(105, 18%), does not want to discuss GT with family (55, 9%), relatives not interested (50, 9%), language (38, 7%), and other (27, 5%). Of 73 enrolled patients, 45 (62%) contacted at least one relative to discuss GT within two months of enrollment. Twelve patients had at least one relative who participated in our facilitated referral pathway, but only 2 (3%) relatives completed GT through our pathway. Two additional relatives underwent GT separately. Of 20 patients who completed 3-month psychological impact questionnaires, 13 (65%) had no regret, and 19 (95%) had none to subclinical range stress.

Conclusions Although over 50% of patients contacted family members regarding GT, only 3% had a relative undergo GT via our facilitated referral pathway. Comprehensive novel efforts to simplify access to GT for relatives are desperately needed.

Abstracts

**EP171/#1044 BESIDE BREAST/OVARIAN MALIGNANCY: OTHER CANCER RISK PROFILE IN BRCA1 AND BRCA2 PATHOGENIC VARIANTS**

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**Objectives** The clinical significance of genetic testing of BRCA1 and BRCA2 Pathogenic Variants (PV) in women with breast and ovarian cancers is widely recognized. However, there is paucity of evidence to include other cancer types in clinical management guidelines. We aim to study the prevalence of different types of cancers in BRCA 1 AND BRCA2 PV carriers and to associate those with the mutation type.

**Methods** This is a cross-sectional study of women carriers of BRCA1 and BRCA2 PV, who attended our designated carrier clinic in a tertiary medical center We compared cancer incidence among the three most prevalence PVs in Israel: 185del, 6174del and 5382insc.

**Results** A total of 2,230 women were included. BRCA1 mutation comprised 62.6% of the cohort, BRCA2 36.6% and 0.8% were carriers of both genes. Breast and ovarian/fallopian tube/peritoneal cancer was prevalent in 106 women(4.7%). Most prevalent other cancers were: cutaneous, 1.1%, colon, endometrial and cervical –0.6% each and thyroidal and pancreatic cancers, 0.4% each. The risk of any cancer was higher in 5382insc than 6174del and 185 del; 54.8% vs. 46.3% vs. 36.0% respectively, p<0.001. When excluding breast and ovarian/fallopian tube/peritoneal cancer, the risk of other cancer was similar among the three mutations: 9.3% in 5382insc, 7.2% in 6174del and 7.3% in 185del, (p=0.521).

**Conclusions** The results of this large-scale study suggest that pathogenic variants in BRCA1 and BRCA2 were associated with non-negligible risk of other cancers. This risk seems not to be associated with the type of mutation, however larger studies are warranted.

**EP172/#350 INTEGRATED MOLECULAR PROFILE OF PLATINUM RESISTANT EPITHELIAL OVARIAN CARCINOMA**

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**Objectives** Epithelial ovarian carcinoma (EOC) is a serious malignancy with high mortality due to late diagnosis and drug resistance development. Drug resistance is one of the major obstacles to successful anticancer therapy. The main aim of our study was to analyze molecular profile based on gene expression, DNA methylation level, and genetic variability in EOC patients stratified by the platinum therapy resistance status.

**Methods** For the present study, we selected 72 EOC patients with sensitive (N=43) or resistant (N=30) status. Whole genome expression of protein-coding genes was profiled by mRNA sequencing technology (N=60), lncRNA expression by whole transcriptome RNA sequencing (N=23), global methylation by DNA microarrays (N=50), and somatic mutation rate by whole exome sequencing (N=50).

**Results** Molecular profile of platinum resistant EOC patients differed from sensitive EOC patients in upregulation of five protein-coding genes (NEURL1, FGCBP, MMP11, NCAMI, and ARMC3) and five lncRNA (ADAMTS9-AS1, TCF21-AS1, ARM3C-AS1, LINC-HIST2H3PS2-35, and LINC-BCR-4), higher methylation of seven protein-coding genes (ABCC4, ABCB10, SLC1A7, SLC19A2, SLC50A1, XPC, and FOXO1) and three lncRNA (LINC00263, LINC00460, NEAT1) and higher frequency of mutations in TP53 gene. On the other hand, three protein-coding genes (LPL, CD36, FABP4), and three lncRNA (LINC-IGGL5, LINC-TME1M121-12, CHST6-AS1) were downregulated, lower methylation was observed for ATP1A1 gene, and the Hippo pathway genes were less mutated in resistant patients.

**Conclusions** Our study shows a complex network of dysregulated genes and gene expression products connected with the resistance status of EOC patients which should be further characterized. Supported by INTER-ACTION LTAUSA19032, GACR no. 21–140825, AZV no. NU010–09–00174 and GAUK no.1074120.