variables were analyzed. Also the functional studies with cervical cancer cell lines were evaluated.
Results The expression of E-cadherin was significantly reduced from normal to cancer, and associated with FIGO stage. Furthermore, E-cadherin expression was negatively correlated with pERK expression in cervical cancers. The Keplan-Meier plots demonstrated that E-cadherin lowexpression was associated with poor DFS and OS, and pERK, overexpression was significantly associated with poor DFS and OS. The DFS and OS with expression of low E-cadherin/high pERK was compared with patients with others which revealed a significant difference in DFS and OS. The Cox proportional hazards model revealed that a low E-cadherin/high pERK expression was an independent prognostic factor with respect to overall survival (HR=8.48 [95% CI, 3.36 – 21.37, p<0.01]. In cervical cancer cell lines, the knock down of E-cadherins promoted proliferation in cervical cancer cells and loss of E-cadherin led to EGFR mobility, which may stimulate EGFR dimerization and further boost its activation.
Conclusion/Implications Low expression of E-cadherin or combined with pERK is an indicator of poor prognosis in cervical cancer, suggesting their potential utility as prognostic test in clinical assessment.

EP002/#234 RESULTS OF CLINICAL RESEARCH (PRUM-IBIO STUDY): ESTABLISHMENT OF PROGNOSTIC BIOMARKERS FOR UTERINE MESENCHYMAL TUMORS

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Introduction Benign uterine leiomyoma (U.LMA) and malignant uterine leiomyosarcoma (U.LMS), which are both uterine mesenchymal tumors, are distinguished by the number of cells with mitotic activity. However, uterine mesenchymal tumors contain tumor cells with various cell morphologies; therefore, making a diagnosis, including differentiation between benign tumors and malignant tumors, is difficult. For example, cotyledonary dissecting leiomyoma (CDL) or uterine smooth muscle tumors of uncertain malignant potential (STUMPs), etc. are a group of uterine mesenchymal tumors for which performing a differential diagnosis is challenging. A standardized classification system for uterine mesenchymal tumors has not yet been established. Furthermore, definitive preoperative imaging techniques or hematological examinations for the potential inclusion of CDL or STUMP in the differential diagnosis have not been defined.
Methods We have been carrying out multicenter clinical research to establish life prognostic markers for uterine mesenchymal tumors.
Results Our clinical research showed that there is correlation between biomarker expression and mitotic rate or tumor recurrence. The candidate factors of immunohistochemical biomarkers can effectively help determine the malignant potential of CDL or STUMPs in patients who wish to become pregnant in the future.
Conclusion/Implications The establishment of gene expression profiles or detection of pathogenic variants by employing next-generation molecular techniques can aid in disease prediction, diagnosis, treatment, and prognosis. Here, we describe the problems in diagnosing uterine mesenchymal tumors along with the results of the latest our clinical research.

EP003/#550 EFFICACY OF COMBINATION CHEMOTHERAPY AND THIRD-GENERATION ONCOLYTIC HERPES VIRUS THERAPY FOR CERVICAL CANCER

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Introduction Radiotherapy and chemotherapy are the most common treatments for advanced or recurrent cervical cancer, but the prognosis is poor. We have investigated viral therapy with triple mutant herpes simplex virus (T-01) in a mouse model of cervical cancer as a novel therapeutic approach. However, its antitumor effect is not sufficiently effective. In this study, we examined whether the combination of viral therapy and anticancer agents could enhance the antitumor effect.
Methods A mouse-derived immortalized cell line TC-1 with HPV16 E6/E7 as tumor antigen was inoculated subcutaneously into the back of C57BL/6 mice to generate cervical cancer model mice. cisplatin (CDDP), etoposide (ETP), fluorouracil (5-FU) from in vitro results, irinotecan (CPT-11), and cyclophosphamide (CPA) were administered to model mice, and anticancer agents that showed antitumor effects were combined with T-01. The combination therapy was evaluated as enhancing the antitumor effect when it exceeded the antitumor effect of viral therapy and chemotherapy.
Results The CPT-11, ETP, and 5-FU combination with T-01 groups showed no enhancement of antitumor effect. In contrast, the CDDP and CPA combination with T-01 group showed significant tumor growth inhibition compared to the chemotherapy and T-01 groups. Although there was no significant difference in survival rates between the chemotherapy and T-01 groups, no mice died during the observation period in either treatment group.
Conclusion/Implications The combination of CDDP or CPA and viral therapy exceeded the tumor growth inhibitory effect of viral therapy and chemotherapy, and we concluded that the combination of the two therapies enhanced the anti-tumor effect.

EP005/#1379 HIGH-THROUGHPUT VIABLE CIRCULATING TUMOR CELL ISOLATION USING TAPERED-SLIT MEMBRANE FILTER BASED CHIPSETS IN DIFFERENTIAL DIAGNOSIS OF OVARIAN TUMORS

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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%, 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.

EP006/#881 DIFFERENTIAL GENE EXPRESSIONS IN ENDOMETRIAL CANCER CELLS WITH ACQUIRED CISPLATIN RESISTANCE

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Introduction To identify differentially expressed genes (DEGs) and signaling pathways in cisplatin-resistant endometrial cancer (EC) cells.
Methods Cisplatin-resistant endometrial cancer cells were established through continuous treatment of endometrial cancer cells (RL95–2 and Ishikawa) with gradually escalating doses of cisplatin. RNA-seq was performed on both original and cisplatin-resistant EC cells to evaluate DEGs. Gene-set enrichment analysis (GSEA) was also performed to find biologic processes or pathways in relation to cisplatin resistance.
Results Common hallmark gene sets enriched in both cisplatin-resistant RL95–2 and Ishikawa include inflammatory response, epithelial-mesenchymal transition, and KRAS signaling_up. Common DEGs include EMP3, CD70, and SERPINE1 in the inflammatory response gene set; LAMA2, BDNF, OXTR, CDH2, VIM, MATN3, ABI3BP, EDIL3, EMP3, CXCL1, SERPINE1, and IL32 in the epithelial-mesenchymal transition gene set; PTPRR, MMD, and KCNN4 in the KRAS signaling_up gene set.
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.

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 HelLa-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. PIP3 generated by activation of PI3K is converted from PIP3 to PIP2 again by PTEN and SHIP, thus inhibiting the signaling pathway activated by PIP3.
Conclusion/Implications In this study, PTEN and EZH2 can be observed in cervical cancer tissues with radiation resistance. Therefore, the Inhibitor of EZH2 ultimately provides clues about the new chemotherapy's role in palliative cervical cancer patients with radiation resistance.

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

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