Introduction Circulating tumor cells (CTCs) have received enormous attention as a novel biomarker in various malignant diseases. The aim of this study was to evaluate CTC detection using tapered-slit membrane filter based chipsets in differential diagnosis of ovarian tumors.

Methods A total of 230 preoperative women with an indeterminate ovarian tumor were prospectively enrolled. Seven patients diagnosed with other primary origin and 20 patients sampled after neoadjuvant chemotherapy were excluded from the analysis. Sensitivity, specificity, accuracy, and area under the receiver operating characteristic curve (AUC-ROC) of CTC detecting chipsets were analyzed according to postoperative pathologic results respectively.

Results 81 (39.9%) benign tumors, 32 (15.8%) borderline tumors, and 90 (44.3%) ovarian cancers were pathologically confirmed. CTC detecting chipsets had sensitivity, specificity, accuracy, and AUC-ROC of 75.6%, 58.0%, 67.3%, and 0.655 (95% confidence interval [CI], 0.570–0.740) for differentiating ovarian cancer from benign ovarian tumor. Sensitivity, specificity, accuracy, and AUC-ROC for detecting ovarian cancer from borderline tumor were 75.6%, 50.0%, 68.9%, and 0.622 (0.505–0.739), respectively. In addition, sensitivity, specificity, accuracy, and AUC-ROC were 68.9%, 58.0%, 64.5%, and 0.622 (0.540–0.703) for differentiating borderline and malignant ovarian tumor from benign tumor. Sensitivity, specificity, accuracy, and AUC-ROC for detecting ovarian cancer from benign to borderline tumor were 75.6%, 55.8%, 64.5%, and 0.645 (0.567–0.723), respectively.

Conclusion/Implications Our study suggests that preoperative high-throughput viable CTC isolation using tapered-slit membrane filter based chipsets could have a potential role in differentiating ovarian malignancy from benign and/or borderline tumors.

EP007/#793
THE MECHANISM THAT AFFECTS CELL DEATHS FOR TUMOR SUPPRESSION GENE-PTEN BY EZH2 ACTIVITY IN A CERVICAL CANCER CELL LINE (HELA-R) WITH RADIATION-TREATMENT RESISTANCE

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Introduction To determine the mechanism affecting cell deaths for tumor suppression gene-PTEN by targeting EZH2 activity as a therapeutic strategy against EGFR inhibitor resistance in the EGFR-Mutant cervical cancer cell line with radiation-treatment resistance

Methods We investigated the mechanism affecting cell deaths for tumor suppression gene-PTEN by targeting EZH2 activity as a therapeutic strategy against EGFR inhibitor resistance in the EGFR-Mutant cervical cancer cell line with radiation-treatment resistance. Epidermal growth factor receptor (EGFR) expression is expressed in various types of tumors, including cervical cancer, in an increased state than normal tissue.

Results EGFR was expressed in the human cervical cancer cell line (Hela cell line) and cervical cancer radiation-resistant cell line (Hela-R cell line), and it was observed that the Hela cell line disappeared by drug-reacting EGFR inhibitor (Apatinib), but the Hela-R cell line did not die. Therefore, it can be inferred that Hela-R cell lines generally have pathways other than EGFR-related signaling cascades (e.g., PI3K/Akt, STAT, and MAPK), which have become interesting in EGFR/PI3K/PTEN/AKT signaling pathways, which serve as PETN tumor suppressors. The role of the PTEN gene in the carcinogenesis of cervical cancer is not well known. PI3 generation activated by activation of PI3K is converted from PI3K to PI3K/PTEN/AKT signaling pathways, which serve as PETN tumor suppressors. Therefore, the Inhibitor of EZH2 ultimately provides clues about the new chemotherapy’s role in palliative cervical cancer patients with radiation resistance.

Conclusion/Implications We identified DEGs and several pathways enriched in cisplatin-resistant EC cells as potential therapeutic targets of cisplatin resistance, which need further validation.

EP008/#896
TRANSCRIPTOME ANALYSIS OF HERV-K ENV KNOCKOUT OVARIAN CANCER CELL LINES

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Abstracts

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