Conclusions Two-thirds of patients with non-*BRCA* genes associated with increased risk of ovarian cancer were referred to gynecologic oncologists, with a 48% of uptake or RRS.

IGCS19-0639

231 RISK FACTORS FOR LYMPH NODES INVOLVEMENT IN OBESE WOMEN WITH ENDOMETRIAL CARCINOMAS

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Objectives To assess risk factors for lymph node involvement in patients with endometrial cancer and a body-mass index (BMI) $\geq$30 kg/m^2.
Methods A retrospective analysis was performed of obese patients diagnosed with endometrial carcinoma between 2007 and 2015, treated in a single center in Montreal. Preoperative variables evaluated were age, BMI, parity, and preoperative ASA score, grade, CA-125 and histology. Odds ratios (OR) and hazard ratios (HR) and their respective 95% confidence intervals (95%CI) were calculated using multivariable logistic regression and Cox proportional hazard models.
Results The study included 230 women with BMI >30, 223 (97.0%) had complete staging. Pelvic lymph node involvement was detected in 26 patients (11.3%). Sentinel node detection and pelvic lymph node dissection decreased with increasing BMI (adjusted OR 0.86, 95% CI 0.76–0.97 and 0.76, 95%CI 0.59–0.96, respectively, per 1 kg/m^2 increment). Pelvic lymph node involvement was inversely correlated with BMI (adjusted OR 0.88, 95% CI 0.79–0.99) and present in 16/85 (18.8%), 6/56 (10.7%), and 4/82 (4.9%) of patients with a BMI of 30.0–34.9, 35.0–39.9, and $\geq$40.0 kg/m^2, respectively. Preoperative CA-125 was associated with lymph node involvement (adjusted OR 2.77, 95%CI 1.62–4.73, per quartile increment). During a median follow-up of 72 months, a higher BMI was not associated with worse recurrence-free survival (adjusted HR 1.04, 95%CI 0.98–1.10), disease-specific survival (adjusted HR 0.97, 95%CI 0.88–1.06), or overall survival (adjusted HR 0.92, 95% CI 0.84–0.99).
Conclusions Pelvic lymph node dissection might be omitted in selected cases of morbidly obese patients with failed sentinel nodes mapping and a low CA-125.

IGCS19-0642

232 SURGICAL WAIT TIME MIGHT IMPACT SURVIVAL IN SELECTED PATIENTS WITH ENDOMETRIAL CARCINOMA

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Objectives To determine whether surgical wait time impacts survival in patients with endometrial cancer (EC).
Methods A retrospective analysis was performed, including all patients diagnosed with EC between 2008 and 2015 treated in a single centre in Montreal. Associations between surgery wait times and other variables were calculated using univariable linear regression models. In survival analysis, Cox proportional hazard models were used to calculate hazard ratios (HR); covariates included age, BMI, year of diagnosis, ECOG score, and tumor histology, grade and stage at surgery.
Results The study included 358 patients with median follow-up of 5.9 years, categorized into four groups based on their wait time: 89, 87, 91, and 91 women with a median wait time of 37 days (8–49), 62 days (50–70), 91 days (71–103), and 91 days (104–869), respectively. Increased surgical wait time was associated with lymph-vascular space invasion and distant metastases. A surgery delay of 71 days or more (median) did not result in significantly worse overall (p=0.54) or EC specific survival (p=0.27), while known prognostic variables such as stage and grade at surgery did (p<0.05). In a subgroup analyses, patients with grade II tumors who had more than 71 days between biopsy and surgery (n=51) had significantly (P=0.037) more EC-related mortality (5-year cancer-specific survival 83.9%, versus 100% for those with surgery within 70 days). Similarly, surgery delay had impact on overall survival also in patients with BMI>30 (HR 1.43, 95% CI 1.00–2.03).
Conclusions While surgery delay might predict outcome in specific subgroups, biological tumor determinants seems more important for survival outcome.

IGCS19-0304

233 CLINICAL NEXT-GENERATION SEQUENCING PIPELINE FOR BRCA MUTATIONS PROVIDES TRACEBACK TO THE FAMILIES OF DECEASED OVARIAN CANCER PATIENTS

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Objectives Advances in Next Generation Sequencing (NGS) allow for multiple gene analysis in an efficient, cost-effective manner. Deceased ovarian cancer patients untested for germline BRCA mutations represent a missed opportunity for clinicians to prevent future cancers in their surviving relatives. Families of this lost cohort can benefit through Traceback initiatives. We sought to validate our London Health Sciences custom Hereditary Cancer Panel using formalin fixed paraffin embedded (FFPE) tumor samples to assess BRCA 1/2 status in a cohort of high grade serous ovarian cancer (HGSC) patients.
Methods FFPE samples from 150 deceased HGSC patients were assessed using an Illumina MiSeq sequencer with a mean coverage of 1000x and average minimum coverage of 700x when 24 samples were tested per run. Validation of a subset of identified variants was then undertaken using Sanger and Multiplex Ligation-dependent Probe Amplication (MLPA).

Results Among 150 HGSC samples, we identified 44 samples (29.3%) with reportable variants with variant allele frequencies from 10.3% - 99.4%. These included 35 point mutations/insertions/deletions, 7 exon/whole gene deletions, and 2 BRCA1 exon 13 duplications. A subset (26) of these variants were then confirmed by targeted assays using Sanger and MLPA.
Conclusions Utilizing NGS technology, we reliably identified BRCA mutations in FFPE tumor samples. A validated NGS pipeline provides a valuable clinical tool to conduct Traceback initiatives to the families of deceased ovarian cancer patients never tested for germline mutations.

IGCS19-0126

234 CLONALITY ANALYSIS OF SYNCHRONOUS ENDOMETRIAL AND OVARIAN CARCINOMAS IN PATIENTS WITH LYNCH SYNDROME

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Objectives Sporadic synchronous endometrial (ECs) and ovarian cancers (OCs) have been shown to be clonally-related and to likely constitute metastases from each other. We sought to define whether synchronous ECs/OCs in patients with Lynch syndrome would be clonally-related or independent tumors.
Methods We subjected synchronous ECs/OCs from four patients with clinically confirmed Lynch syndrome to massively parallel sequencing targeting 468 cancer-related genes. Somatic mutations, copy number alterations, clonal relatedness and clonal decomposition were performed using state-of-the-art bioinformatics methods.
Results All synchronous ECs/OCs were considered independent primaries based on clinico-pathologic criteria. Sequencing analysis revealed that the ECs and OCs of three cases harbored strikingly similar repertoires of somatic mutations and gene copy number alterations and were clonally related. Specifically, in one case (LS2), a subset of subclonal mutations in the EC became clonal in the OC, suggesting that the ovarian tumor originated from the endometrial tumor. In contrast, in another case (LS5), the EC and OC harbored distinct somatic mutation profiles with no shared mutations; consistent with them constituting two independent primary tumors. In this case (LS5), a *PTEN* mutation and loss of protein expression were found to be restricted to the EC.
Conclusions Akin to sporadic synchronous ECs/OCs, the majority of Lynch syndrome-related synchronous ECs/OCs originate from a single primary tumor at variance with their clinical-pathologic diagnosis. Given that in the context of Lynch syndrome, synchronous ECs/OCs may be independent primary tumors, Lynch syndrome testing should be considered when synchronous ECs/OCs present with distinct genetic or immunohistochemical profiles.

IGCS19-0671

235 ABO, RH, KELL AND MN SYSTEMS WITHIN UTERINE CANCER

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Objectives The blood group antigens are found in several human cell types. There is a variable expression of histo-blood groups antigens within normal endometrium, which are also involved in hormonal regulation of glycosyltransferase activity. We aimed to investigate ABO, RH-hR, Kel and MN systems in women with Uterine cancer (UCa) in Adjara (Georgia) population.
Methods Internationally recognized immunoserology methods were used to reveal the erythrocyte group antigens. The obtained results were statistically processed by using appropriate formulas.
Results From ABO system alleles, r and q alleles frequencies were higher within the patients with UCa compared with control group of patients. From Rh-Hr system, the distribution frequencies of D, c and e alleles, Cc, Ee and EE genotypes, cDe, cDE haplotypes are increased in UCa. From Kell q(k) and p (K) alleles, p(k) allele tends towards to UCa in the tumor. Notably, MN system p (M) and q(N) alleles, revealed q(N) allele increased frequency in UCa compared to control group.
Conclusions Based on our study, ABO, RH-hR, Kell and MN systems antigens may be may be useful for UCa predisposition and development.

IGCS19-0606

236 BEHAVIORAL AND PSYCHOLOGICAL IMPACT OF ONCOGENETIC COUNSELING FOR HEREDITARY BREAST CANCER

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Objectives Oncogenetic counseling for hereditary breast cancer is an important tool for populational cancer risk screening. Individuals submitted to oncogenetic counseling must be aware about the social and emotional impact of genetic test result in their lives.
Methods Ten years follow-up of a cohort submitted to oncogenetic counseling in Brazil was evaluated for the health perception, depressive or anxious symptoms and life habits/behaviour before and after the oncogenetic counseling.Survival analyzes were performed using the Kaplan-Meier method and the Log-Rank test was used to verify the existence of significant differences.
Results 139 patients have been submitted to oncogenetic counselling, 135 women and 4 men; 113 patients had early onset