S02-3 **Cross-talking between EMT and cellular metabolic remodeling**

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Epithelial-mesenchymal transition (EMT) is a process that converts adherent epithelial cells into motile mesenchymal-like cells and is known to be involved in metastasis/invasion and chemoresistance in tumor cells. Recent studies have shown that EMT promotes cellular metabolic remodeling and thereby up-regulates glycolysis and glutaminolysis, suggesting that EMT induces tumor progression by increasing cellular motility and metabolic remodeling. However, molecular mechanisms of metabolic remodeling by EMT are largely unknown. We recently identified the transcriptional cofactor VGLL3 as a novel EMT inducer. Stable expression of VGLL3 was found to cause E-cadherin reduction and SNAIL induction and increase cellular motility in the human lung cancer A549 cells. Metabolome analyses showed that the amount of metabolites in the glycolysis and glutaminolysis pathways and polyamines was increased in VGLL3-expressing cells. Proliferation of VGLL3-expressing cells was increased compared with control cells, and this increment was repressed by treatment with the polyamine synthetase inhibitor DFMO. These results suggest that VGLL3 promotes EMT together with metabolic remodeling and thereby induces cellular motility and proliferation. Analyses of the detailed molecular functions of VGLL3 and its involvement in tumorigenesis are currently underway.