(EEC) are p53abn. There is debate whether these are misclassified glandular variants of serous EC, and whether the risk of recurrence justifies adjuvant therapy. Here, we aim to determine if blinded expert pathology review classifies p53abn EC as low-grade EEC and assess risk of recurrence.

Methods p53abn low-grade EEC from retrospective cohorts and the PORTEC-1&2 trials were included. Review of histotype and grade was performed by six expert gynaecopathologists, blinded to molecular class and study aim. Cases were considered low-grade p53abn EEC if ≥1 expert assigned it as such. Kaplan-Meier’s method and the log-rank test were used for survival analysis.

Results 72 low-grade p53abn EEC were included. ≥1 pathologists assigned low-grade EEC in 53 (73.6%), and ≥50% of pathologists in 36 (50%) cases. The 5-year recurrence-rate was 31.9% (95%CI: 19.4–42.5%) and 29.5% (95%CI 16.9–40.3%) among those (N=67) with stage I disease. Within stage I and those assigned by ≥1 pathologist as low-grade p53abn EEC, the 5-year recurrence-rate was 22.9% (95%CI: 9.2–34.6%); 21.2% (95%CI: 2.3%-36.4%) in stage IA and 26.3% (95%CI: 3.6–43.7%) in stage IB (figure 1).

Conclusions We show the p53abn molecular subtype of EEC encompasses a subset of low-grade EEC which are associated with a substantial risk of disease recurrence. Assessment of molecular classification in all low-stage low-grade ECs will enable detection of patients with p53abn EC who may benefit from adjuvant therapy.

EP117/#763 APPLICATION OF SHALLOW WHOLE GENOME SEQUENCING TO IDENTIFY THERAPEUTIC OPPORTUNITIES IN P53ABN ENDOMETRIAL CANCERS

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10.1136/ijgc-2022-igcs.208

Objectives Shallow whole genome sequencing (sWGS) has been successfully used to derive copy number (CN) signatures in high grade serous ovarian cancer (HGSOC), recognizing two signatures associated with homologous recombination deficiencies (HRD). p53abn ECs share genomic features with HGSOC, supporting application of this platform to stratify this aggressive EC molecular subtype.

Methods DNA was extracted from formalin fixed paraffin embedded (FFPE) tumor cores of 203 p53abn ECs and sWGS performed. CN signatures were derived from absolute copy numbers using Rascal (relative to absolute copy number scaling tool), CN amplification of CCNE1 and ERBB2 was called based on CN alterations ≥5, and comparisons were made to CCNE1 and HER2 immunohistochemistry (IHC).

Results HRD-related signatures 3 and 7 were found in 42 p53abn ECs (30 and 12 respectively) encompassing 26% of the 161 cases where CN signatures could be assigned. CN amplification in CCNE1 was identified in 26/203 (13%) with CCNE1 IHC overexpression (2/3+) found in 64% of cases. ERBB2 amplification was observed in 22/203 (11%) with HER2 IHC overexpression (2/3+ on whole stained sections) in 21% and significant intratumor heterogeneity was noted.

Conclusions sWGS is a relatively inexpensive tool that can be performed on FFPE, and may be used to identify opportunities for PARPi therapy, with 26% of p53abn EC identified as having HRD signatures. Opportunities for anti-HER2 therapy and targeting CCNE1 (Wee1) were also identified, although IHC detected a significantly greater proportion of p53abn ECs overexpressing HER2 and CCNE1 compared to CN amplification calls on sWGS. This may, in part, be explained by intratumor heterogeneity.

EP118/#744 PROGRESS IN UTERINE CANCER SURVIVAL IN THE UNITED STATES FROM 2004–2016: WHO WAS LEFT BEHIND?

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10.1136/ijgc-2022-igcs.209

Objectives To establish trends in uterine cancer survival rates based on histology and race in the U.S. over a 13-year period.

Methods Data for patients with uterine cancer were acquired from the National Cancer Database from 2004–2016. Demographics, clinico-pathologic factors, and survival information were extracted and tested using Kaplan-Meier and Cox proportional-hazard models for each time interval.

Results Of 487,385 women with uterine cancer, 467,258 (95.9%) had epithelial and 17,184 (3.5%) mesenchymal tumors. The study period was divided into three time intervals from 2004–2007, 2008–2012, and 2013–2016. The survival rate over time increased from 49.9 to 50.7 to 51.1% (p<0.001). Younger patients (<50 years old) had no improvement in survival (55.4 to 55.4 to 55.5%; p=0.9), whereas older patients had slight improvement (49.1 to 50.0 to 50.6%; p<0.001). There was a marginal clinical increase in Blacks from 41.6 to 43.1 to 44.2% (p<0.001), Whites 50.8 to 51.5 to 51.8% (p<0.001), and Hispanics from 51.0 to 52.0 to 52.86% (p<0.001); however, no change observed in Asians (52.4 to 52.3 to 52.9%; p=0.16). Furthermore, there was a lack of improvement in clear cell carcinomas (41.5 to 41.8 to 42.9%; p=0.099) and mesenchymal tumors (37.0 to 37.2 to 36.8%; p=0.27). Survival of those with serous carcinomas has the largest increase (38.6 to 40.8 to 42.6%; p<0.001).

Conclusions In this large population study, the overall survival of uterine cancer patients had improved statistically, but may not be clinically meaningful. Moreover, there was a lack of improvement among young patients, Asians, clear cell and mesenchymal tumors.