Introduction Microbiome plays an important role in development of cancer and response to chemotherapy. We aim to examine the significance of vaginal microbiome in epithelial ovarian cancer.

Methods A prospective cohort study was conducted for evaluating the vaginal microbiome in newly diagnosed epithelial ovarian cancer (NEOC) patients, post-chemotherapy (PC) patients and healthy women. Samples were collected using a swab. DNA was extracted and amplified by PCR using universal primers of the prokaryotic 16S ribosome. Next-generation sequencing and taxonomical classification was then performed.

Results Vaginal swab samples were collected from 21 NEOC patients, 27 PC and 22 controls. The microbiome analysis revealed statistically significant findings. Clostridiales bacterium S5 A14a and Anaerovoracaceae family were found to be abundant in patient who had previous malignancies (p<0.01). Clostridiales bacterium S5 A14a was also abundant in patient who had no pregnancies in the past (p<0.001). Clostridia UCG 014 family was found prominent in patient who died of disease (p<0.01).

Conclusion/Implications We demonstrated a significant difference in vaginal microbiome in EOC patients who had a history of other malignancies. Interestingly, the Clostridia species that are prominent are studied as a risk factor for developing tumors in PTEN carrier and Anaerovoracaceae was linked to esophageal cancer. We also demonstrated Clostridia UCG 014 abundance in patients that died of disease, a finding that was previously shown in mice studies and in patients with lung cancer. This may suggest a group of patients that can benefit from microbiome mapping as a screening tool and as a marker for poor prognosis.

Introduction Improved overall survival (OS) with PARP inhibitors maintenance therapy in platinum-sensitive relapsed ovarian cancer (PSROC) was not determined. We performed treatment-free survival (TFS) with quality-adjusted OS analyses to measure their effects.

Methods We pooled available data for TFS analysis from three trials (Study 19, SOLO2, and ARIEL3). OS was divided into time on protocol treatment exposure (T), time to subsequent treatment initiation or death (TFS), and time after the first subsequent therapy or death (REL). TFS with quality-adjusted OS analyses were calculated by multiplying mean time in each health state by its assigned utility (quality-adjusted OS = uT + TFS + uREL × REL) for two cohorts: all (BRCAm, BRCAwt, or unknown) or BRCAm patients, the area under each KM curve estimated using restricted mean time with threshold utility analyses.

Results Restricted mean treatment duration was longer with PARP inhibitors than with placebo (all patients: 16.3 vs. 7.4 months, difference, 8.9 months, 95%CI 8.1–9.7; BRCAm: 17.1 vs. 8.1 months, 9.0, 8.1–9.9). Mean TFS was longer with PARP inhibitors (7.9 months) than with placebo (4.3 months; difference, 3.6, 2.0–5.1) among BRCAm patients. REL was longer with placebo (all patients: 11.7 vs. 20.5 months, -8.8, -10.1 to -7.6; BRCAm: 8.9 vs. 19.7 months, -10.8, -12.5 to -9.0). PARP inhibitors provided more quality-adjusted OS than placebo with a wider range of utility-weight values for BRCAm patients (-6.0 to +7.0 months) rather than all patients (-7.0 to +5.0 months).

Conclusion/Implications BRCAm patients rather than all-patients population benefited from PARP inhibitors maintenance therapy in PSROC.
2-stage design is utilized. Target accrual is 22 patients in the first stage; 13 or more patients without progressive disease within 6 months is required to proceed to second stage.

**Results** We report the results from the first stage. Median age was 56 years old, high grade serous and most of the patients (90.9%) had high-grade serous carcinoma. Of the 22 patients from the first stage, 9 had progressive disease within 6 months (6-month PFS rate 66.5%, 95% CI 48.9–90.2%). The efficacy boundary to proceed to the second stage was met. No grade 4 or 5 treatment-related adverse events (TREAs) were reported, and no TRAEs leading to treatment discontinuation.

**Conclusion/Implications** Our findings indicate encouraging safety and activity of niraparib + bevacizumab as a maintenance therapy in platinum-sensitive ovarian cancer patients who were previously treated with a PARPi.

### Abstract PR055/#936

**INFLUENCE OF SPATIAL TUMOUR HETEROGENEITY ON HOMOLOGOUS RECOMBINATION DEFICIENCY SCORES FOR HIGH GRADE SEROUS OVARIAN CANCER PATIENTS**

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**Introduction** Extensive genomic instability and heterogeneity are characteristics of high-grade serous ovarian cancer (HGSOC), with deficiency in homologous recombination (HR) repair has been reported for approximately 50% of HGSOC patients. Testing tumours for HR-deficiency (HRD) status is now a clinically applicable test, with a HR score of >42 suggestive of HRD and thus, platinum or PARP-inhibitor sensitivity in HGSOC. We aimed to determine the influence of spatial heterogeneity on HR scores in disseminated HGSOC.

**Methods** An algorithm detecting genomic scar HR scores was applied to genomic data from three cohorts of multi-site tumour samples – in-house Hammersmith hospital (HH) 45 patients n=5 tumours per patient; GSE38787 (n=24 patients) and GSE40546 (n=14 patients). A HR score is an unweighted sum of three independent measures of genomic instability: loss of heterozygosity (LOH), telomeric allelic imbalance (TAI), and large-scale state transition (LST). A cut-off >42 denotes HR-deficient.

**Results** Heterogeneity in tumour HR scores were detected in each cohort with patients presenting with either all HRD tumours, all HRP tumours or mixed HR scores, showing both HRD and HR-Proficient (HRP) tumour scores: HH (22%); GSE38787 (17%); and GSE40546 (28%). Within the HH cohort, survival analysis revealed that patients with all HRP tumours and mixed HR status had worse survival (progression-free survival p=0.0052; overall-survival p=0.00092) than patients with all HRD tumours.

**Conclusion/Implications** Our data demonstrating differences in HRD/HRP scores proposes that HR status may vary across disseminated HGSOC. Thus, implying that testing a single tumour biopsy may not accurately portray HGSOC tumour biology and could incorrectly guide clinical decision-making.

### Abstract PR056/#32

**CLINICAL CHARACTERISTICS AND ONCOLOGICAL OUTCOMES OF RECURRENT ADULT GRANULOSA CELL TUMOR OF OVARY: A RETROSPECTIVE STUDY OF SEVENTY PATIENTS**

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**Introduction** To describe the clinicopathological characteristics of recurrent adult granulosa cell tumor (AGCT) and identify the risk factors for recurrence.

**Methods** Seventy recurrent AGCT patients between 2000–2020 were retrospectively reviewed (figure 1). The primary outcomes were progression free survival after first recurrence (PFS-R), overall survival after first recurrence (OS-R) and recurrence frequency. The Kaplan-Meier (KM) analysis, Cox proportional hazard analysis, and the Prentice, Williams and Peterson counting process (PWP-CP) model were adopted.

**Results** The 5-year PFS-R was 29.3%, and the 5-year OS-R was 94.9%. KM analysis demonstrated that patients with distant recurrence and PFS1 ≤ 60 months had worse PFS-R (P<0.05), and patients with PFS-R ≤ 33 months had worse of heterozygosity (LOH), telomeric allelic imbalance (TAI), and large-scale state transition (LST). A cut-off >42 denotes HR-deficient.

**Results** Heterogeneity in tumour HR scores were detected in each cohort with patients presenting with either all HRD tumours, all HRP tumours or mixed HR scores, showing both HRD and HR-Proficient (HRP) tumour scores: HH (22%); GSE38787 (17%); and GSE40546 (28%). Within the HH cohort, survival analysis revealed that patients with all HRP tumours and mixed HR status had worse survival (progression-free survival p=0.0052; overall-survival p=0.00092) than patients with all HRD tumours.

**Conclusion/Implications** Our data demonstrating differences in HRD/HRP scores proposes that HR status may vary across disseminated HGSOC. Thus, implying that testing a single tumour biopsy may not accurately portray HGSOC tumour biology and could incorrectly guide clinical decision-making.