prognostic impact was independent of clinicopathological and molecular factors (adjusted HR 0.32 95%CI 0.14-0.74, p=0.0077).

Conclusion* L1CAM identifies tertiary lymphoid structures with germinal centres. Our data suggest a pivotal role of TLS in the risk of recurrence of EC. L1CAM IHC is simple, available across many study cohorts and could be readily implemented as biomarker of TLS in future trials and clinical care.

486 CLINICOPATHOLOGICAL CHARACTERISTICS OF WOMEN WITH *CTNNB1*-MUTATED ENDOMETRIAL CANCER

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Introduction/Background* The molecular characterisation of endometrial cancer (EC) represents a step towards personalised management. The current ESGO-ESTRO-ESP guidelines classify EC into four groups: POLE mutated (POLEmut), p53 abnormal (p53abn), mismatch repair deficient (MMRd) and the largest group of no specific mutational profile (NSMP). Women with NSMP tumours generally have a good prognosis, but if disease recurs, the prognosis tends to be poor. A proposed additional molecular classifier to improve the risk assessment are mutations of catenin beta 1 (*CTNNB1*). The aim of this study was to assess the clinicopathological characteristics of women with *CTNNB1*-mutated tumours for further risk assessment.

Methodology This prospective observational study included women diagnosed with endometrial cancer between January 2020 – March 2021 at the University Medical Centre Maribor, Slovenia. Immunohistochemical (IHC) staining was used to evaluate the expression of p53 and mismatch repair proteins MLH, MSH2, MSH6 and PMS2. Sanger sequencing of exons 9, 13 and 14 was used to determine the *POLE* status and of exon 3 for *CTTNB1* status. Statistical analysis was performed using IBM SPPS version 23. Descriptive statistics were calculated for numerical variables. Chi-Square (χ^2) test was used to evaluate the relationship between *CTNNB1* status and the tumour stage, depth of tumour invasion and lymph node involvement.

Result(s)* Out of 45 women included in the study, 5 (11.1%) were found to have a mutation in the exon 3 of *CTNNB1*; 2 women in D32V (40%), 2 women in S32C (40%) and 1 woman in S37P (20%). Among them, 4 women (80%) were classified as NSMP and 1 (20%) as p53abn. Moreover, 2 women (40%) were diagnosed with early stage (FIGO I-II) and 3 (60%) with advanced stage (FIGO IIIa or more) EC. *CTNNB1* status was not correlated with lymph-node involvement (p>.418) and myometrial (p>.802) or lympho-vascular space invasion (p>.855).

Conclusion* *CTNNB1* testing could be used for further classification of molecularly undefined EC. Especially in the NSMP group, this could provide more information about the disease biology and lead to better management of women. Further evaluation of the long-term impact of *CTNNB1* mutations on recurrence-free survival and overall survival is needed.

497 ENDOMETRIAL HYPERPLASIA: RISK OF COEXISTENCE AND PROGRESSION TO ENDOMETRIAL CARCINOMA. RETROSPECTIVE COHORT STUDY

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Introduction/Background* Endometrial hyperplasia (EH) is characterized by an irregular proliferation of the endometrial glands with an increased gland/stroma ratio compared to the proliferative endometrium. The risk of malignancy depends on the presence of the atypia. The purpose of this study was to determine incidence of concomitant endometrial carcinoma (EC) and the risk of malignancy of the disease.

Methodology

Retrospective cohort study was performed It includes a total of 120 patients diagnosed of EH by endometrial biopsy at Hospital Universitario 12 de Octubre between January 2015 to January 2020. The epidemiological and clinical characteristics of the patients were analysed. The incidence of concomitant EC was investigated in patients in whom hysterectomy was performed. Also in cases of expectant management and/or medical treatment, the rate of regression, persistence an progression of EH was studied.

Result(s)* According to the criteria of the 2014 WHO classification, 70.8% of cases were EH without atypia and 29.2% with atypia. The mean age at diagnosis was 48.9 years and 71.7% of the patients were premenopausal.. The most frequent symptom was abnormal uterine bleeding and the most ultrasound finding was pathological endometrial thickness (52.5%), followed. A suspected endometrial polyp (21.7%). Hysterectomy was performed in 25% of the total cases. In this group of patients, the incidence of concomitant EC was 33.3%, all of them in EH with atypia. In the group of patients with medical or expectant treatment, after a mean follow-up time of 25 moths, a regression of the disease was observed in 95.4% and a progression to EC in 2.3%.

Conclusion* Although EH is a benign disease entity, with a high regression-cure rate, its risk of coexistence with EC is not negligible, especially in cases where atypia is observed.

501 LOX1 AND NALP3: FROM IMMUNE TOLERANCE DISRUPTION IN PREGNANCY COMPLICATIONS TO IMMUNE ESCAPE IN ENDOMETRIAL CANCER

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Introduction/Background* Endometrial cancer (EC) patients have a good prognosis at early stages, but for recurrent or metastatic EC the prognosis remains poor. EC treatments are related to known prognostic factors included in ESMO-ESGO-ESTRO risk classes classification, but they are not sufficient to predict outcomes or recurrence rate of early stages. To improve patient clinical management and allow personalized therapy a better characterization of risk classes in EC is needed. To fill this gap, we investigated EC immune escape processes, customized on the knowledge of maternal-fetal interface immune mechanisms, since the two processes share common pathways.

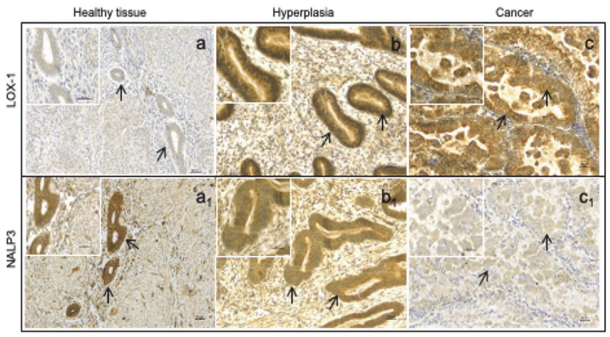
Methodology This has been addressed by the identification of potential shared immune-based signatures between maternal-fetal interface and EC, such as those linked to lectin-type oxidized LDL receptor 1 (LOX-1) and NALP3 inflammasome, in order to achieve a potential immune score implementation to better characterize EC risk classes. The immunohistochemical assessment of LOX-1 and NALP3 was performed on formalin-fixed paraffin-embedded (FFPE) endometrial tissues.

Result(s)* 41 patients divided in 3 groups were enrolled: healthy endometrial tissue, endometrial hyperplasia and EC. We detected an increased expression of LOX-1, by immunohistochemistry (IHC), within the endometrial carcinoma tissues, a lower expression in cases of hyperplasia, to arrive to an absent staining in the healthy endometrial tissue (*p< 0.05, *Kruskal-Wallis followed by Mann-Whitney test). This grading is inverted in NALP3, which expression appears to be lower in EC (*p< 0.05). A proportional relationship between LOX-1 and NALP3 expression was demonstrated (p=0.006, Spearman test, confirmed through a linear regression test): increasing the expression of LOX-1, NALP3 decreases.

Conclusion* An increased LOX-1 and a decreased NALP3 expression seems be associated with EC progression. To identify patients at risk of developing EC from pre-cancerous lesions, by searching potential immune prognostic factors, such as LOX-1 and NALP3 on endometrial biopsy, could redefy the actual EC risk classes through a potential 'immune score' creation. Nevertheless, further studies are needed to define EC transcriptome immune-based signature. Furthermore, pathways detected by deciphering the immune changes linked to EC progression, could be potential target for immunotherapy.

Abstract 501 Table 1 Clinical and pathologic features of enrolled features

CHARACTERISTICS	Physiologic endometrium (n 7)	Endometrial Hyperplasia (n 22)	Endometrial cancer (n 12)
Mean Age ± S.D.	58 ± 13 years	51.8 ± 10.4	50.7 ± 10.5
Mean BMI ± S.D.	26.5 ± 5.1 kg/m^2	26.8 ± 7.2	32 ± 9.1
Menopausal Status			
YES	5	10	9
NO	2	12	3
FIGO Stage			
IA	NA	NA	6
IB			6
Hysteroscopy with endometrial biopsy		11	
Total hysterectomy	7	11	
Radical Hysterectomy			
Type A			12
Lymphadenectomy			
Not performed			6
Pelvic	NA	NA	6
Para-Aortic			0
Grading			
1			4
2	NA	NA	4
3			4
EC Histology			
Endometrioid	NA	NA	12
Recurrences	NA	NA	0



Abstract 501 Figure 1 Immunohistochemical staining on formalin-fixed paraffin-embedded samples of endometrical tissue showing expression of LOX-1 and NALP3 markers

506 DOES ANANDAMIDE PREVENT ENDOMETRIAL CANCER GROWTH THROUGH ANTI-PROLIFERATION OR MODULATION OF APOPTOSIS?

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Introduction/Background* Previously, we and others have demonstrated that the N-acylethanolamine, anandamide, inhibits endometrial cancer (EC) cell growth in-vitro1,2. The mechanism probably involves cellular apoptosis2, through modulation of pro-apoptotic (BAX) and anti-apoptotic molecules (Bcl-2). Our aim here was to investigate the distribution patterns of these proteins and that of a cell proliferation marker in patients with and without EC.

Methodology Endometrial biopsies from patients with Type 1 (n=18), Type 2 EC (n=10) and normal atrophic endometria (n=6) were subjected to immunohistochemistry with commercial antibodies to BAX, Bcl-2 or Ki-67. Histomorphometric analyses of the glands and stroma were measured independently on 10 random fields from each sample using Imagescope software3. The mean ± SEM were calculated, and significance (p<0.05) determined using ANOVA.