Characteristics for the Study Population

Characteristic	Intermittent Relacorilant (150 mg) + Nab-Paclitaxel (80 mg/m^2; n = 60)	Continuous Relacorilant (100 mg) + Nab-Paclitaxel (80 mg/m^2; n = 58)	Nab-Paclitaxel Monotherapy (100 mg/m^2; n = 60)	Overall (N = 178)
Age, median (range), years	60 (38-81)	60 (45-75)	61.5 (41-81)	61 (38-81)
Platinum refractory,[a] No. (%)	23 (38.3)	20 (34.5)	22 (36.7)	65 (36.5)
Primary platinum refractory,[b] No. (%)	7 (11.7)	3 (5.2)	1 (1.7)	11 (6.2)
No. of prior systemic anticancer therapies,[c] median (range)	2.5 (1-4)	3 (1-5)	3 (1-4)	3 (1-5)
No. of prior chemotherapies, median (range)	2 (1-4)	2 (1-4)[d]	2 (1-4)	2 (1-4)[d]
≥4 prior lines of therapy,[c] No. (%)	7 (11.7)	15 (25.9)	9 (15.0)	31 (17.4)
Bevacizumab, No. (%)	31 (51.7)	37 (63.8)	37 (61.7)	105 (59.0)
PARP inhibitor, No. (%)	18 (30.0)	27 (46.6)	20 (33.3)	65 (36.5)
Molecular profiling[b]				
BRCA1(+), n/N (%)	5/42 (11.9)	4/42 (9.5)	7/48 (14.6)	16/132 (12.1)
BRCA1 unknown, n/N (%)	1/42 (2.4)	0/42 (0.0)	1/48 (2.1)	2/132 (1.5)
BRCA2(+), n/N (%)	1/36 (2.8)	3/39 (7.7)	3/39 (7.7)	7/114 (6.1)
BRCA2 unknown, n/N (%)	1/36 (2.8)	1/39 (2.6)	1/39 (2.6)	3/114 (2.6)
Presence of ascites,[e,f] No. (%)	16 (26.7)	15 (25.9)	16 (26.7)	47 (26.4)
Treatment-free interval from most recent taxane, No. (%)				
Relapse within 6 months[e,g]	29 (48.3)	29 (50.0)	29 (48.3)	87 (48.9)
≤6 months[h]	30 (50.0)	26 (44.8)	27 (45.0)	83 (46.6)
>6 to ≤12 months	6 (10.0)	12 (20.7)	14 (23.3)	32 (18.0)
>12 months	23 (38.3)	20 (34.5)	19 (31.7)	62 (34.8)
Missing	1 (1.7)	0	0	1 (0.6)
Measurable disease at baseline, No. (%)	56 (93.3)	54 (93.1)	53 (88.3)	163 (91.6)
Histology, No. (%)				
High-grade serous	56 (93.3)	53 (91.4)	57 (95.0)	166 (93.3)
Endometrioid	0	1 (1.7)	1 (1.7)	2 (1.1)
Carcinosarcoma	1 (1.7)	0	1 (1.7)	2 (1.1)
Mixed	1 (1.7)	1 (1.7)	1 (1.7)	3 (1.7)
Others	1 (1.7)	1 (1.7)	0	2 (1.1)

Colombo N, et al. *J Clin Oncol*. 2023;41(30):4779-4789.

Randomized Phase 2 Trial Nab-Paclitaxel with Recorilant

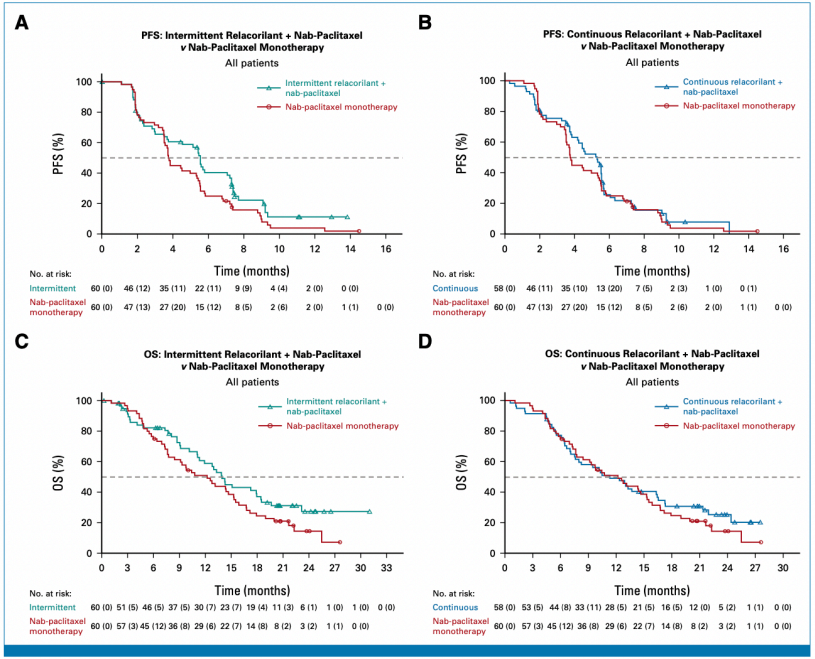


FIG 2. PFS and OS for intermittent relacorilant + nab-paclitaxel versus nab-paclitaxel monotherapy and continuous relacorilant + nab-paclitaxel versus nab-paclitaxel monotherapy. (A) and (B) PFS in all patients. At the primary analysis, 47 of 60 (78.3%, intermittent), 50 of 58 (86.2%, continuous), and 57 of 60 (95.0%, nab-paclitaxel monotherapy) patients had experienced a PFS event (PD by RECIST v1.1 or death). The median follow-up was 11.1 months. Intermittent relacorilant + nab-paclitaxel improved PFS compared with nab-paclitaxel monotherapy (log-rank, $P = .038$). (C) and (D) OS in all patients. At the OS analysis, 37 of 60 (61.7%, intermittent), 42 of 58 (72.4%, continuous), and 49 of 60 (81.7%, nab-paclitaxel monotherapy) OS events had occurred. The median follow-up was 22.5 months. A trend toward improved OS was observed in the intermittent arm (HR, 0.67; 95% CI, 0.43 to 1.03; $P = .066$). OS, overall survival; PD, progressive disease; PFS, progression-free survival.

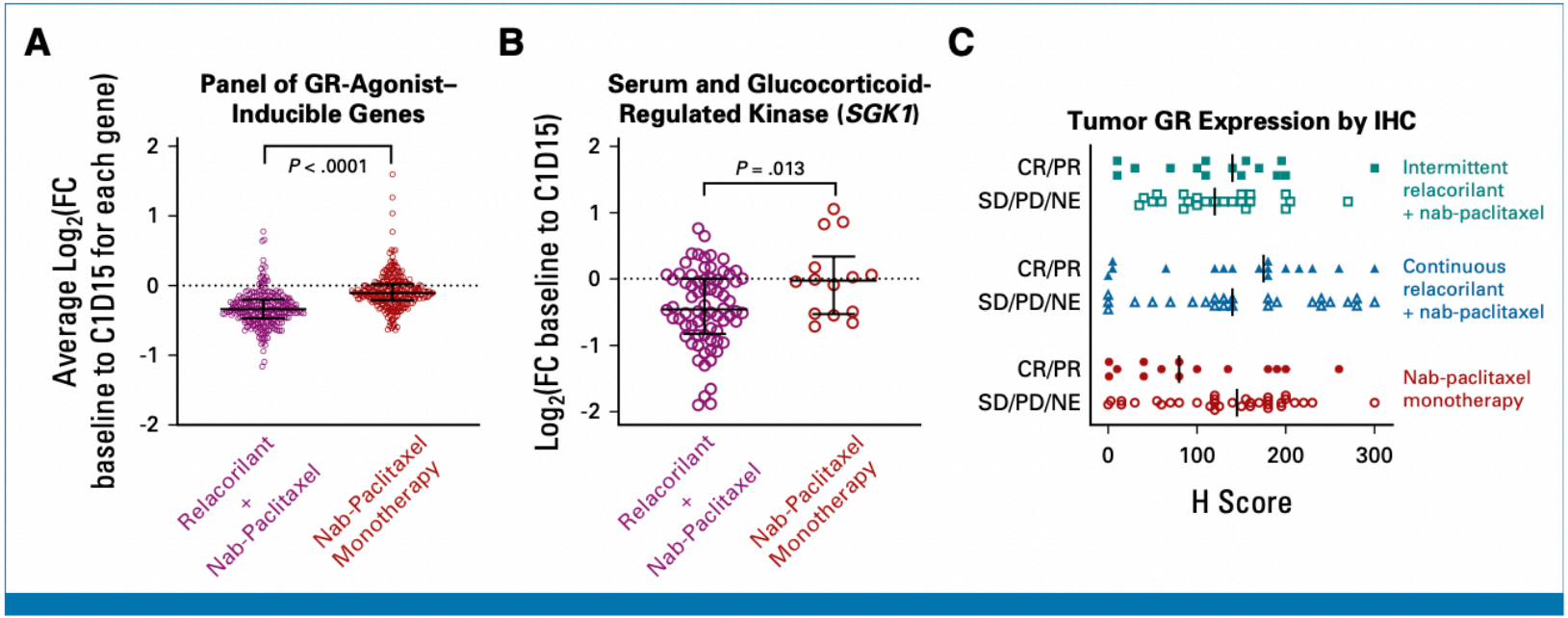
TABLE 2. BOR and Objective Response in Patients With Measurable Disease at Baseline and DOR in the Intent-to-Treat Population

Response	Intermittent Relacorilant (150 mg) + Nab-Paclitaxel (80 mg/m^2; n = 56)	Continuous Relacorilant (100 mg) + Nab-Paclitaxel (80 mg/m^2; n = 54)	Nab-Paclitaxel Monotherapy (100 mg/m^2; n = 53)
BOR, No. (%)			
CR	1 (1.8)	4 (7.4)	2 (3.8)
PR	19 (33.9)	15 (27.8)	17 (32.1)
SD	20 (35.7)	23 (42.6)	21 (39.6)
PD	14 (25.0)	9 (16.7)	12 (22.6)
NE	2 (3.6)	3 (5.6)	1 (1.9)
ORR, No. (%)	20 (35.7)	19 (35.2)	19 (35.8)
Two-sided 95% CI	23.4 to 49.6	22.7 to 49.4	23.1 to 50.2
DOR, median (95% CI), months	5.55 (3.75 to 5.88)	3.79 (2.33 to 5.55)	3.65 (2.89 to 5.09)
Stratified HR (95% CI)	0.36 (0.16 to 0.77)	0.72 (0.33 to 1.58)	—
Log-rank *P* value *v* nab-paclitaxel monotherapy	.006	.423	—

NOTE. Intermittent relacorilant dosed once on the day before, once the day of, and once the day after nab-paclitaxel infusion. Continuous relacorilant dosed once daily. Nab-paclitaxel dosed once per day on days 1, 8, and 15 of each 28-day cycle.
Abbreviations: BOR, best overall response; CR, complete response; DOR, duration of response; HR, hazard ratio; NE, not evaluable; ORR, objective response rate; PD, progressive disease; PR, partial response; SD, stable disease.

Colombo N, et al. *J Clin Oncol*. 2023;41(30):4779-4789.

Randomized Phase 2 Trial Nab-Paclitaxel with Recorilant



Colombo N, et al. *J Clin Oncol*. 2023;41(30):4779-4789.

ROSELLA: A Phase 3 Study of Relacorilant in Combination with Nab-Paclitaxel versus Nab-Paclitaxel Monotherapy in Patients with Platinum-Resistant Ovarian Cancer

(GOG-3073, ENGOT-ov72, APGOT-Ov10, LACOG-0223, and ANZGOG-2221/2023)

Alexander Olawaiye,[1] Laurence Gladieff, Lucy Gilbert, Jae-Weon Kim, Mariana Scaranti, Vanda Salutari, Elizabeth Hopp, Linda Mileshkin, Alix Devaux, Michael McCollum, Ana Oaknin, Aliza L. Leiser, Nicoletta Colombo, Andrew Clamp, Boglárka Balázs, Giuseppa Scandurra, Emilie Kaczmarek, Hristina I. Pashova, Sachin G. Pai, and Domenica Lorusso

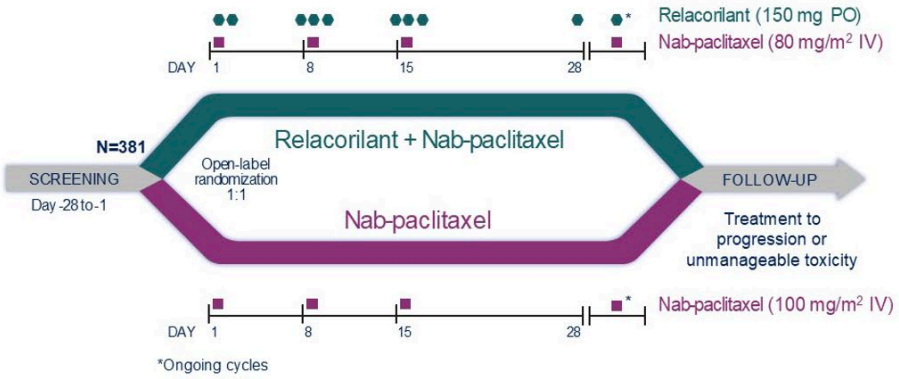
Olawaiye A, et al. *J Clin Oncol.* 2025;43:LBA5507.

ROSELLA: Study Schema

Population

- Epithelial ovarian, primary peritoneal or fallopian tube cancer
- ECOG performance status 0 or 1
- Progression <6 months after the last dose of platinum therapy (excluding no response to, or progression in <1 month of primary platinum)
- 1–3 prior lines of therapy
- Prior bevacizumab required

NCT05257408



Stratification Factors
- Prior lines of therapy (1 vs >1)
- Region (North America vs Europe vs Korea, Australia, & Latin America)

Dual Primary Endpoints

- Progression-free survival (PFS) by RECIST v1.1 per blinded independent central review
- Overall survival

Secondary Endpoints

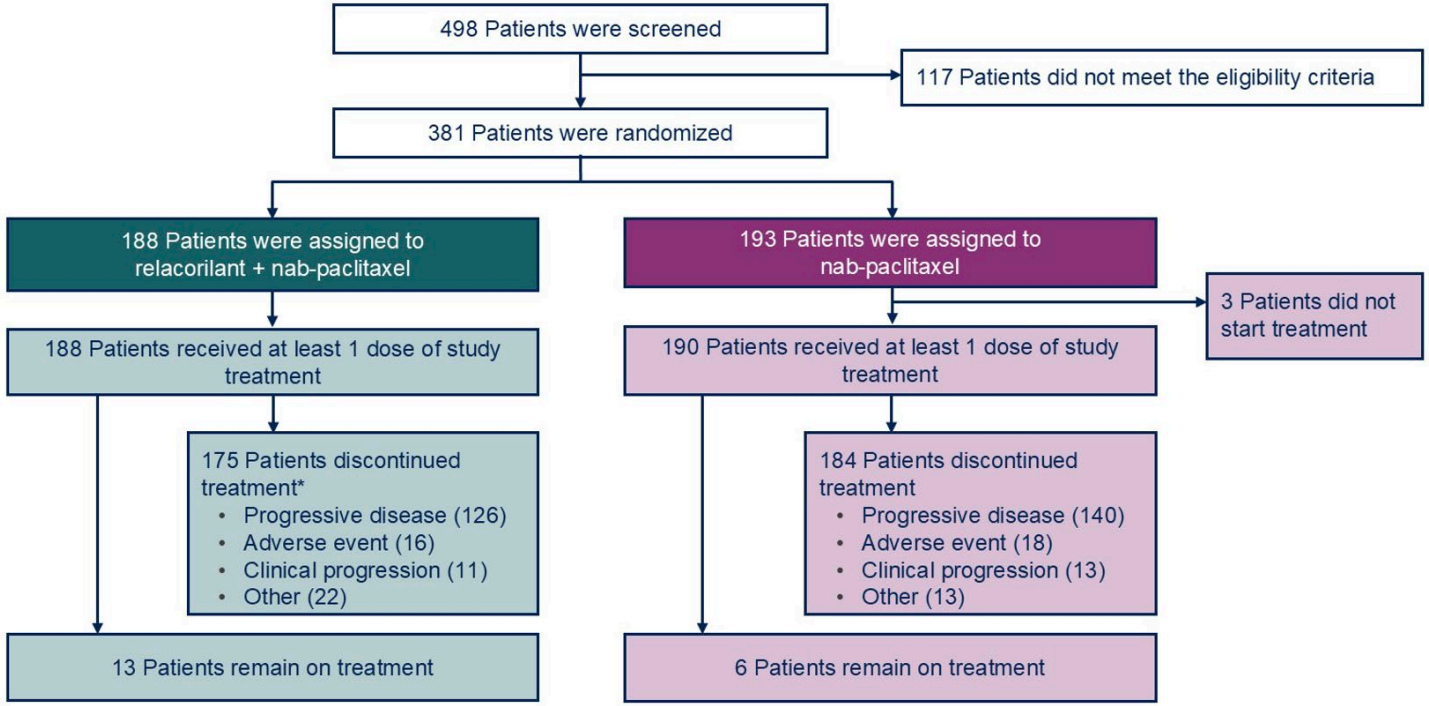
- PFS by RECIST v1.1 per Investigator
- ORR, DoR, CBR (RECIST v1.1)
- Response by CA-125 GCIG criteria
- Combined response (RECIST v1.1 and CA-125 GCIG criteria)
- Safety

First patient enrolled: 5th January 2023
Last patient enrolled: 8th April 2024
Data cutoff: 24th February 2025
Conducted at 117 sites in 14 countries.

CBR = clinical benefit rate; GCIG = Gynecological Cancer Intergroup; RECIST = Response Evaluation Criteria in Solid Tumors.

Olawaiye A, et al. *J Clin Oncol.* 2025;43:LBA5507.

ROSELLA: Patient Disposition



Numbers shown are for nab-paclitaxel discontinuations. Progressive disease refers to radiographic progression.
Data cutoff: Feb 24, 2025.

Olawaiye A, et al. *J Clin Oncol.* 2025;43:LBA5507.

Baseline Characteristics Were Well Balanced

		Relacorilant + Nab-paclitaxel (N=188)	Nab-paclitaxel (N=193)
Age, median (range), years		**61** (26–85)	**62** (33–86)
Race, n (%)	White Black or African-American Asian (92% Korean) Other / Not Reported	136 **(72.3)** 3 **(1.6)** 22 **(11.7)** 27 **(14.4)**	135 **(69.9)** 2 **(1.0)** 26 **(13.5)** 30 **(15.5)**
Ethnicity, n (%)	Hispanic	16 **(8.5)**	17 **(8.8)**
Region	North America Europe Korea, Australia, and Latin America	45 **(23.9)** 107 **(56.9)** 36 **(19.1)**	45 **(23.3)** 109 **(56.5)** 39 **(20.2)**
ECOG Performance Status, n (%)*	1 or 2	53 **(28.2)**	63 **(32.6)**
BRCA1/2 Mutation, n (%)	Yes	23 **(12.2)**	24 **(12.4)**
Prior Lines of Therapy, n (%)	1 2 3	15 **(8.0)** 92 **(48.9)** 81 **(43.1)**	18 **(9.3)** 89 **(46.1)** 86 **(44.6)**
Primary Platinum Refractory, n (%)†	Yes	13 **(6.9)**	13 **(6.7)**
Prior Lines of Therapy in the Platinum-resistant Setting, n (%)	≥1	67 **(35.6)**	82 **(42.5)**
Prior Taxane in the Platinum-resistant Setting, n (%)	Yes	8 **(4.3)**	7 **(3.6)**
Prior Therapies, n (%)	Bevacizumab Taxanes Pegylated Liposomal Doxorubicin PARP Inhibitor	188 **(100)** 187 **(99.5)** 121 **(64.4)** 114 **(60.6)**	193 **(100)** 192 **(99.5)** 125 **(64.8)** 120 **(62.2)**

In the nab-paclitaxel monotherapy arm, 1 patient had an ECOG performance status of 2. †Progressed within 3 months of the last dose of platinum from their first line platinum regimen. 97% of patients had high-grade serous carcinoma, 8 patients had high-grade endometrioid carcinoma and 2 patients had carcinosarcoma. Data cutoff: Feb 24, 2025

BRCA = breast cancer gene.

Olawaiye A, et al. *J Clin Oncol.* 2025;43:LBA5507.

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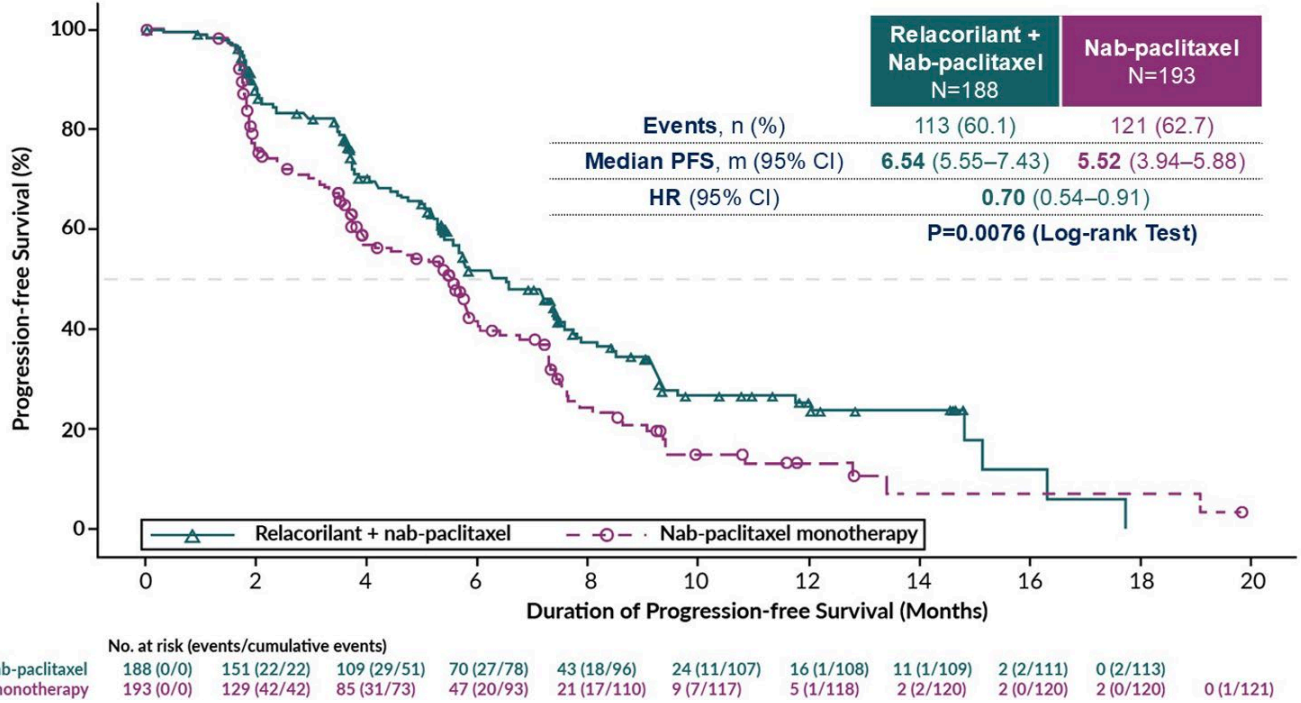
Olawaiye A, et al. *J Clin Oncol.* 2025;43:LBA5507.

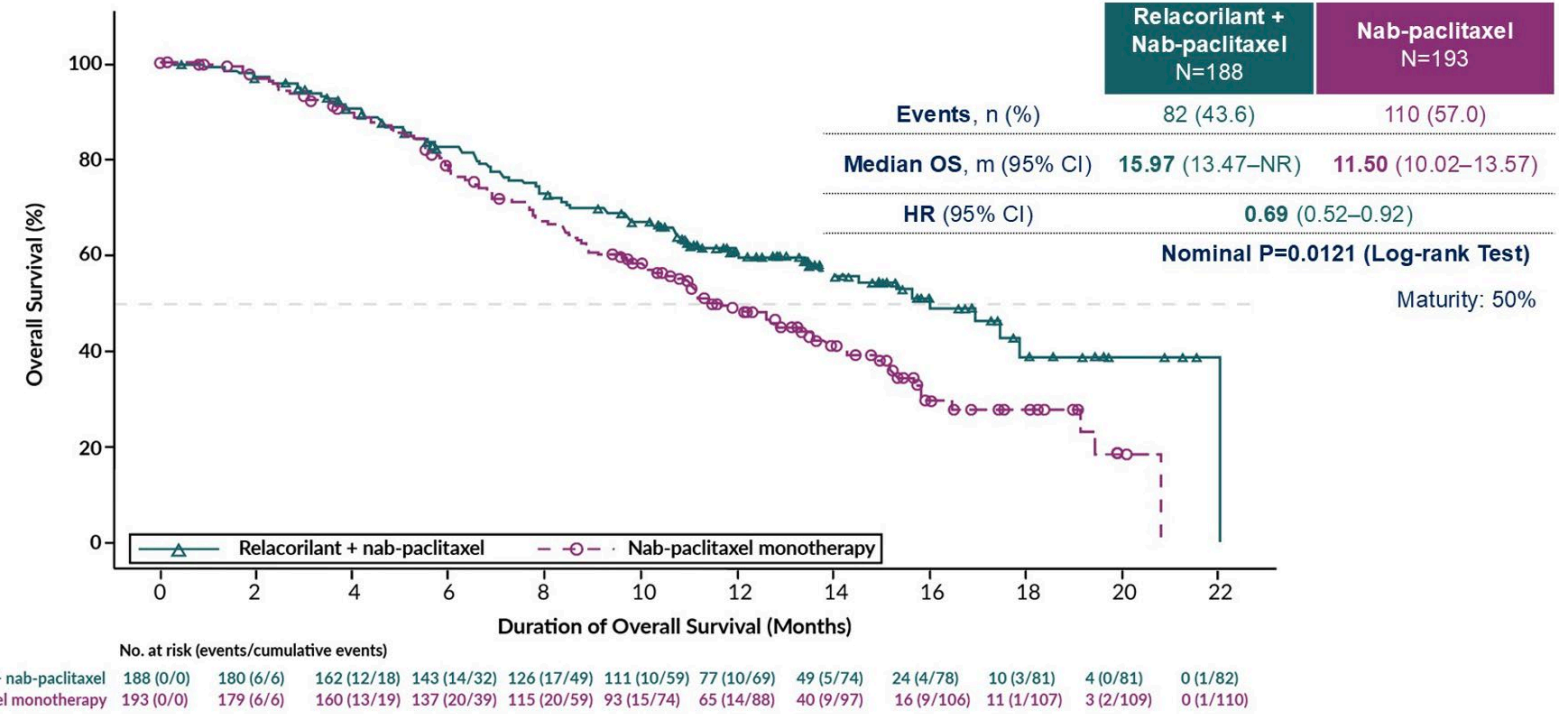
Relacorilant Significantly Improved PFS Assessed by Blinded Review



Median follow-up time 9.0 months; statistical significance threshold, *P*=0.04. The Kaplan–Meier method was used to estimate the curves; median estimates and the 95% CIs for PFS in each treatment arm. The HR and the associated 95% CI were estimated using a Cox regression model with treatment group as the main effect and stratification factors at randomization as covariates. Data cutoff: Feb 24, 2025.

Olawaiye A, et al. *J Clin Oncol.* 2025;43:LBA5507.

Relacorilant Improved OS at This Interim Analysis



Median follow-up time: 13.9 months; statistical significance threshold at the interim analysis: *P*≤0.0001; statistical significance threshold at the final analysis: *P*≤0.0499. The Kaplan–Meier method was used to estimate the curves; median estimates and the 95% CIs for OS in each treatment arm. The HR and the associated 95% CI were estimated using a Cox regression model with treatment group as the main effect and stratification factors at randomization as covariates. Data cutoff: Feb 24, 2025.

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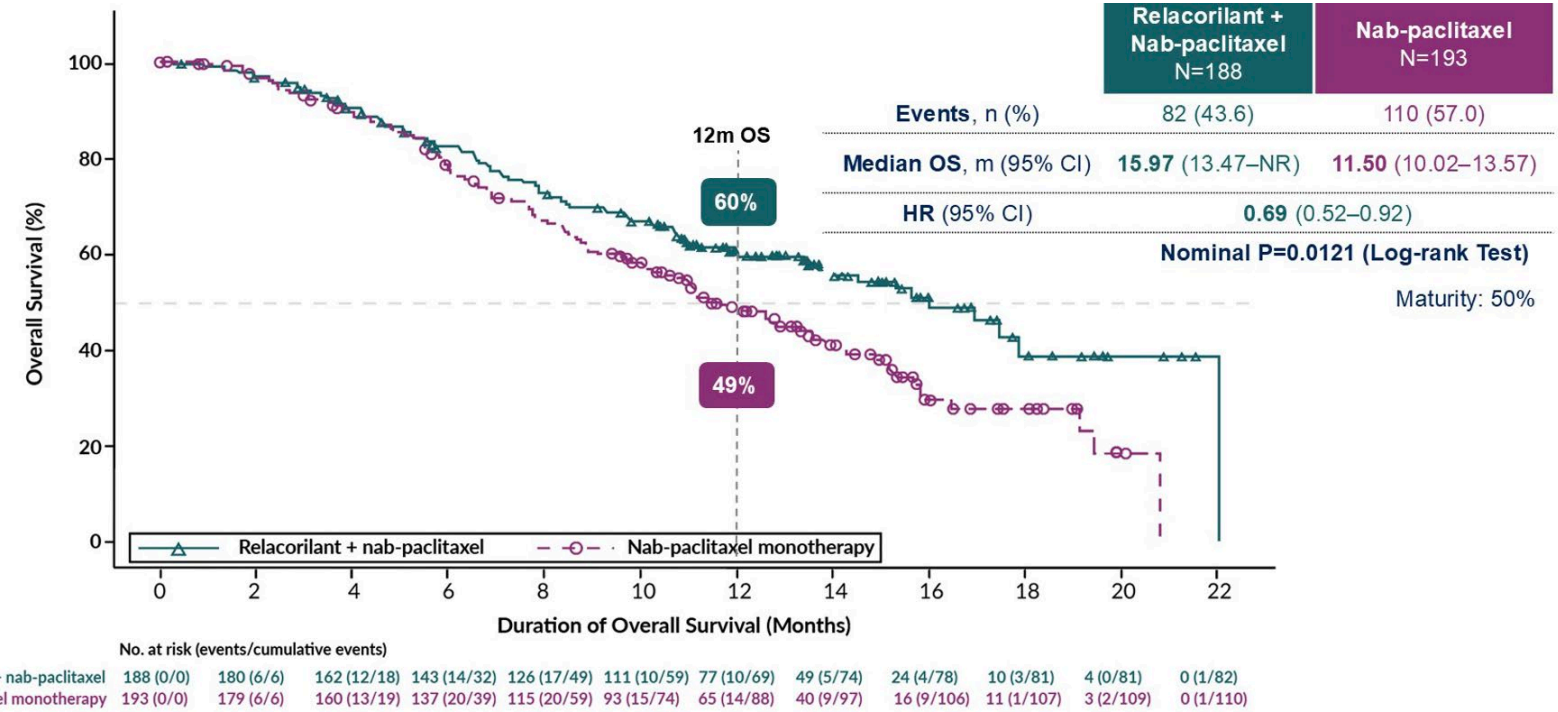

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Olawaiye A, et al. *J Clin Oncol.* 2025;43:LBA5507.

Relacorilant Improved PFS & OS across Subgroups

Subgroup		Patients, n	Events, n	Hazard Ratio for PFS (BICR), (95% CI)	Events, n	Hazard Ratio for OS, (95% CI)
All Patients		381	234	0.70 (0.54–0.91)	192	0.69 (0.52–0.92)
Age	<65 years	229	140	0.76 (0.54–1.08)	119	0.83 (0.57–1.20)
	≥65 years	152	94	0.61 (0.40–0.94)	73	0.55 (0.34–0.89)
Region	North America	90	56	0.62 (0.36–1.07)	45	0.69 (0.38–1.27)
	Europe	216	130	0.73 (0.52–1.04)	111	0.67 (0.46–0.98)
	Korea, Australia, Latin America	75	48	0.70 (0.39–1.26)	36	0.76 (0.39–1.48)
ECOG Performance Status	0	262	154	0.72 (0.52–1.00)	118	0.72 (0.50–1.05)
	1	115	80	0.62 (0.39–0.98)	74	0.59 (0.36–0.97)
Prior Lines of Therapy	1	33	21	0.88 (0.35–2.22)	21	0.80 (0.32–1.97)
	2	181	119	0.63 (0.43–0.91)	91	0.74 (0.49–1.12)
	3	167	94	0.71 (0.47–1.08)	80	0.66 (0.42–1.04)
Prior PARP Inhibitor	Yes	234	138	0.60 (0.42–0.85)	116	0.77 (0.53–1.13)
	No	147	96	0.84 (0.55–1.28)	76	0.66 (0.42–1.05)
Primary Platinum-free Interval	≤6 months	112	73	0.50 (0.30–0.84)	62	0.52 (0.31–0.89)
	>6 months	269	161	0.78 (0.57–1.06)	130	0.82 (0.58–1.16)
BRCA1/2 Mutation	Positive	47	32	1.08 (0.49–2.37)	23	0.82 (0.33–2.07)
	Negative / Unknown	334	202	0.65 (0.49–0.87)	169	0.70 (0.52–0.96)
Largest Target Lesion	<5 cm	299	181	0.68 (0.51–0.92)	141	0.65 (0.46–0.91)
	≥5 cm	45	30	0.50 (0.23–1.09)	25	0.58 (0.25–1.34)

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Olawaiye A, et al. *J Clin Oncol.* 2025;43:LBA5507.

Relacorilant + Nab-Paclitaxel Was Associated with High Objective Response and CBRs (by Investigator)

Endpoint	Relacorilant + Nab-paclitaxel	Nab-paclitaxel
Objective Response Rate, n (%)	69 **(36.9)**	58 **(30.1)**
	6.8% improvement P=0.17 (Stratified Cochran-Mantel-Haenszel Test)	
Complete Response, n (%)	6 **(3.2)**	4 **(2.1)**
Partial Response, n (%)	63 **(33.7)**	54 **(28.0)**
Stable Disease, n (%)	77 **(41.2)**	68 **(35.2)**
Progressive Disease, n (%)	32 **(17.1)**	52 **(26.9)**
Not Evaluable, n (%)	9 **(4.8)**	15 **(7.8)**
Clinical Benefit Rate, n (%) (Response or stable disease maintained for 24 weeks)	96 **(51.1)**	75 **(38.9)**
	12.2% improvement P=0.016 (Stratified Cochran-Mantel-Haenszel Test)	

ORR was assessed in the subset of ITT population with measurable disease at baseline, per investigator assessment (n=380 patients). CBR was assessed in the intent-to-treat population (n=381 patients). Per RECIST v1.1 guidelines confirmatory scans were not required for this randomized controlled trial.

Olawaiye A, et al. *J Clin Oncol.* 2025;43:LBA5507.

Safety Summary

Relacorilant + Nab-Paclitaxel was Well-Tolerated, with a Favorable Safety Profile

Safety Population Who Received at Least One Dose of Study Drug (N=378)	Relacorilant + Nab-paclitaxel (N=188)	Nab-paclitaxel (N=190)
Weeks of Nab-paclitaxel Therapy, mean (range)	**23.2** (0.1–90.3)	**18.6** (0.1–68.1)
Any TEAEs, n (%)	188 **(100)**	189 **(99.5)**
Grade ≥3 TEAEs, n (%)	140 **(74.5)**	113 **(59.5)**
Serious AEs, n (%)	66 **(35.1)**	45 **(23.7)**
All Deaths on Treatment or Within 30 Days of the Last Dose, n (%)	10 **(5.3)**	8 **(4.2)**
Dose Reductions of Relacorilant Due to TEAEs, n (%)	13 **(6.9)**	—
Dose Reductions of Nab-paclitaxel Due to TEAEs, n (%)	91 **(48.4)**	60 **(31.6)**
Interruptions of Nab-paclitaxel (+ Relacorilant) Due to TEAEs, n (%)*	137 **(72.9)**	104 **(54.7)**
Discontinuations of Nab-paclitaxel (+ Relacorilant) Due to TEAEs, n (%)*	17 **(9.0)**	15 **(7.9)**

*Relacorilant was always interrupted or discontinued when nab-paclitaxel was interrupted or discontinued. AEs, adverse events; TEAEs, treatment-emergent adverse events.

AEs leading to treatment discontinuation in >2 patients included intestinal obstruction and paresthesia. There were no relacorilant-related fatal AEs.

Olawaiye A, et al. *J Clin Oncol.* 2025;43:LBA5507.

Common (>20%) AEs

Peripheral neuropathy occurred with similar frequency in both arms (19.1% and 17.4%).
5 SAEs of febrile neutropenia were reported, 4 (2.1%) with relacorilant + nab-paclitaxel and 1 (0.5%) with nab-paclitaxel monotherapy.
5 SAEs of sepsis were reported, 3 (1.6%) with relacorilant + nab-paclitaxel and 2 (1.1%) with nab-paclitaxel monotherapy.

TEAEs that occurred in ≥20% of patients. Assessed in the safety population of patients who received at least one dose of study drug, N=378. Combined terms are presented for neutropenia (neutropenia, reduced neutrophil count, and febrile neutropenia), anemia (anemia, reduced hemoglobin, and reduced red blood cell count) and fatigue (fatigue and asthenia).

SAEs = serious adverse events.

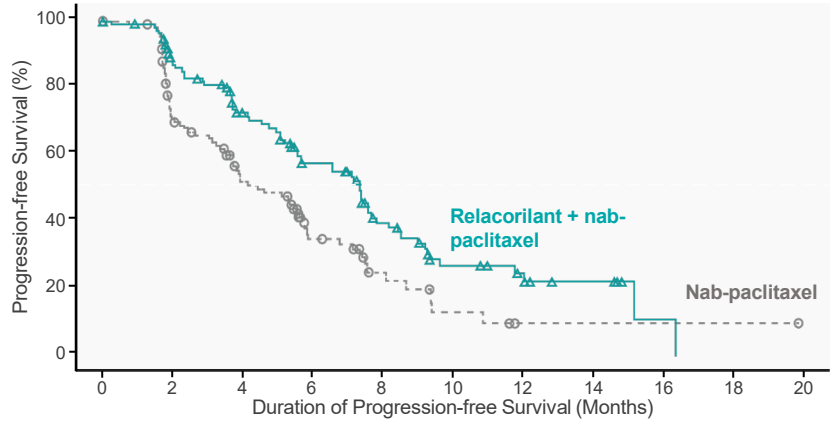
Olawaiye A, et al. *J Clin Oncol.* 2025;43:LBA5507.

ROSELLA (GOG-3073/ENGOT-OV72/MITO)

Phase 3 Study of Relicorilant + Nab-Paclitaxel in Ovarian Carcinoma

PFS[a] in Subgroup of Patients Who Received Prior PARP Inhibitor

(data cutoff: February 24, 2025)



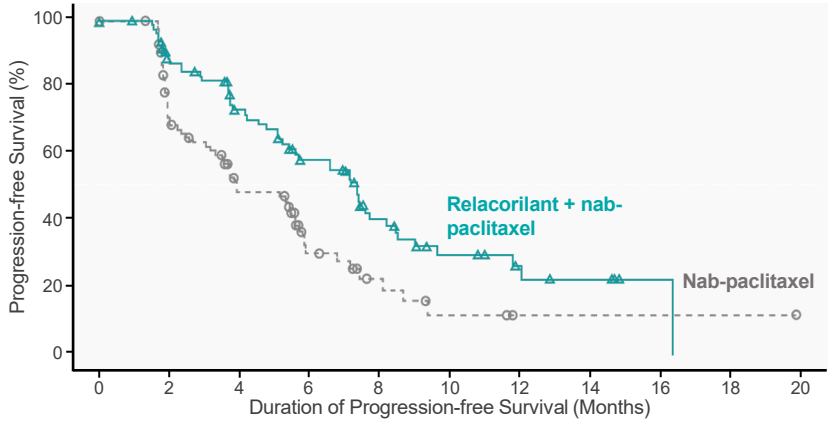
No. at risk (events/cumulative events)

REL + NAB-PAC	114 (0/0)	90 (12/12)	66 (16/28)	46 (13/41)	27 (13/54)	15 (8/62)	10 (1/63)	6 (1/64)	6 (1/65)	0 (1/66)
NAB-PAC	120 (0/0)	74 (30/30)	47 (19/49)	22 (13/62)	10 (5/67)	4 (4/71)	1 (1/72)	1 (0/72)	1 (0/72)	0 (0/72)

	Relacorilant + nab-paclitaxel (n = 114)		Nab-paclitaxel (n = 120)	
Progression-Free Survival *(by BICR)*				
Events, n %	66	57.9%	72	60.0%
Median PFS (95% CI)	**7.36 months** (5.59, 8.18)		**4.63 months** (3.55, 5.72)	
HR (95% CI)	**0.60** (0.42, 0.85)			
Nominal P-value[b]	**0.0035**			
ORR *(by investigator)*				
n %	45	**39.5%**	37	**30.8%**

PFS[a] in Subgroup of Patients Who Progressed on PARP Inhibitor

(data cutoff: February 24, 2025)



No. at risk (events/cumulative events)

REL + NAB-PAC	86 (0/0)	67 (9/9)	50 (11/20)	36 (10/30)	21 (10/40)	12 (5/45)	7 (1/46)	5 (1/47)	6 (0/47)	0 (1/48)
NAB-PAC	97 (0/0)	58 (24/24)	34 (17/41)	14 (10/51)	7 (3/54)	3 (3/57)	1 (0/57)	1 (0/57)	1 (0/57)	0 (0/57)

	Relacorilant + nab-paclitaxel (n = 86)		Nab-paclitaxel (n = 97)	
Progression-Free Survival *(by BICR)*				
Events, n %	48	55.8%	57	58.8%
Median PFS (95% CI)	**7.36 months** (5.39, 8.44)		**3.94 months** (3.32, 5.72)	
HR (95% CI)	**0.56** (0.37, 0.84)			
Nominal P-value[b]	**0.0046**			
ORR *(by investigator)*				
n %	30	**34.5%**	26	**26.8%**

a. By BICR (blinded-independent central review). The Kaplan–Meier method was used to estimate the curves, median estimates, and the 95% CI for PFS (progression-free survival) in each treatment arm. The HR and the associated 95% CI were estimated using a Cox regression model with treatment group as the main effect and stratification factors at randomization as covariates. ORR (overall response rate) was assessed among patients with baseline measurable disease. b. Log-rank test.

1. Lorusso D, et al. Presented at: European Society For Medical Oncology (ESMO) 2025; October 17 -21, 2025; Berlin, Germany. Abstract LBA45.

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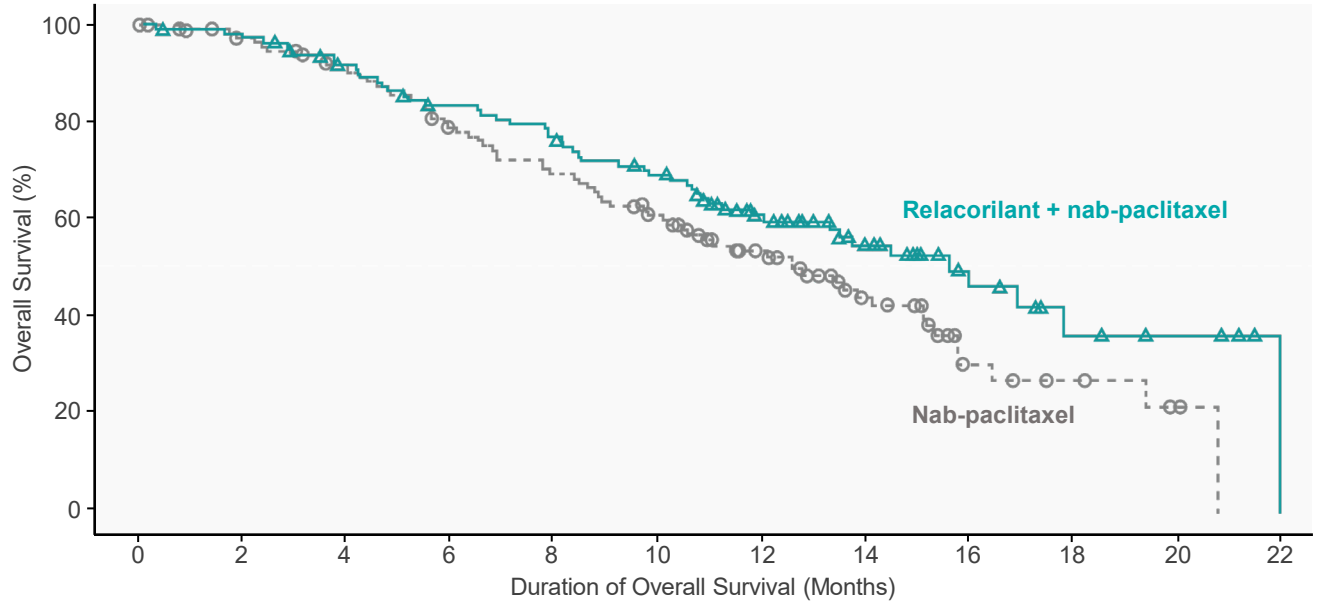
ROSELLA (GOG-3073/ENGOT-OV72/MITO)

Phase 3 Study of Relicorilant + Nab-Paclitaxel in Ovarian Carcinoma

Kaplan-Meier Estimate[a] of Overall Survival (OS) in Patients With Prior PARP Inhibitor Treatment (Interim Analysis)

(data cutoff: February 24, 2025)



No. at risk (events/cumulative events)

REL + NAB-PAC	114 (0/0)	109 (3/3)	99 (6/9)	88 (9/18)	81 (7/25)	71 (8/33)	48 (8/41)	31 (4/45)	14 (3/48)	6 (2/50)	4 (0/50)	0 (1/51)
NAB-PAC	120 (0/0)	110 (3/3)	99 (6/9)	83 (14/23)	73 (10/33)	61 (9/42)	45 (7/49)	26 (7/56)	9 (6/62)	6 (1/63)	3 (1/64)	0 (1/65)

Interim Analysis, Maturity 50%

	Relacorilant + nab-paclitaxel n=114		Nab-paclitaxel n=120	
Events, n %	51	44.7%	65	54.2%
Median OS (95% CI)	15.61 months (12.02, NR)		12.58 months (10.09, 15.18)	
HR (95% CI)	**0.77** (0.53, 1.13)			
Nominal P-value[b]	0.1834			

a. The Kaplan-Meier method was used to estimate the curves, median estimates, and the 95% CI for OS in each treatment arm. The HR and the associated 95% CI were estimated using a Cox regression model with treatment group as the main effect and stratification factors at randomization as covariates. b. Log-rank test.
NR = not reached

1. Lorusso D, et al. Presented at: European Society For Medical Oncology (ESMO) 2025; October 17 -21, 2025; Berlin, Germany. Abstract LBA45.

ROSELLA: Conclusions

1 **ROSELLA met its primary endpoint of improving PFS**	Relacorilant, **a first-in-class, oral, SGRA, extended progression-free survival** by BICR (log-rank test P=0.0076, HR 0.70) compared to nab-paclitaxel monotherapy in patients with platinum-resistant ovarian cancer, in a population including patients who progressed within 1–3 months after their primary platinum regimen
2 **Median survival prolonged by 4.5 months**	**At this interim overall survival analysis, the addition of relacorilant to nab-paclitaxel showed a clinically meaningful improvement in overall survival** (nominal log-rank test P=0.0121, HR 0.69, median 16.0 vs 11.5 months)
3 **Well-tolerated, favorable safety profile**	Relacorilant plus nab-paclitaxel was well-tolerated, with a favorable safety profile that was comparable between treatment arms when adjusted for duration of exposure. The safety profile was consistent with previously reported data; no new signals were identified
4 **A new standard for PROC**	Intermittently dosed relacorilant plus nab-paclitaxel offers an efficacious treatment regimen for women with platinum-resistant ovarian cancer, without the need for a biomarker

Olawaiye A, et al. *J Clin Oncol.* 2025;43:LBA5507.

Overall Survival Primary Endpoint Met in Phase 3 ROSELLA Trial of Relacorilant in Patients with Platinum-Resistant Ovarian Cancer

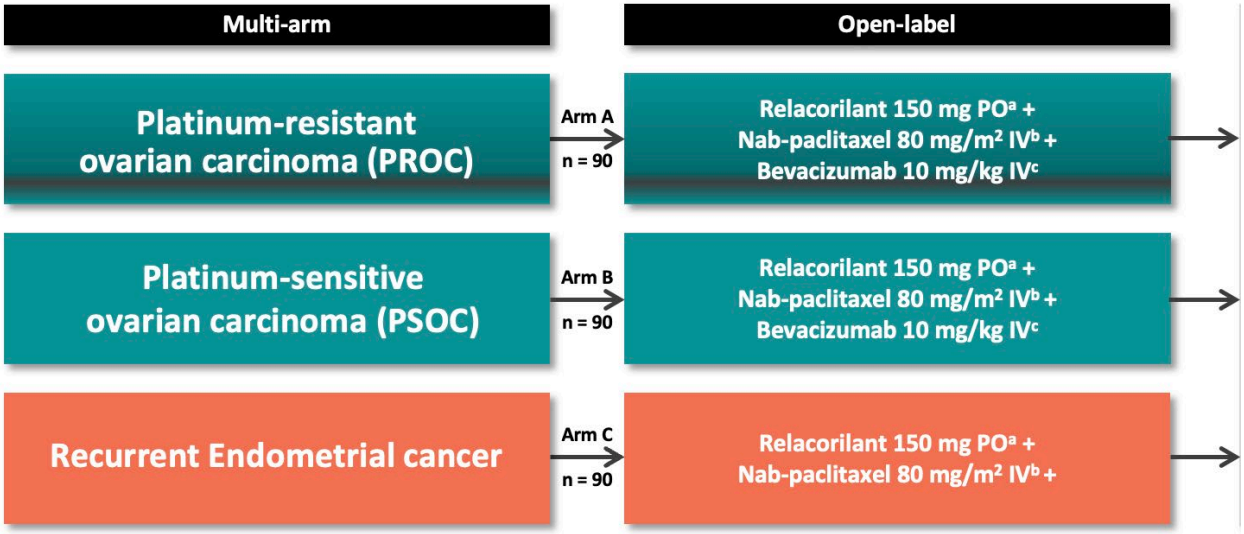
January 22, 2026

- Data demonstrate a 35 percent reduction in the risk of death
- Both dual primary endpoints (progression-free and overall survival) were met, without the need for biomarker selection and without increased safety burden
- Relacorilant's New Drug Application (NDA) is under review by the US Food and Drug Administration (FDA) as a treatment for patients with platinum-resistant ovarian cancer with a Prescription Drug User Fee Act (PDUFA) target action date of July 11, 2026
- Relacorilant's Marketing Authorization Application (MAA) for patients with platinum-resistant ovarian cancer is also under review by the European Medicines Agency (EMA)

BusinessWire [www.businesswire.com]. Last updated January 22, 2026. https://finance.yahoo.com/news/overall-survival-primary-endpoint-met-130000681.html.

Ongoing Clinical Trials
Relacorilant In Gynecologic Oncology

BELLA: Phase 2 Study of Relacorilant + Nab-Paclitaxel +/- Bevacizumab in Pts with Gynecologic Cancers



- Primary endpoints
  - PFS[b]
- Secondary endpoints include
  - Efficacy
    - ORR
    - DoR
    - CBR at 24 weeks
    - OS
  - Safety

[a]Once on the day before, once the day of, and once the day after nab-paclitaxel (Cycle 1: Once the day of, and once the day after nab-paclitaxel); [b]By Investigator and RECIST v1.1.

Conclusions

Biologic Discoveries in Ovarian Cancer Tumorigenesis Can Inform Novel Therapy Development and Combination Approaches to Treatment

Antibody Drug Conjugates

- **Mirvetuximab soravtansine [FRα]**; phase 3[16]
- **Trastuzumab deruxtecan [HER2]** + bev; phase 3[17]
- **Rinatabart sesutecan [FRα]**; phase 3[18]
- **Raludotatug deruxtecan [CDH6]**; phase 2/3[19]

Immune Checkpoint Inhibitors

- **Dostarlimab [PD-1]** + SOC +/- PARPi; phase 3[2]
- **Pembrolizumab [PD-1];** + pac +/- bev; phase 3[3]

Bispecific Antibodies

- **Ubamatamab [MUC16/CD3];** phase 2[4]
- **CTIM-76 [CLDN6/CD3];** phase 1a/1b[5]
- **Navicixizumab [DLL4/VEGF]** + pac; phase 1b[6]

Oncolytic Virus Therapy (OVT)

- **Olvi-Vec** + SOC; phase 3[7]
- **TILT-123 [TNF/IL-2 production]** + pembro; phase 1a[8]

DNA-Based Immunotherapy

- **IMNN-001 [IL12 production]** + SOC; phase 3[9]

Engineered Cytokines

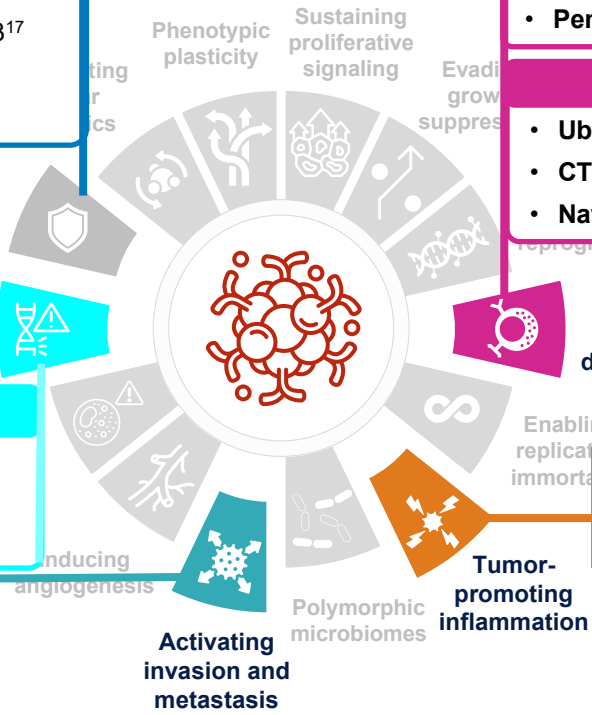
- **ALKS-4230 [IL-2 mimic]** + pembro; phase 3[10]
- **N-803 [IL-15 mimic]** + M-CENK + gem; phase 2[11]

DNA Damage Response Therapies

- **GSK4524101 [POLθ]** +/- PARPi; phase 1[13]
- **LY2606368 [CHK1];** phase 2[14]
- **KSQ-4279 [USP1]** +/- PARPi/carbo; phase 1[15]

Antiglucocorticoid Therapies

- **Relacorilant [GR]** + nab-pac; phase 3[12]



CDH6 = cadherin-6; CHK1 = checkpoint kinase 1; CLDN6 = claudin-6; DLL4 = delta-like canonical notch ligand 4; FOLR1/ FRα = folate receptor alpha; GR = glucocorticoid receptor; HER2 = human epidermal growth factor receptor 2; IL = interleukin; MUC16 = mucin 16; PD-1 = programmed cell death protein 1; POLθ = DNA polymerase theta; USP1 = ubiquitin-specific peptidase 1; VEGF = vascular endothelial growth factor; SOC = standard of care.

ClinicalTrials.gov identifiers: 2. NCT05855200; 3. NCT02142738; 4. NCT06444880; 5. NCT06515613; 6. NCT05043402; 7. NCT05281471; 8. NCT05271318; 9. NCT06915025; 10. NCT05092360. 11. NCT06710288; 12. NCT05257408; 13. NCT06077877; 14. NCT02203513; 15. NCT05240898; 16. NCT04209855; 17. NCT06819007; 18. NCT06619236; 19. NCT06161025.

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Conclusions

- Many options for PROC patients
- Mirvetuximab single-agent and consider combination therapy
- Trastuzamab deruxtecan—exciting data
- Pembrolizumab + paclitaxel +/- bevacizumab—FDA approval
- Glucocorticoid pathway
  - Novel target
  - Imminent FDA review
  - Phase 2/phase 3 data positive
- Many ongoing clinical trails
- Sequencing—TBD
- ADC after ADC—TBD
- FDA—SOC arm?

TBD = to be determined.

Thank You

Questions