

tumours, 1 primary juvenile granulosa cell tumour and 1 primary Sertoli-Leydig cell tumour. Three samples were obtained from treatment-naïve GCT (2 immature teratomas and one dysgerminoma). For each phenotype of tumour cells, immune cells, endothelial cells and cancer-associated fibroblasts, we identified specific transcriptomic markers.

Results Based on differential expression analysis and expression of transcriptomic markers, we identified 27 clusters consisting of 9 tumour cell and 18 stromal cell clusters. The first results of subcluster analysis revealed nearly absence of B cells in all granulosa cell tumours. In addition, the immune cell subcluster mainly consists of T cells derived from the dysgerminoma (58%) and Sertoli-Leydig cell (20%) samples. Further characterisation and differentiation of distinct subclusters is currently ongoing and will be presented.

Conclusion With this analysis we aim to generate a publicly accessible comprehensive blueprint of the tumour micro-environment, aiding other researchers to gain high-resolution insights in the heterogeneity and complexity of these rare ovarian cancers.

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EFFICACY OF DOSTARLIMAB IN ENDOMETRIAL CANCER BY MOLECULAR SUBTYPE: A POST HOC ANALYSIS OF THE GARNET STUDY

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Introduction/Background Biomarkers are used to classify endometrial cancer (EC) into molecular subtypes such as TCGA and/or a surrogate classification (POLE mutated [mut], mismatch repair/microsatellite instability [MMR/MSI], TP53mut, and no specific mutation profile [NSMP]) or by estrogen receptor (ER) status. Here, we report on a post hoc analysis of objective response rate (ORR) by a surrogate classification for EC in patients receiving dostarlimab monotherapy.

Methodology GARNET is a multicentre, open-label, single-arm phase 1 study. Patients were assigned to cohort A1 (MMR deficient/MSI-high [dMMR/MSI-H EC]) or A2 (MMR

proficient/microsatellite stable [MMRp/MSS] EC) based on local assessment. Patients received 500 mg of dostarlimab IV Q3W for 4 cycles, then 1000 mg Q6W until disease progression, discontinuation, or withdrawal. The primary endpoints were ORR and duration of response by blinded independent central review. Molecular subtype was determined by POLE and TP53 mutation status by Foundation Medicine, and MMR/MSI status was determined by local immunohistochemistry or next-generation sequencing; all others were assigned as NSMP. The hierarchy for classification was POLEmut → MMR/MSI → TP53 status → NSMP. ER status was determined by local immunohistochemistry testing. Only patients with samples available for additional biomarker testing were included in the biomarker assessment.

Results 143 patients with dMMR/MSI-H EC and 156 patients with MMRp/MSS were included in the efficacy-evaluable population. ORRs were determined for molecular subtypes and ER expression (table 1). Safety has been previously reported.

Abstract 2022-RA-1194-ESGO Table 1

	A1	A2
Overall	65/143, 45.5% (37.1–54.0)	24/156, 15.4% (10.1–22.0)
Molecular subtype	N=101	N=153
Poimut	2/3, 66.7% (0.4–99.2)	0/2, 0% (0.0–84.2)
dMMR/MSI-H	43/98, 43.9% (33.9–54.3)	
	TP53mut 10/26, 38.5% (20.2–59.4)	TP53wt 33/72, 45.8% (34.0–58.0)
	ERpos 19/44, 43.2% (28.3–59.0)	ERneg 1/2, 50.0% (1.3–98.7)
		ERunk 23/52, 44.2% (28.3–59.0)
TP53mut		17/94, 18.1% (10.9–27.4)
		ERpos 4/28, 14.3% (4.0–32.7)
		ERneg 7/19, 36.8% (16.3–61.6)
		ERunk 6/47, 12.8% (4.8–25.7)
NSMP		7/57, 12.3% (5.1–23.7)
		ERpos 2/19, 10.5% (1.3–33.1)
		ERneg 2/11, 18.2% (2.3–51.8)
		ERunk 3/27, 11.1% (2.4–29.2)

Data are n/N, % (95% CI). Data cut: 1 November 2021.

dMMR, mismatch repair deficient; ERneg, ER negative; ERpos, ER positive; ERunk, ER unknown; MSI-H, microsatellite instability-high; mut, mutated; NSMP, no specific mutation profile; wt, wild-type.

Conclusion The observed ORRs in each molecular subgroup were consistent with the overall ORR in each cohort. Differences by ER expression status were not observed. These findings support the importance of testing patients with EC for MMR/MSI biomarker status as a predictor of response. Additionally, data suggest that TP53 mutation or ER expression should not modify treatment approach. The data are of interest for hypothesis generation.

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LONGITUDINAL STUDY OF VAGINAL MICROBIOME PRE- AND POST-TREATMENT IDENTIFIES BIOMARKERS FOR CERVICAL INTRAEPITHELIAL NEOPLASIA 3 (CIN3)

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Introduction/Background Increasing evidence suggests vaginal dysbiosis is associated with persistence of human papillomavirus (HPV) infection and cervical intraepithelial neoplasia (CIN1–3) development. In this pilot study we aimed to investigate the potential of vaginal microbiome biomarkers to predict CIN3 development in high risk HPV positive (hr-HPV+) women.

Methodology 59 women with normal cytology at initial screening and follow-up over six years were enrolled from ARTISTIC trial. The cohort included 14 hr-HPV negative (hr-HPV-) and 15 hr-HPV+ women through whole follow-up.