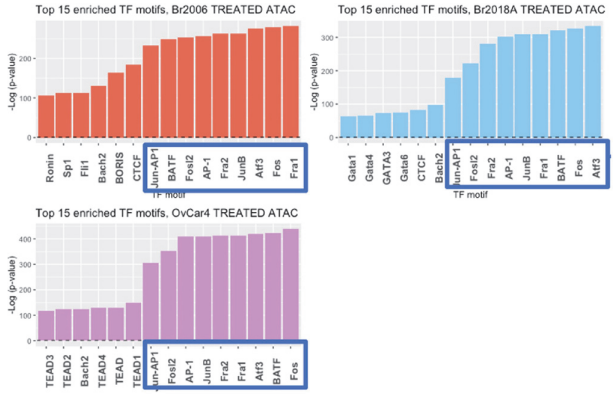
samples included in the experiment, with a subsequent increased transcription of the related genes.



Abstract #597 Figure 1 Top 15 enriched transcription factor motifs across different primary cell cultures (Br2006 and Br018A) and established cell lines (OvCar4)

Conclusion Platinum resistance may occur after multiple carboplatin pulses; epigenetic changes certainly represent a field which deserves to be explored. Although AP1-TF has already been investigated as a therapeutic target in other malignancies, it has never been explored in ovarian cancer. Further comparisons will be certainly needed in order to increase the statistical power.

Disclosures none

#674 EXPLORING THE IMPACT OF ANDROGEN EXPOSURE ON THE TRANSCRIPTIONAL PROFILE OF ANDROGEN RECEPTOR OVEREXPRESSING ENDOMETRIAL CANCER CELLS THROUGH RNA-SEQUENCING

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Introduction/Background Endometrial cancer (EC) is the fourth most common cancer in women in the UK. While oestrogen and progesterone have been extensively researched, the role of androgen in EC has been relatively under-investigated. We aim to study the role of androgen in EC through the development of EC cell lines that overexpress androgen receptor (AR) with varying expression levels and investigate the consequences of AR overexpression on EC cell biology.

Methodology We transduced Ishikawa EC cell line with doxycycline-inducible ectopic AR, or constitutionally active ectopic AR in varying doses through lentivirus transduction resulting in varying levels of AR expression. This was followed by antibiotic selection and validation experiments to confirm ectopic AR expression. RNA was isolated from both cell models and RNA-sequencing was performed with and without androgen exposure to investigate the impact on the EC cells' transcriptional profile.

Results Dose-dependent AR overexpression was observed using western plot, immunofluorescence, and qPCR, validating the expected post-transduction ectopic AR expression levels. RNA-seq analysis of the AR-transduced EC cells identified various differentially expressed genes in response to androgen exposure including CDHR1, KCNF1, COL8A1, and TIPARP. This effect on the transcriptional profile of EC cells was shown to be more pronounced with higher expression levels of AR. The differentially expressed genes were enriched for pathways associated with KRAS-signalling (nominal P-value=0.008), epithelial mesenchymal transition (nominal P-value<0.001), and apoptosis (nominal P-value=0.03) supporting the role of AR in EC pathogenesis.

Conclusion We successfully established AR-overexpressing EC cell lines which will serve as valuable models in investigating the AR role in EC. Our RNA-seq transcriptional profiling revealed pathways involved in tumorigenesis affected by AR expression highlighting the potential implications of AR signalling in EC. Future work will be performed to functionally validate the AR role in EC and ChIP-seq experiments will be conducted to identify direct AR target sites.

Disclosures -

#836 LIQUID BIOPSY ISOLATION OF CIRCULATING TUMOUR CELLS FROM EPITHELIAL OVARIAN CANCER PATIENTS AND THEIR PROGNOSTIC SIGNIFICANCE

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Introduction/Background Cancer cells that transit from primary tumours into the blood circulatory system are known as circulating tumour cells (CTCs). Research has highlighted the difficulties with detection of CTCs from ovarian cancer patients using EpCAM-based techniques, with classical EpCAM-CTC enumeration alone having limited prognostic significance. This study aims to isolate epithelial ovarian CTCs from patients using ANGLE-Parsortix technology.

Methodology Peripheral blood specimens [n=106] were prospectively collected from 54 newly diagnosed epithelial ovarian cancer patients since November 2020. Samples were taken pre and post-neoadjuvant chemotherapy, pre-surgery and during cytoreduction surgery from the central ovarian vein. Longitudinal sampling is ongoing. CTCs were isolated using Parsortix microfluidic device and immunophenotyped (CTC-ID; DAPI, CD45, CK7/panCK/EpCAM] by immunofluorescence and confocal microscopy.

Results 66% of patients recruited had at least 1 CTC detected [CTC range of 1–22 cells per 7.5 ml of blood]. CTCs were present in 74% of ovarian vein [n=19] samples [CTC range of 1–2475 cells]. Patients with ≥2 CTCs had higher CA125 levels. Median PFS was significantly lower at 13.5 months in patients with ≥2 CTCs compared to 21 months with <2 CTCs. No statistical difference was seen between pre and post neoadjuvant CTC counts, however, a number of poor chemotherapy responders had persistent CTC levels following treatment. CTC clusters were significantly isolated from the ovarian vein. Follow up of treatment response, PFS, overall survival and 1 year blood-sampling follow up is currently