Conclusions Molecular profiles and TME are associated with OS. TME differs per profile, with higher immune cell densities showing a favorable OS, even within the profiles. HGSOc does not reflect one entity but comprises different entities based on molecular profile and TME which could assist with patient-tailored treatment in the future.
E-poster viewing: Basic/translational science

**EP001/#442 THE EFFECT OF MILD HYPERTHERMIA ON IMMUNE EVASION ACTORS PD-L1 AND NRRC5 IN OVARIAN CANCER**

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**Objective** Multiple preclinical studies have demonstrated the benefit of augmenting immunotherapy with hyperthermia (HT) considering the proven ability of HT to enhance immune cell immunogenicity and to stimulate an antitumor immune response primarily via heat shock proteins (HSP). However, antitumor immune responses are often invalidated by immune evasion mechanisms such as the overexpression of programmed death-ligand 1 (PD-L1) and the loss of MHC class I expression. In this context, we sought to investigate the effects of HT on PD-L1 and the transcriptional activation of MHC class I genes NRRC5 and their interplay in ovarian cancer.

**Methods** A co-culture of ovarian cancer cell lines (IGROV1 and SKOV3) with peripheral blood mononuclear cells was set up. Culture media conditioned with IGROV1 or SKOV3 subjected to HT were tested on untreated cell cultures. Knockdown of HSPA1 and HSPB1 and inhibition of STAT3 activation were performed. Expression levels of PD-L1, NRRC5, proinflammatory cytokines and HSP were measured.

**Results** HT produced a significant concomitant decrease of PD-L1 and NRRC5 expression in co-culture. Notably, however the conditioned media by heat shocked cells increased their expression. Knockdown of the HSPB1 gene reversed this increase, an effect enhanced by STAT3 activation inhibition. Correlation analysis showed a positive correlation between NRRC5 and PD-L1 (r=0.54, p-value <0.001) in TCGA database.

**Conclusions** Our results revealed that HSP27 induces a concomitant upregulation of PD-L1 and NRRC5 expression through the activation of a common regulator ‘STAT3’.