Result(s) Of 806 patients enrolled, 142 (17.6%) were ≥70 years old; 104 of these were in the olaparib arm. There was no notable difference in baseline characteristics between olaparib and placebo arms (table 1). In evaluation of safety, moderately increased rates of grade ≥3 treatment-related AEs (TRAEs), dose reductions and interruptions, and treatment discontinuation due to TRAEs were reported in older than younger patients (table 2). Grade 3–4 any-cause anaemia (21.2% vs 16.5%) and neutropenia (9.7% vs 5.1%) were more frequent in older patients. The most common grade 3–4 non-haematological AE was hypertension (26.9% vs 16.7%). No acute myeloid leukaemia, myelodysplastic syndrome, or treatment-related death was reported among older patients.

Interestingly, GHS during the first 2 years of maintenance was similar between arms. Median PFS was 21.1 months with olaparib vs 14.3 months with placebo. Analyses by GVS and homologous recombination deficiency subgroups are ongoing and will be presented.

Conclusion Among older patients in PAOLA-1, olaparib plus bevacizumab maintenance had a manageable safety profile and had no adverse impact on GHS. Median PFS in older patients was similar to the overall population. Analyses stratified by GVS will provide further insight into the safety profile of maintenance olaparib plus bevacizumab in older patients.
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 similar 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-BRCA- ness 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.