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.

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**Abstract EP276/#634 Table 1**

<table>
<thead>
<tr>
<th>Variables</th>
<th>PFS</th>
<th>OS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Univariate HR [95% CI] P-value</td>
<td>Univariate HR [95% CI] P-value</td>
</tr>
<tr>
<td>Age &gt;50 vs. ≤50</td>
<td>1.494 [0.699–3.195] 0.301</td>
<td>2.185 [0.871–5.481] 0.096</td>
</tr>
<tr>
<td>Stage ≥ II vs. I</td>
<td>24.548 [10.139–59.436] &lt;0.001</td>
<td>58.835 [13.551–255.441] &lt;0.001</td>
</tr>
<tr>
<td>Residual tumor after surgery</td>
<td>29.536 [12.420–70.188] &lt;0.001</td>
<td>37.691 [13.968–101.704] &lt;0.001</td>
</tr>
<tr>
<td>Incomplete staging operation</td>
<td>1.532 [0.668–3.411] 0.576</td>
<td>1.516 [0.477–2.858] 0.715</td>
</tr>
<tr>
<td>Histologic subtype</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infiltrative vs. Expansile</td>
<td>5.452 [2.382–12.479] &lt;0.001</td>
<td>7.878 [2.017–28.573] &lt;0.001</td>
</tr>
</tbody>
</table>

Abbreviations: PFS, progression free survival; OS, overall survival; HR, hazard ratio; CI, confidence interval; CA, cancer subtype.

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**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 CCK-8 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.

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**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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10.1136/ijgc-2023-IGCS.348
Introduction  Compared with HRD-negative ovarian cancer patients, HRD-positive patients are more sensitive to platinum-based chemotherapy and benefit from PARPi is more significant. However, there are still some HRD-positive patients with platinum and PARPi resistance, resulting in a poor prognosis.

Methods  In this study, a key differential gene, Oysterel binding protein like 10 (OSBPL10), was identified by bioinformatics analysis of platinum-resistant and platinum-sensitive HRD-positive ovarian cancer patients after first-line chemotherapy in TCGA database. Western Blot and Immunohistochemistry were performed on tumor tissues from Qilu Hospital of Shandong university to verify the differential expression of OSBPL10. Target genes of OSBPL10 were identified by RNA-seq and validated by RIP. In addition, we established PDX models for high-grade serous ovarian cancer (HGSOC) patients to validate the efficacy of targeting OSBPL10.

Results  The expression of OSBPL10 in platinum-resistant HRD-positive ovarian cancer patients were significantly higher than that in platinum-sensitive patients. Inhibiting OSBPL10 enhanced the sensitivity to cisplatin and Niraparib of ovarian cancer cells. Apolipoprotein E (APOE), as a target gene of OSBPL10, was involved in lipid transport and lipoprotein metabolism. Overexpression of OSBPL10 could active the expression of APOE, enhance DNA damage repair function, and up-regulate cholesterol levels in intracellular, extracellular and tumor microenvironment, leading to increased exhaustion of CD8+ T cells, and further promoting resistance to cisplatin and Niraparib. Combined with Niraparib, OSBPL10 adenovirus (shRNA-OSBPL10) was more effective in repressing tumor growth of PDX models than Niraparib monotherapy.

Conclusion/Implications  OSBPL10-APOE pathway regulated DNA damage repair and cholesterol metabolism, leading to cisplatin and Niraparib resistance of HRD-positive HGSOC.

EP282/#263  ONCOLOGIC OUTCOME OF PRIMARY TREATMENT IN PATIENTS DIAGNOSED WITH EPITHELIAL OVARIAN/TUBAL/PERITONEAL CARCINOMA WHOM UNDERWENT SUBOPTIMAL SURGERY

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Introduction  To evaluate the response rate of primary treatment, its predicting factors, and to analyze survival outcomes in patients with epithelial ovarian/tubal/peritoneal carcinoma whom underwent suboptimal surgery.

Methods  This study included women whom received suboptimal surgery between May 2006 and December 2020. The data of patient’s clinical information, histopathology, tumor stage, surgical methods and outcomes, adjuvant treatment, and primary treatment outcomes were collected. Follow-up data was documented until 31 March 2023. The oncologic outcomes were analyzed.

Results  Total of 320 study patients, overall response rate was 58.1%. The median progression free survival (PFS) duration was 13.167 months [6.675–20.583], and the median overall survival (OS) was 32.850 months [15.008–53.642]. The factors significantly associated with response were received neo-adjuvant chemotherapy (NAC) with adjusted odd ratio (aOR) 3.342 (95% CI 1.619–6.900, P=0.001), and high-grade serous carcinoma (HGSC) (aOR 0.153, 95% CI 0.092–0.255, P<0.001). HGSC associated with longer median PFS (15.9 months vs 7.2 months, P<0.001) and median OS (35.221 months vs 11.994 months, P<0.001) compared to non-HGSC.

Conclusion/Implications  The oncologic outcomes of study patients were comparable to the landmark trials. HGSC has higher response rate, longer PFS and OS than non-HGSC.