Conclusion In addition to the nephroprotective benefit, ST also appears to be associated with better cytoreductive results. Hyperhydration does not provide any additional benefit.

**UNRESECTABLE PERITONEAL METASTASES FROM STAGE III OVARIAN CANCER TREATED WITH BIDIRECTIONAL APPROACH OF PRESSURIZED INTRAPEITONEAL AEROSOL CHEMOTHERAPY (PIPAC) AND SYSTEMIC CHEMOTHERAPY MAY LEAD TO SECONDARY COMPLETE CYTOREDUCTIVE SURGERY: A PILOT STUDY**

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**METABOLOMICS SHOWED LSR PROMOTED LIPID METABOLISM IN EOC**

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**INTRODUCTION/BACKGROUND**

Previously, we identified lipolysis-stimulated lipoprotein receptor (LSR) as a new target of epithelial ovarian cancer (EOC), and we reported anti-tumor effect of our newly developed monoclonal antibody (mAb) against LSR-positive EOC cells in vitro and in vivo. We also demonstrated that in high-fat diet (HFD) mouse, anti-LSR mAb showed strong anti-tumor effect. In this study, we performed metabolomic analysis using HFD mouse serum and analyzed metabolic pathway of EOC via LSR.

**METHODOLOGY**

We established HFD mouse model and evaluated the tumor growth of LSR-positive EOC cell line and anti-tumor effect of anti-LSR mAb in this model. Moreover, we obtained serum samples from normal-diet (ND) and HFD mouse, and performed metabolomic analysis. Finally, we analyzed lipid metabolites profile of HFD mouse compared to ND mouse.

**RESULTS**

Tumor growth of LSR-positive EOC cells was significantly promoted in HFD mouse (p < 0.05) and anti-LSR mAb showed stronger anti-tumor effect in HFD mouse than that in ND mouse (57.2% and 26.6%, respectively). Metabolomic analysis using HFD and ND mouse serum detected 210 metabolites and The Human Metabolome Database provided comprehensive information of 83 metabolites. Principal component analysis and cluster analysis using these data showed obviously different metabolic properties between ND and HFD mouse. Partial Least Squares-Discriminant Analysis showed significantly high score of lipid metabolites including α-Tocopherol and cholesterol.

**CONCLUSION**

Metabolomics showed the activation of lipid metabolism in HFD mouse and suggested that LSR contributed tumor growth via lipid metabolism.