group, whereas 131 proteins were in the poor response group. Proteins significantly upregulated in the good response group included ribosomal- and infection-related proteins. Proteins significantly upregulated in the poor response group included extracellular matrix receptor- and coagulation-related proteins. To identify a protein signature that stratifies good and poor responders to PARP inhibitors, we performed four feature selection algorithms with leave-one-out cross-validation to improve the accuracy. High expression of Proteins A and B were associated with worse and better progression-free survival, respectively.

Conclusions We successfully identified protein signatures associated with response to PARP inhibitors. This study was the most extensive proteomic analysis to predict PARP inhibitor response in ovarian cancer.

Conclusions Our study results demonstrate the survival benefits of BEV and secondary CRS in patients with platinum-sensitive relapsed OCCC.

Objectives About 70% of ovarian cancer patients experience recurrence, and resistance is induced by repeated chemotherapy. So, research for novel therapeutic approach is urgently needed. FK506-binding protein like (FKBPL) is involved in immune & inflammatory responses, and signaling pathways regulating various cancers. However, the role of FKBPL in epithelial ovarian cancer (EOC) has not been elucidated.

Methods Immunohistochemical analysis of FKBPL expression using tissue microarray was performed on 398 epithelial ovarian tissues (186 cancer, 49 borderline, 84 benign, and 79 normal tissues). The clinico-pathological parameters and those data were compared. It was also performed in vitro to investigate the functional role of FKBPL in ovarian cancer cell lines.

Results The expression of FKBPL in ovarian cancer tissue was upregulated than other epithelial tissues (all p < 0.001). Importantly, FKBPL expression was associated with stage, tumor grade, cell type, and chemotherapy response (p ≤ 0.05). Multivariate survival analysis showed that overexpression of FKBPL was associated with poor overall survival (HR = 3.58; 95% CI: 1.87–6.84, p < 0.001) and disease-free survival (HR = 3.1; 95% CI: 1.97–4.87, p < 0.001). In-vitro results also showed that knockdown of FKBPL was associated with decreased cell proliferation, inhibited colony formation, and induction of G1 phase cell cycle arrest, supporting an oncogenic role of FKBPL in ovarian cancer cell lines.

Conclusions Overexpression of FKBPL could be a significant biomarker for predicting poor survival after chemotherapy. In addition, future research that reveals the mechanism of FKBPL on the cancer cell cycle will lead to the development of new anticancer drugs.