(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.

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

1,2Karolina Seborova*, 1,2Viktor Hlavac, 1,2Petri Holy, 3Sunivra Bjerkund, 4Thomas Fleischer, 1,2Albetta Spalenkova, 1Luksas Rob, 1Martin Hrudza, 1Petr Cernaj, 1Jiri Bouda, 1Vesela N Kristenst, 1,2Pavel Soucek, 1,2Radka Vlachikova, 1National Institute of Public Health, Toxicogenomics Unit, Prague, Czech Republic; 4Biomedical Center, Laboratory of Pharmacogenomics, Pilsen, Czech Republic; 3Institute of Clinical Medicine, Faculty of Medicine, University of Oslo, Department of Medical Genetics, Oslo, Norway; 3Institute of Cancer Research, Norwegian Radium Hospital Oslo University Hospital, Department of Cancer Genetics, Oslo, Norway; 5University Hospital Krakoove Virohodny and Third Faculty of Medicine, Department of Gynecology and Obstetrics, Prague, Czech Republic; 3University Hospital in Pilsen, Department of Gynecology and Obstetrics, Pilsen, Czech Republic; 4National Institute of Public Health, Toxicogenomics Unit, Praha, Czech Republic

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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, NCAM1, and ARMC3) and five lncRNA (ADAMTS9-AS1, TCF21-AS1, ARM3C-AS1, LINC-HIST2H3P52-35, 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-TMEM121-12, 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. NU20-09-00174 and GAUK no.1074120.