Abstract 172 Table 1 Summary of TEAEs in patients aged, <70 and ≥70 years

n (%)	Aged <70 years (n=192)		Aged ≥70 years (n=87)	
	All grades	Grade ≥3	All grades	Grade ≥3
Any TEAE*	183 (95)	53 (28)	84 (97)	28 (32)
Nausea	93 (48)	0	42 (48)	1 (1)
Fatigue/asthenia†	82 (43)	4 (2)	41 (47)	5 (6)
Anaemia†	73 (38)	26 (14)	36 (41)	12 (14)
Dysgeusia	32 (17)	0	7 (8)	0
Vomiting	28 (15)	2 (1)	17 (20)	1 (1)
Neutropenia†	29 (15)	2 (1)	15 (17)	3 (3)
Abdominal pain	26 (14)	0	10 (11)	0
Diarrhoea	25 (13)	0	15 (17)	0
Thrombocytopenia†	23 (12)	2 (1)	12 (14)	4 (5)
Cough	22 (11)	0	7 (8)	0
Decreased appetite	18 (9)	0	14 (16)	0
Arthralgia	16 (8)	0	10 (11)	0
Urinary tract infection	13 (7)	0	14 (16)	0
Back pain	10 (5)	0	15 (17)	0
Serious TEAE	37 (19)	–	18 (21)	–
Dose interruption due to TEAE	83 (43)	–	48 (55)	–
Dose reduction due to TEAE	38 (20)	–	25 (29)	–
Treatment discontinuation due to TEAE	12 (6)	–	9 (10)	–

*Data are shown for TEAEs occurring in >10% of patients aged <70 or ≥70 years.

†Grouped term.

TEAE, treatment-emergent adverse event.

(median 74.0 years; range 70–85). Among patients aged <70 vs ≥70 years, 73% vs 59% were Eastern Cooperative Oncology Group (ECOG) performance status 0, 27% vs 41% were ECOG performance status 1, 64% vs 49% had received only two prior platinum regimens, 36% vs 51% had received ≥3 prior platinum regimens, and 35% vs 28% had a complete response and 64% vs 70% had a partial response to their latest platinum regimen. At data cut-off (2 Oct 2020), median PFS and 18-month PFS rates were similar in both age groups (figure 1). Median TFST was 15.6 months (95% CI 12.2–18.1) in patients aged <70 years and 11.4 months (95% CI 9.7–15.6) in patients ≥70 years. The safety profile of maintenance olaparib (median treatment duration 9 months) was generally similar in both age groups (table 1).

Conclusion* Efficacy and safety data support the use of maintenance olaparib in non-gBRCAm PSR OC patients irrespective of age.

175 COST-EFFICIENT ANALYSIS OF BRCANESS STATUS IN HIGH-GRADE SEROUS OVARIAN CARCINOMAS

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Introduction/Background* Paclitaxel/carboplatin (TCbP) is a standard therapy for high-grade serous ovarian cancer (HGSOC), however many patients do not benefit from this combination. **Methodology** Genetic profiling was performed for 71 HGSOC consecutive patients, who received neoadjuvant chemotherapy (NACT).

Result(s)* BRCA1/2 germline mutation carriers (n = 22) had longer treatment-free interval (TFI) than non-carriers (n = 49) (9.5 vs. 3.8 months; P = 0.007). 51 HGSOCs with sufficient quality of tumor DNA were examined by the SeqCap EZ CNV/LOH Backbone Design NGS panel, which systematically

spans the entire genome at 50 kb intervals. All 13 tumors obtained from BRCA1/2 germline mutation carriers and 12 sporadic HGSOCs had high number of evenly spread chromosomal breaks, that was defined as a BRCAness phenotype; median TFI for this combined group approached 9.5 months. The remaining 26 HGSOCs had similarly high global LOH score (above 20%); however, in contrast to BRCAness tumors, LOH involved large chromosomal segments; these patients had significantly lower TFI (3.7 months; P = 0.006). Comparison between this newly developed BRCAness test, which discriminated tumors simply by the number of affected genomic segments, and the commonly accepted HRD scoring system, revealed high concordance of the results and at least non-inferior clinical performance of our assay. Virtually all tumors with BRCAness (23/25 [92%]) demonstrated gain at MYC locus, while this event was less common in non-BRCAness HGSOCs (12/26 [46%]; P = 0.0006). All patients with CCNE1 amplification (n = 7), TP53 R175H substitution (n = 6), and RB1 mutation (n = 4) had poor response to TCbP. **Conclusion*** BRCA1/2 germ-line testing has superior performance in identifying responders to TCbP. Simple and rapid PCR-based tests for MYC and CCNE1 amplification allow to classify patients for potential responders and non-responders with a reasonable level of accuracy. BRCAness phenotype can be reliably detected by a laboratory-scale NGS assay, which evaluates the total number of chromosomal breaks. It is of concern that TCbP is being routinely administered both to potential responders and to potential non-responders to this scheme. Novel treatment options for the latter category of HGSOC patients need to be searched within preclinical and clinical studies.

177 EFFICACY OF NIRAPARIB BY TIMING OF SURGERY AND RESIDUAL DISEASE: A POST-HOC ANALYSIS OF PATIENTS IN THE PRIMA/ENGOT-OV26/GOG-3012 STUDY

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Introduction/Background* Niraparib is a poly(ADP-ribose) polymerase (PARP) inhibitor approved as maintenance treatment for patients (pts) with newly diagnosed advanced or recurrent ovarian cancer following a response to platinum-based chemotherapy (CT) doublet. The PRIMA/ENGOT-OV26/GOG-3012 (NCT02655016) study showed that niraparib following first-line treatment improved progression-free survival (PFS) in the overall intention-to-treat (ITT) population (hazard ratio [HR] 0.62; 95% CI 0.50–0.76).

Methodology This double-blind, placebo (PBO)-controlled, phase 3 trial evaluated niraparib in pts with newly diagnosed, advanced, high-grade serous or endometrioid ovarian, primary peritoneal, or fallopian tube cancer with a complete or partial response to first-line CT. Pts were considered to be at a high risk for disease progression based on their clinical characteristics. This post-hoc analysis presents the efficacy of niraparib, measured by PFS, based on time of surgery and residual disease status, and was not powered to determine differences among the subgroups.

Result(s)* Data cutoff was May 2019. In total, 733 pts were randomized in the PRIMA study. Efficacy outcomes by surgical timing, either primary debulking surgery (PDS) or interval debulking surgery (IDS), and postoperative residual disease status, either no visible residual disease (NVRD) or visible residual disease (VRD), are shown in table 1. Pts who underwent PDS or IDS had similar efficacy with niraparib maintenance treatment versus PBO in the ITT population (PFS HRs were 0.67 and 0.57, respectively). Niraparib treatment reduced risk of progression by 42% in pts who received PDS and had VRD, 35% in those with IDS and NVRD, and 59% in those with IDS and VRD. Efficacy was not evaluable for pts with PDS and NVRD due to low sample size.

Conclusion* In this post-hoc analysis, the impact of residual disease after PDS or IDS on the efficacy of niraparib was comparable across subgroups. Pts with IDS and VRD had the highest reduction in the risk of progression.

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REAL-WORLD TREATMENT PATTERNS AND OUTCOMES WITH FIRST-LINE MAINTENANCE OLAPARIB FOR BRCAm ADVANCED OVARIAN CANCER: A PAN-EUROPEAN STUDY (OVAL-1)

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Introduction/Background* Standard therapy for advanced ovarian cancer (OC) includes radical debulking surgery followed by first-line platinum-based chemotherapy, although neo-adjuvant chemotherapy can be used. Most women with newly diagnosed advanced OC relapse within 3 years of standard treatment.

In SOLO1, patients with advanced OC and a *BRCA1* and/or *BRCA2* mutation (BRCAm), in complete or partial response following first-line platinum-based chemotherapy, received maintenance olaparib or placebo for up to 2 years or until progression. At 5-year follow-up, median progression-free survival (PFS) was 56 months with olaparib vs 14 months with placebo (hazard ratio 0.33; 95% confidence interval 0.25–0.43). The OVAL-1 study will provide evidence on real-world effectiveness of olaparib in patients with BRCAm advanced OC treated in the first-line maintenance setting in France, Italy and the UK.

Methodology Retrospective cohort, pan-European multicentre observational study with data abstracted from medical records at several time points until ≥ 3 years after first olaparib dose (index date).

Eligible patients are adult females with a BRCAm and advanced (FIGO stage III/IV) OC, who received their first olaparib (tablet) dose between January 2019 and June 2020 in

Abstract 177 Table 1 Efficacy results by time of surgery and visible residual disease status

		ITT	NVRD (R0)	VRD (R1/R2)
All patients	N HR (95% CI) P mPFS (nir vs PBO) Δ mPFS	733* 0.62 (0.5–0.76) P<0.0001 13.8 vs 8.2 5.6		
PDS	N HR (95% CI) mPFS (nir vs PBO) Δ mPFS	236** 0.67 (0.468–0.964) 13.7 vs 8.2 5.5	37 NE NE NE	183 0.58 (0.391–0.864) 11.8 vs 7.8 4
IDS/NACT	N HR (95% CI) mPFS (nir vs PBO) Δ mPFS	481*** 0.57 (0.441–0.731) 14.2 vs 8.2 6	304 0.65 (0.461–0.91) 18.2 vs 10.9 7.3	149 0.41 (0.269–0.620) 11.1 vs 5.6 5.5

*16 patients had no debulking surgery.

**16 patients had unknown residual disease status.

***28 patients had unknown residual disease status.

HR, hazard ratio; IDS, interval debulking surgery; ITT, intention-to-treat; mPFS, median progression-free survival; NACT, neoadjuvant chemotherapy; NE, not evaluable; nir, niraparib; NVRD, no visible residual disease; PBO, placebo; PDS, primary debulking surgery; VRD, visible residual disease.