chemotherapy with radiotherapy conferred better OS than chemo alone (36 months vs 4 months, $p = 0.006$).

Conclusion* Non-uterine carcinosarcomas had poorer survival than their uterine counterparts. Our cohort of uterine carcinosarcomas had more patients with early-stage disease.. Nodal involvement carries poor prognosis. On subgroup survival analysis, adjuvant radiation in combination with chemotherapy showed benefit, with significant effect seen in stage III cancers.

573 CIMICIFUGA RACEMOSA EXTRACT EFFECTS ON ENDOMETRIAL AND OVARIAN CELL LINES

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Introduction/Background* In postmenopausal women estrogen levels depend exclusively upon the local formation from steroid precursors dehydroepiandrosterone-sulfate and estrone-sulfate (E1-S). The reduced estrogen levels are associated with menopausal symptoms, which often occur in peri- and postmenopausal patients. To mitigate these symptoms nowadays more women choose medicine of natural origin, e.g. extracts from *Cimicifuga racemosa* (CE) instead of hormone replacement therapy, which is associated with increased risk of breast cancer, stroke and pulmonary embolism. While CE treatment is considered as safe, little is known about its effects on healthy endometrial or ovarian tissue and even less on hormone-dependent malignancies like endometrial and ovarian cancer that arise in this population of women. The aim of our study was to examine the influence of CE on the expression of genes encoding E1-S transporters and estrogen biosynthetic and metabolic enzymes in control and cancerous endometrial and ovarian cell lines.

Methodology Control endometrial cell line (HIEEC), control ovarian cell line (HIO80) and cell lines of well differentiated endometrial carcinoma (Ishikawa and HEC-1-A), moderately differentiated adenosquamous carcinoma (RL-95-2), poorly differentiated endometrial carcinoma (KLE) and high grade ovarian serous adenocarcinoma (Kuramochi, COV62 and OVSAHO) were exposed to CE in different concentrations for 72h. The expression of 9 E1-S transporter genes (*SLCO4C1*, *SLC51A*, *ABCC1*, *ABCC4*, *SLC10A6*, *SLCO1A2*, *SLCO2B1*, *SLCO3A1*, *SLCO4A1*) and 4 genes encoding estrogen biosynthetic/metabolic enzymes (*HSD17B1*, *HSD17B2*, *STS* and *SULT1E1*) and estrogen receptors (*ESR1* and *ESR2*) were measured using RT-qPCR.

Result(s)* The results revealed that CE affects the expression of genes encoding E1-S transporters and estrogen biosynthetic and metabolic enzymes only at very high concentrations of CE (50µg/ml or 100µg/ml), while no changes in expression were observed with concentrations that are similar to those detected in plasma of CE users.

Conclusion* Our research presents an insight of CE effects on endometrial or ovarian cancer CLs at the mRNA level showing additional proof of safe usage of CE in healthy women and women with hormone-dependent malignancies like endometrial and ovarian cancer.

577 MOLECULAR FEATURES AND PROGNOSTIC IMPACT OF MELF TYPE MYOMETRIAL INVASION IN THE PORTEC-1/2 COHORT OF EARLY STAGE ENDOMETRIAL CANCERS

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Introduction/Background* Microcystic, elongated fragmented (MELF) pattern of myometrial invasion is a distinct histologic feature occasionally seen in low-grade endometrial carcinomas (EC). The prognostic relevance of the presence of MELF is uncertain due to conflicting data and has not appropriately been studied in the context of the novel molecular EC classification. We aimed to determine the relation of MELF pattern of invasion with clinicopathological and molecular characteristics, and define its prognostic relevance in early stage (high) intermediate risk EC.

Methodology Single haematoxylin and eosin (H&E) stained whole tumour slides of 929 of the 1141 early stage (high) intermediate risk EC of patients included in the post-operative radiation therapy in endometrial carcinoma (PORTEC)-1/-2 trials were available for review for the presence of MELF. Histological type, stage and grade, presence and extent of lymphovascular-space-invasion (LVSI), molecular subclass, L1-cell-adhesion-molecule (L1CAM) overexpression, and β -catenin exon-3 (*CTNNB1*) and *KRAS* mutational status were compared between MELF-positive and negative cases. Differences in patient and tumour characteristics were analysed with chi-square or Fisher's exact test for categorical and Mann-Whitney *U* test for continuous variables. Time-to-event analyses were done using the Kaplan-Meier method, log-rank tests and Cox' proportional hazards models.

Abstract 577 Table 1 Clinicopathological features of MELF positive and negative cases

	Total (n=929)	No MELF (n = 800)	MELF (n = 129)	P-value
Age				
mean (range)	68 (67 – 68)	68 (67 – 68)	68 (67 – 69)	0.88
Histotype				
endometrioid	907 (97.7%)	781 (97.6%)	127 (98.4%)	0.54
non-endometrioid#	21 (2.3%)	19 (2.4%)	2 (1.6%)	
Stage				
IA	287 (30.9%)	280 (35.0%)	7 (5.4%)	<0.0001
≥IB*	641 (69.1%)	520 (65.0%)	122 (94.6%)	
Grade				
1-2	792 (85.3%)	668 (83.5%)	125 (96.9%)	<0.0001
3	136 (14.7%)	132 (16.5%)	4 (3.1%)	
LVSI				
none or mild	823 (95.3%)	712 (96.0%)	112 (91.8%)	0.06
substantial	40 (4.6%)	30 (4.0%)	10 (8.2%)	
L1CAM				
≤10% (negative)	750 (94.6%)	638 (94.0%)	113 (98.3%)	0.03
>10% (positive)	43 (5.4%)	41 (6.0%)	2 (1.7%)	
TCGA molecular subgroups				
POLE mutant	57 (7.1%)	55 (8.0%)	2 (1.8%)	
MMRd	226 (28.2%)	212 (30.8%)	14 (12.5%)	
p53 abnormal	38 (4.7%)	37 (5.4%)	1 (0.9%)	
NSMP	480 (60.0%)	386 (55.9%)	95 (84.8%)	
CTNNB-1				
wild type	684 (79.7%)	575 (77.8%)	110 (91.7%)	<0.0001
mutation	174 (20.3%)	164 (22.2%)	10 (8.3%)	
KRAS				
wild type	714 (83.2%)	623 (84.3%)	92 (76.7%)	0.05
mutation	144 (16.8%)	116 (15.7%)	28 (23.3%)	