patients. We sought to determine if participation in any clinical trial is associated with a survival benefit in patients with newly diagnosed advanced-stage ovarian cancer.

Methods We retrospectively investigated the patients who treated for newly diagnosed advanced-stage ovarian cancer at Yonsei Cancer Hospital between 2019 and 2021. This study included 202 patients with stage III-IV, 82 patients who participated in clinical trials and 120 participants receiving standard-of-care therapy (SOC).

Results The median follow-up duration was 31.5 months. Disease recurrence occurred in 123 (60.9%) patients and 45 (22.3%) patients died. Among the patients in both groups, there were no significant differences in age, histologic type, stage, median CA-125 level, comorbidities, and BRCA 1/2 status. There were also no differences in the incorporation of hyperthermic intraperitoneal chemotherapy, neoadjuvant chemotherapy, residual disease after cytoreductive surgery. The patients involved in clinical trials were associated with significantly improvement in progression-free survival (PFS) (31.4 vs. 19.2 months; HR, 0.67; 95% CI, 0.46 to 0.97; p = 0.034) compared to SOC. There was no difference in overall survival between two groups (P = 0.164).

Conclusion/Implications Clinical trial participation was associated with improved PFS in patients with newly diagnosed advanced-stage ovarian cancer. Clinical trial participation is considered to be beneficial to patients with newly diagnosed advanced-stage ovarian cancer.

EP274/#837 CHARACTERIZATION OF A THREE-DIMENSIONAL CULTURE SYSTEM REPRESENTATIVE OF DISEASE PROGRESSION IN HIGH-GRADE SEROUS OVARIAN CANCER

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Introduction PEO1, PEO4 and PEO6 are cell lines derived from a single patient with high-grade serous ovarian cancer, the most common disease subtype, which illustrate disease progression. In cell culture-treated flat-bottom flasks, PEO1 and PEO4 form two-dimensional cellular aggregates and PEO6 form three-dimensional structures. This project aims to determine if differences in morphology, viability, proliferation, and metabolic activity exist between the three cell lines when grown in an ultra-low attachment plate more representative of in-vivo conditions.

Methods PEO1, PEO4 and PEO6 cells were grown in ultra-low attachment plates. Live/dead cell imaging, apoptosis and proliferation detection as well as ATP quantitation assays were performed using microscope imaging, cytometry and spectrophotometry methods.

Results The cell lines were morphologically different, mimicked the multilayered structure of in-vivo tumors and had a similar proliferation pattern. PEO1 displayed the highest aggregation level, PEO6 the highest compaction level, and PEO4 the lowest aggregation and compaction levels. All three cell lines were found to mimic poorly vascularized tumors by forming a multilayered structure with an outer layer of live cells and an inner core of apoptotic cells, but at different times. It was observed that PEO1, PEO4 and PEO6 cells proliferate mostly in the cell masses' periphery. PEO6 cells produced a higher amount of ATP followed by PEO4 and then PEO1 cells after 4 and 7 days.

Conclusion/Implications Three-dimensional cell culture of established ovarian cancer cell lines in such environment likely will serve as a preclinical model of disease to provide experimental responses to therapeutic agents.

EP276/#634 THE IMPACT OF HISTOLOGIC SUBTYPES ON SURVIVAL OUTCOMES IN PRIMARY MUCINOUS OVARIAN CANCER

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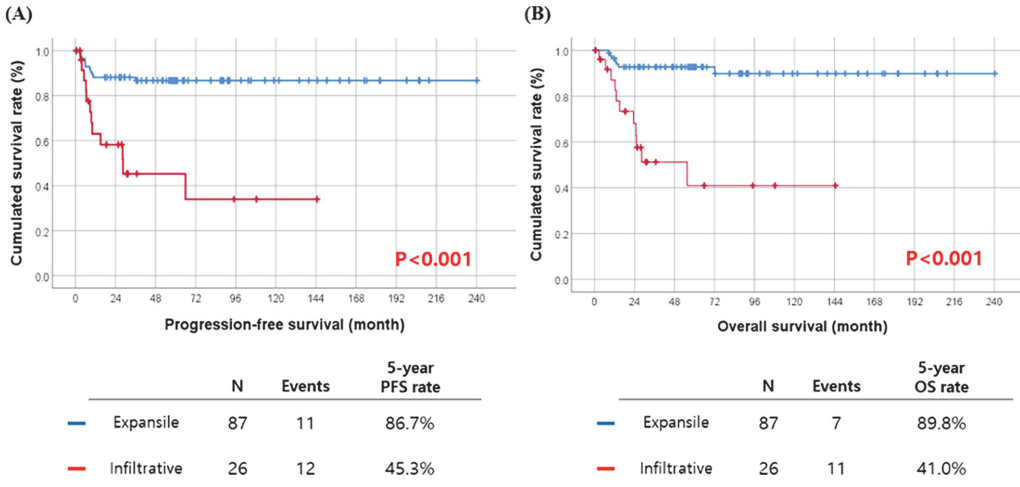
Introduction Primary mucinous ovarian cancer (PMOC) is a unique and rare subtype of ovarian cancer. In 2014, the World Health Organization introduced a new histologic classification by dividing PMOC into two subtypes: expansile or



Abstract EP276/#634 Figure 1 (A) PFS (B) OS according to histologic classification among all patients

Abstract EP276/#634 Table 1 Univariate and multivariate analysis of PFS and OS in the study population

Variables	PFS				OS			
	Univariate		Multivariate		Univariate		Multivariate	
	HR [95% CI]	*P*-value	HR [95% CI]	*P*-value	HR [95% CI]	*P*-value	HR [95% CI]	*P*-value
Age								
>50 vs. ≤50	1.494 [0.698−3.195]	0.301	-	-	2.185 [0.871−5.481]	0.096	-	-
Stage								
≥II vs. I	24.548 [10.139−59.436]	<0.001	6.506 [1.681−25.178]	0.007	58.835 [13.551−255.441]	<0.001	16.762 [2.455−114.427]	0.004
Initial serum CA-125								
>40 IU/mL vs. ≤40 IU/mL	3.192 [1.391−7.326]	0.006	1.847 [0.673−5.073]	0.234	3.969 [1.435−10.975]	0.008	1.080 [0.342−3.415]	0.895
Residual tumor after surgery								
Present vs. absent	29.536 [12.429−70.188]	<0.001	4.892 [1.386−17.271]	0.014	37.691 [13.968−101.704]	<0.001	5.982 [1.246−28.716]	0.025
Incomplete staging operation								
Yes vs. no	1.532 [0.688−3.411]	0.296	-	-	1.168 [0.477−2.858]	0.735	-	-
Histologic subtype								
Infiltrative vs. Expansile	5.452 [2.382−12.479]	<0.001	1.586 [0.609−4.126]	0.345	7.878 [3.017−20.573]	<0.001	1.377 [0.427−4.437]	0.592

Abbreviation: PFS, progression free survival; OS, overall survival; HR, hazard ratio; CI, confidence interval; CA, cancer antigen

infiltrative. In this study, we investigated the clinical implications of their histological subtypes on survival outcomes.

Methods We identified patients with PMOC who had undergone primary surgery between 2003 and 2021. Patients with other types of ovarian cancer or severe comorbidities were excluded. We collected patients' baseline characteristics, surgical details, and pathological information. Progression-free survival and overall survival were calculated, while prognostic factors were also investigated.

Results We included 131 patients in total. The median age was 50 years, and 103(78.6%) patients had stage I disease. During 55.9 months of median follow-up, there were 27 recurrences and 20 deaths. Among them, 113 patients were classified into 87(77%) expansile and 26(23%) infiltrative subtypes after the slide review. Advanced stage, lymph node involvement, and residual tumors after surgery were more common in the infiltrative subtype. The infiltrative group showed worse 5-year progression-free and overall survival rates (figure 1). In multivariate analyses, advanced stage and residual tumor after surgery were associated with worse prognosis, while the infiltrative subtype showed no statistical significance (table 1). In the subgroup analysis of stage I disease, there was no difference in survival between the two groups.

Conclusion/Implications In PMOC, the infiltrative histological subtype showed worse prognosis than the expansile subtype, with a higher proportion of advanced-stage tumors. However, it remains uncertain whether infiltrative subtype is an independent prognostic factor.

EP277/#794 STING INHIBITORS REVERSE PLATINUM RESISTANCE IN OVARIAN CANCER BY INHIBITING THE CGAS-STING PATHWAY IN CANCER-ASSOCIATED FIBROBLASTS

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Introduction Chemoresistance is a key factor limiting the cure rate of ovarian cancer. Cancer-associated fibroblasts (CAFs) have been shown to be actively involved in cancer progression and chemoresistance. Intratumoral inflammatory environments affect many therapeutic responses, but the underlying mechanisms by which CAF participates in chemoresistance by modulating the tumor's inflammatory environment are largely unknown.

Methods We cultured primary ovarian cancer-associated fibroblasts, using indirect co-culture to study how ovarian cancer cells activate CAF to participate in ovarian cancer platinum-resistant. WB shows activation of the cGAS-STING pathway in CAF, and IHC shows differential expression of STING in cisplatin-sensitive/resistant patients. Cell viability or apoptosis using CCK8 or apoptosis kits, respectively. The STING antagonist H-151 and the STING agonist MSA-2 investigated the contribution of the cGAS-STING pathway to the chemoresistance of ovarian cancer.

Results After treatment with cisplatin for ovarian cancer cells, the supernatant was indirectly co-cultured with CAFs, and mRNA sequencing showed that the cGAS-STING-IFNB1 pathway was activated in CAFs. We found that IFNB1 can promote platinum resistance in cancer cells by inhibiting cisplatin-induced DNA damage and promoting HRR. Anti-IFNB1 restores platinum sensitivity. In addition, the expression of STING in the tumor stroma is associated with poor prognosis in patients. In vivo experiments showed that inhibition of STING expression could restore platinum sensitivity, while activating STING could not significantly enhance immune killing ability.

Conclusion/Implications Our study shows that activation of the cGAS-STING pathway in CAFs is involved in platinum resistance in ovarian cancer, and STING inhibitors are able to restore ovarian cancer chemotherapy sensitivity.

EP279/#598 OSBPL10-APOE PATHWAY INCREASES RESISTANCE OF PLATINUM AND PARPI BY REGULATING CHOLESTEROL METABOLISM AND DNA DAMAGE REPAIR IN PDX MODEL OF HRD-POSITIVE HGSOC

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