

of CRC in the two groups. Chi-squared testing was used to assess for differences in the proportion of CRC between MMR groups with $p < 0.05$ considered significant.

Results Among 988 patients with EC not associated with a germline MMR mutation, 16% (n=162) had MLH-1 promoter hypermethylation and 84% (n=826) did not. Among those with MLH-1 promoter hypermethylation there were 6 cases (3.6%) of CRC vs. 34 cases (4.1%) in those with MSS disease ($p=.743$).

Conclusions We found no difference in incidence of CRC in individuals with MLH-1 promoter hypermethylated EC as compared with those with MSS disease. Patients with MLH-1 promoter hypermethylated EC should follow general CRC screening guidelines.

EPV096/#110

LSR ACTIVATES MAPK PATHWAY AND PROMOTES CELL PROLIFERATION AND INVASION IN ENDOMETRIAL CANCER: ANALYSIS OF BIOINFORMATICS-BASED SIGNAL TRANSDUCTION

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Objectives Lipolysis-stimulated lipoprotein receptor (LSR) is a membrane protein that has been studied in various malignant tumors. We previously reported that high expression of LSR was associated with poor prognosis, advanced stage, deep myometrial invasion, and metastasis in endometrial cancer (EC). However, the mechanism by which LSR affects patient's prognosis remains largely unclear. Here, we aimed to investigate the functions of LSR in EC.

Methods Cell proliferation and invasion were analyzed using LSR-knockdown cell lines (HEC1 and HEC116), and the activity of several signaling pathways were examined by Western blotting. To investigate the function of LSR in EC cells, the pathway enrichment and ontology analysis were performed using the publicly available proteomic data.

Results LSR-knockdown significantly suppressed cell proliferation in WST-8 assay. The pathway analysis demonstrated that MAPK signaling pathway was enriched in proteins correlated with high LSR expression. In ontology analysis, we found several biological processes, including 'regulation of ERK1/2' and 'MAPK cascade.' Following the results of pathway enrichment and ontology analysis, we confirmed that LSR-knockdown downregulated the phosphorylation of MEK/ERK pathway, including MEK1/2, ERK1/2, and p90RSK in western blotting. Cell invasion assay and western blot analysis demonstrated that LSR-knockdown suppressed MT1-MMP/MMP2 expression and cell invasion. Interestingly, ERK1/2-knockdown also suppressed MT1-MMP/MMP2 expression, suggesting that LSR activated MT1-MMP/MMP2 via ERK1/2 and promoted cell invasion.

Conclusions Our results of in vitro study and bioinformatic analysis showed that LSR regulated cell proliferation and invasion via MEK/ERK pathway, and contributed poor prognosis in EC. LSR may be a new therapeutic target of advanced EC.

EPV097/#140

APPLICATION OF A MACHINE LEARNING ALGORITHM TO IDENTIFY PREDICTORS OF RECURRENCE AND RECURRENCE FREE SURVIVAL IN HIGH GRADE ENDOMETRIAL CANCER

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Objectives To train various machine learning algorithms to predict recurrence and recurrence-free survival (RFS) in high-grade endometrial cancer (HGEC)

Methods Data was retrospectively collected across 8 Canadian centers including 1237 patients and divided arbitrarily 50% training, 25% validation and 25% testing. Four models were trained to predict recurrence: random forests, boosted trees, and 2 neural networks. Receiver operating characteristic curves (ROC) were used to determine model performance and select the best model based on highest area under the curve (AUC) in the test set. For time to recurrence models, we trained a random forest and Lasso model compared to Cox Proportional hazards. Concordance was reported using a c-statistic.

Results Among the 4 models tested, the bootstrap random forest had the best AUC in the test set and was the best model to predict recurrence in HGEC; the AUCs were 85.2%, 74.1% and 71.8% in the training, validation and test sets respectively. The top 5 predictors were: stage, uterus height, specimen weight, adjuvant chemotherapy and pre-operative histology. When stratified by stage, the AUC in the test set increased to 77% for Stage III and 80% for Stage IV. For time to recurrence, there was no difference between the Lasso and Cox Proportional Hazards models (test set c-index 71%) while the random forest had a c-index of 60.5%.

Conclusions A bootstrap random forest model best predicted recurrence in HGEC; model prediction further improved in Stage III and IV patients. Machine learning survival models performed similar to Cox Proportional Hazards but could be conducted with greater efficiency.

EPV098/#177

PROSPECTS FOR IMPROVING THE METHODS OF COMPLEX TREATMENT OF PATIENTS WITH ENDOMETRIAL CANCER STAGE I

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Objectives The analysis was performed in 968 women with endometrioid stage I endometrial cancer who underwent hysterectomy without/with adjuvant therapy (radiation or chemotherapy) in the Oncogynecology Research Department of the