the calculated predictive probability values were significantly different between the LNM-positive and -negative groups ($P = 1.39 \times 10^{-10}$), and high diagnostic accuracy of 83.6% area under the curve (AUC) was obtained. The LNM diagnosis requires essentially minimize the time difference between the diagnosis and hysterectomy. Therefore, reverse transcription-polymerase chain reaction enabled quantification from RNA in one step within 30 min, for intraoperative diagnosis.

Conclusion This diagnostic method uses rapid nucleic acid amplification for intraoperative quantification of biomarkers in the primary tissue. Furthermore, the predictive model combined with various clinical variables can be used to discriminate LNM with high accuracy and facilitate individualization of the surgical treatment.

**2022-RA-809-ESGO** UNDERLYING CAUSES AND PROGNOSIS OF MISMATCH REPAIR DEFICIENCY IN ENDOMETRIAL CANCER OTHER THAN MLH1 PROMOTER HYPERMETHYLATION

**Abstract 2022-RA-809-ESGO Figure 1** Kaplan-Meier survival curves for recurrence-free survival for patients with LS-associated EC (germline mutation in MMR gene) and other non-LS-associated MMR EC. All Cases with MMRd phenotype without POLE mutation and to explore the reasons why AEH patients opted for conservative management.

**Conclusion** Identification of an underlying cause for unmethylated MMRd is feasible in the majority of EC cases applying matched tumor-normal tissue NGS. A significant proportion was confirmed to be LS-associated or sporadic MMRd, while only a small subset remained unresolved. Although this distinction did not carry prognostic relevance, identification of definitive sporadic cases may release patients and relatives from burdensome LS-surveillance.

**2022-RA-815-ESGO** ENDOMETRIAL CANCER INCIDENCE IN PATIENTS WITH ATYPICAL ENDOMETRIAL HYPERPLASIA ACCORDING TO MODE OF MANAGEMENT

**Abstract 2022-RA-815-ESGO**

Introduction/Background The vast majority of mismatch repair-deficient (MMRd) endometrial carcinomas (EC) are due MLH1 promoter hypermethylation. Here, we aimed to investigate the prevalence, prognosis and underlying causes (including Lynch syndrome (LS)) of MMRd EC other than MLH1 promoter hypermethylation.

Methodology From the 409 MMRd ECs that were identified by MMR-immunohistochemistry (IHC) in the PORTEC-1,-2 and -3 trials, 97 cases did not have MLH1 promoter hypermethylation. These 97 cases were analyzed by matched tumor-normal tissue NGS. A significant proportion of the 409 MMRd is feasible in the majority of EC cases applying matched tumor-normal tissue NGS. A significant proportion was confirmed to be LS-associated or sporadic MMRd, while only a small subset remained unresolved. Although this distinction did not carry prognostic relevance, identification of definitive sporadic cases may release patients and relatives from burdensome LS-surveillance.

Results In 34 cases (35%) a germline MMR mutation (LS-associated) was identified of which 8 (24%) had a second somatic hit. Upon excluding LS-associated ECs, a somatic alteration in MMR genes was observed in 52% ($n=33$), including double somatic hits in 35% ($n=22$). In the remaining 48% of cases ($n=30$) no MMR mutation was found of which the majority ($n=22$) confirmed MSI. Rereview of all (discrepant) MMR-IHC did not reveal misinterpretation of MMRd status. Somatic POLE mutations were identified in 7/97 cases (7%). The 5-year RFS did not differ significantly between LS-associated and non-LS-associated MMRd EC (5-year RFS 94.1% [95% CI 86.5–100%] vs 93.5% [95% CI 87.5–99.9%], respectively; $p=0.72$; figure 1).