malignant germ cell tumors have no complete intra peritoneal surgical staging.

**EPV195/#37**
**VEGF-A INHIBITOR INDUCED TUMOR-ASSOCIATED MACROPHAGE REPROGRAMMING AND PD-L1 OVEREXPRESSION VIA A DUAL ROLE OF IFN-γ IN OVARIAN CANCER**

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**Objectives** The beneficial effects of vascular endothelial growth factor A (VEGF-A) inhibitor are only observed in a subset of patients with advanced ovarian cancer. The lack of response of VEGF-A inhibitors is thought to be related with the immuno-suppressive microenvironment; however, this is still controversial. A better understanding of the underlying mechanism of VEGF-A inhibitor (Bevacizumab)-mediated the immune escape could benefit the development of therapeutic regimens for patients with ovarian cancer.

**Methods** The polarization of tumor-associated macrophages (TAMs), IFN-γ secretion of macrophages and ovarian cancer cells, PD-L1 expression in ovarian cancer cells, and phagocytic function of macrophages after Bevacizumab intervention were examined. In addition, the efficacy of combined Bevacizumab with anti-PD-1 antibody (aPD-1) was evaluated in a murine ovarian cancer model.

**Results** We first identified that Bevacizumab stimulated IFN-γ secretion from macrophages and ovarian cancer cells. Moreover, we demonstrated that Bevacizumab upregulated PD-L1 expression in an IFN-γ-P13K-NF-kB-dependent manner in ovarian cancer. Interestingly, although Bevacizumab elicited antitumor immunity by inducing macrophage polarization to an M1 phenotype via elevated IFN-γ secretion, the phagocytic function of macrophages was suppressed by upregulated PD-1 expression in macrophages. Furthermore, Bevacizumab combined with an aPD-1 significantly decreased the tumor burden and prolonged survival time in mice with ovarian cancer.

**Conclusions** Here, we demonstrated a dual role of IFN-γ induced by Bevacizumab in ovarian cancer. Specifically, IFN-γ promoted immune activation in terms of M1 polarization and immune suppression through PD-L1/PD-1 upregulation. The combination of Bevacizumab and aPD-1 improved the local immune status and provided a promising novel therapeutic regimen against ovarian cancer.

**EPV197/#394**
**BCARE- FUNCTIONALLY ASSESSING TREATMENT RESPONSE IN OVARIAN CANCER PATIENTS**

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**Objectives** Treatment regimens in oncology frequently become personalized and multimodal. Therefore, reliable biomarkers to predict drug responses are needed. Here, we establish a functional assay reflecting response to DNA damage-based treatment approaches.

**Methods** Our Bimodal prediction of ovarian CAncer treatment REsponse (BCARE) score combines irradiation-induced DNA damage with functional assessment of homologous recombination (HR) DNA repair visualized by immunofluorescence detection of γH2AX and cyclinA2/RAD51 positive cells. BCARE is quantified by automated image analysis using Image J and R/Bioconductor. Treatment response of cancer cells to PARP inhibition, radiotherapy, and chemotherapy is analyzed by colony formation and MTT cell viability assays.

**Results** BCARE reflects the percentage of RAD51/cyclinA2 double positive cells. It significantly correlated with response to various drugs tested in 6 ovarian cancer cell lines: olaparib (R=0.92, p=0.0095), radiotherapy (R=0.78, p=0.0374), cisplatin (R=0.85, p=0.0325), doxorubicin (R=0.92, p=0.0095), and carboplatin (R=0.77, p=0.0738). Additionally, we show that the BCARE coincides with the