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 MLH1 promoter hypermethylation and 84% (n=826) did not. Among those with MLH1 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 MLH1 promoter hypermethylated EC as compared with those with MSS disease. Patients with MLH1 promoter hypermethylated EC should follow general CRC screening guidelines.

**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.

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

**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 HGE C; the AUCs were 85.2%, 74.1% and 71.8% in the training, validation and test sets respectively. The top 5 predictors were: stage, uterine 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 HGE C; 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.

**Objectives** The analysis was performed in 968 women with endometrioid stage I endometrial cancer who underwent hysterec tomy without/with adjuvant therapy (radiation or chemotherapy) in the Oncogynecology Research Department of the
National Cancer Institute from 2015 to 2020. Although three-quarters received adjuvant treatment, recurrences occurred on average during the first three years.

**Methods** To evaluate the survival of patients with endometrioid stage I endometrial cancer depending on the type of treatment or their combination. The following statistical methods were used: standard descriptive and parametric. Survival of patients was analyzed by Kaplan-Meier method. P values of < 0.05 were considered significant.

**Results** Overall recurrence-free survival was 92.58 ± 7.38% with a median non-recurrence survival of 34.3 ± 14.7 months. A total of 68 relapses were detected - 7.02%. The median time from hysterectomy to the first recurrence, local and regional, was 6–18 months, respectively, and 24–36 months after combination treatment. The best survival was in the group of patients who received both surgical and chemotherapeutic treatment - averages of 59.5 months, and the worst after surgery - an average of 26.8 months (X^2 = 1,031,417, p = 0, 59708) (See figure 1).

**Conclusions** Hysterectomy shows the most common recurrences of loco-regional, and the combination of surgical treatment with radiation therapy - increases the frequency of distant metastases. Surgical treatment with radiation or chemotherapy leads to improve recurrence-free survival.

**EPV099/#179 ROBOTIC-ASSISTED SURGERY FOR ENDOMETRIAL CANCER IN MORBIDLY AND EXTREMELY MORBIDLY OBESE PATIENTS**

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**Objectives** We sought to evaluate the outcome of robotic-assisted surgery for endometrial cancer in morbidly obese (MO) and extremely morbidly obese (EMO) patients. We retrospectively reviewed all robotic gynecologic oncologic surgeries performed for endometrial cancer, in women with a BMI ≥40kg/m2, from 2012 to 2017 in our center. Patients were divided into two groups (MO: 40–49kg/m2, EMO: ≥50kg/m2). Complications and outcome were compared. Fisher’s test, t-test and Kaplan-Meier were used for statistical analyses.

**Results** Eighty-seven women were included: 64 (74%) MO and 23 (26%) EMO. The main histology was endometrioid adenocarcinoma (77% of MO and 61% of EMO) and endometrial intraepithelial neoplasia (19% of MO and 35% of EMO). The median blood loss was 100mL in MO and 75mL in EMO (p=NS). The median length of stay was one day for each group (range: 0–11). Two EMO (9%) and none of the MO patients required conversion to laparotomy due to poor surgical field exposure (p=0.067). Overall, 5 MO patients (8%) and 5 EMO (22%) had a surgical complication (p=0.12), but only 3 patients (1 MO and 2 EMO) required re-hospitalization within 30 days. The median follow-up was 47.7 months (range: 1.43–93.6). Recurrence occurred in 9% in each group, with no difference in recurrence-free survival (p=0.96). Only one MO patient died of cancer recurrence.

**Conclusions** The robotic-assisted surgery for endometrial cancer in morbidly obese patients is a safe and feasible procedure. The morbidity and extreme morbidly obese patients appear to have similar oncologic outcome, length of hospital stay, blood loss and low surgical complications.