Methodology We performed targeted transcriptomics analysis of 50 genes using qPCR and estrogen metabolism analyses using LC-MS/MS. The model systems were high-grade serous ovarian cancer (HGSOC) cell lines OVSAHO, Kuramochi, COV632, and immortalized normal ovarian epithelial HIO-80 cells. The results in cell lines were compared with public transcriptome and proteome data for the HGSOC tissues.

Results In all model systems, HGSOC cell lines and tissues, high steroid sulfatase expression, and weak/undetected aromatase (*CYP19A1*) expression supported the formation of estrogens from the E1-S precursor. In ovarian cancer cells, the metabolism of E1-S to estradiol was the highest in OVSAHO, followed by Kuramochi and COV362 cells, and decreased with increasing chemoresistance. In addition, higher *HSD17B14* and *CYP1A2* expressions were observed in highly chemoresistant COV362 cells and platinum-resistant tissues compared to HIO-80 cells and platinum-sensitive tissues. The HGSOC cell models differed in *HSD17B10, CYP1B1*, and *NQO1* expression. Proteomic data also showed different levels of HSD17B10, CYP1B1, NQO1, and SULT1E1 between the four HGSOC subtypes: differentiated, immunoreactive, proliferative, and mesenchymal.

Conclusion The results of our study suggest that in HGSOCs, the metabolism of E1-S precursor into estrogens decreases with increasing chemoresistance and that HGSOC subtypes form different levels of estrogens and their metabolites. The estrogen-biosynthesis-associated targets identified in our research present a base for further studies leading to potential personalized treatment development.

2022-RA-1505-ESGO OVARIAN CANCER RETROSPECTIVE EUROPEAN (O'CARE) OBSERVATIONAL STUDY: ANALYSIS OF FIRST-LINE (1L) OUTCOMES IN PATIENTS WITH OVARIAN CANCER (OC) STRATIFIED BY NUMBER OF RISK FACTORS FOR PROGRESSION

[1]Jonathan Krell, [2]Thumulurua K Madhuri, [3]Danielle Shaw, [4]John McGrane, [5]Anjana Anand, [6]Andreas Hartkopf, [7]Ana Herrero, [8]Cheng Yeoh, [9]Maria Masvidal, [10]Jean-David Fumet, [11]Gunther Rogmans, [12]Francesco Raspagliesi, [13]Celine Gavoille, [14]Whitney York, [14]Jeanne M Schilder, [15]Barbara Mascialino, [15]Eleanor McDermott, [16]Linda Kalilani, [17]Lars Hanker. *[1]Imperial College London, London, UK; [2]Royal Surrey NHS Foundation Trust, Guildford, UK; [3]The Clatterbridge Cancer Centre NHS Foundation Trust, Liverpool, UK; [4]Royal Cornwall Hospitals NHS Trust, Cornwall, UK; [5]Nottingham University Hospitals NHS Trust, Nottingham, UK; [6]Universitätsklinikum Tübingen, Tübingen, Germany; [7]Hospital Universitario Miguel Servet, Zaragoza, Spain; [8]Queen Alexandra Hospital, Portsmouth, UK; [9]Hospital Universitari Sant Joan de Reus, Tarragona, Spain; [10]Centre Georges François Leclerc, Dijon, France; [11]Helios Klinik, Krefeld, Germany; [12]Fondazione IRCCS Istituto Nazionale dei Tumori, Milano, Italy; [13]Institut de Cancerologie de Lorraine (ICL) Nancy – Unicancer, Vandœuvre-lès-Nancy, France; [14]GSK, Philadelphia, PA; [15]GSK, Stevenage, UK; [16]GSK, Research Triangle, NC; [17]Arbeitsgemeinschaft Gynäkologische Onkologie (AGO), University Hospital Schleswig-Holstein, Campus Lübeck, Germany*

10.1136/ijgc-2022-ESGO.732

Introduction/Background The O'CaRE study assessed real-world burden of disease, treatment patterns, and outcomes in patients with OC in 5 European countries (UK, France, Germany, Spain, and Italy). The analysis presented provides real-world data on the cumulative impact of risk factors (RFs) on disease progression and survival following 1L treatment.

Methodology O'CaRE was a multicentre, noninterventional retrospective medical chart review study of patients aged ≥18 years diagnosed with epithelial ovarian, fallopian tube, or primary peritoneal cancer from 1 January 2014 to 31 December 2015. Patients were classified into moderate- or high-risk categories based on number of RFs for progression (Table). High-risk patients were further grouped by total number of RFs. Patients were followed from index date (date of diagnosis) until last activity or study end (maximum follow of 4 years). Kaplan-Meier methodology was used to estimate progression-free survival (PFS) and overall survival (OS).

Results The analysis included 412 patients: 7 (1.7%) had moderate risk of progression, whereas 405 (98%) had high risk of progression (table 1). For those with high risk, 84 (20.4%), 133 (32.3%), 139 (33.7%), and 49 (11.9%) had 1, 2, 3, and 4 RFs, respectively. Median PFS was 31.3 months for patients with 0 RFs and 12.6, 7.9, 5.9, and 3.5 months for patients with 1, 2, 3, or 4 RFs, respectively. Median OS was 41.9 months for patients with 0 RFs and not reached, 25.0, 18.0, and 7.4 months for patients with 1, 2, 3, or 4 RFs, respectively.

Abstract 2022-RA-1505-ESGO Table 1 Outcomes by risk factors

Risk Factor Classifications		
Moderate risk	**High risk**	
Stage III disease No VRD PDS *BRCA*m	Presence of ≥1 of the following: Stage IV disease VRD IDS or other surgery *BRCA*wt or *BRCA*unk	

	Moderate risk	High risk			
	0 Risk Factors	1 Risk Factor	2 Risk Factors	3 Risk Factors	4 Risk Factors
n (%)	7 (1.7)	84 (20.4)	133 (32.3)	139 (33.7)	49 (11.9)
mPFS (95% CI), months	31.3 (14.2–NR)	12.6 (10.8–19.6)	7.9 (6.3–11.1)	5.9 (4.5–8.2)	3.5 (1.9–5.0)
mOS (95% CI), months	41.9 (41.9–NR)	NR (34.4–NR)	25.0 (21.1–30.2)	18.0 (13.7–24.2)	7.4 (5.1–19.4)

*BRCA*m, *BRCA* mutant; *BRCA*unk, *BRCA* unknown; *BRCA*wt, *BRCA* wild-type; IDS, interval debulking surgery; mOS, median overall survival; mPFS, median progression-free survival; NR, not reached; PDS, primary debulking surgery; VRD, visible residual disease.

Conclusion This real-world analysis of patients with OC from 5 European countries demonstrated that higher numbers of RFs were associated with shorter median PFS and OS. This analysis provides real-world data relating to 1L treatment outcomes for patients with OC; if validated in clinical trials, the number of RFs could be a stratification factor for future 1L OC trials.

2022-RA-1515-ESGO IMPACT OF SURGICAL STAGING ON SURVIVAL OF LOW GRADE ENDOMETRIOID OVARIAN CANCER APPARENTLY CONFINED TO THE OVARY

[1]Nicolò Bizzarri, [2]Majdi Imterat, [3]Robert Fruscio, [4]Anna Myriam Perrone, [5]Rosanna Mancari, [2]Alexander Traut, [1]Andrea Rosati, [2]Andreas du Bois, [3]Debora Ferrari, [4]Pierandrea de Iaco, [1]Raffaella Ergasti, [2,6]Beyhan Ataseven, [3]Silvia Volontè, [4]Marco Tesei, [1]Maria Teresa Perri, [2,7]Florian Heitz, [2]Nicole Concin, [1]Francesco Fanfani, [5]Enrico Vizza, [1]Giovanni Scambia, [2]Philipp Harter, [1]Anna Fagotti. *[1]UOC Ginecologia Oncologica, Fondazione Policlinico Universitario A. Gemelli, IRCCS, Dipartimento per la Salute della Donna e del Bambino e della Salute Pubblica, Rome, Italy; [2]Department of Gynecology and Gynecologic Oncology, Ev. Kliniken Essen-Mitte, Essen, Germany; [3]Clinic of Obstetrics and Gynecology, University of Milan Bicocca, San Gerardo Hospital, Monza, Italy; [4]Division of Oncologic Gynecology, IRCCS Azienda Ospedaliero-Universitaria di Bologna, Bologna, Italy; [5]Gynecologic Oncology Unit, IRCCS – Regina Elena National Cancer Institute, Rome, Italy; [6]Department of Obstetrics and Gynecology, University Hospital, LMU Munich, Munich, Germany; [7]Department for Gynecology with the Center for Oncologic Surgery Charité Campus Virchow-Klinikum, Charité – Universitätsmedizin Berlin, corporate member of Freie Universität Berlin, Humboldt-Universität zu Berlin, and Berlin Institute of Health, Berlin, Germany*

10.1136/ijgc-2022-ESGO.733