O47. **Emodin represses TWIST1-induced epithelial-mesenchymal transitions in head and neck squamous cell carcinoma cells by inhibiting the β-catechin and Akt pathways**

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Head and neck squamous cell carcinoma (HNSCC) is one of the leading causes of cancer deaths worldwide. In recent studies, a crucial link has been discovered between the acquisition of metastatic traits and tumour-initiating abilities in cancer cells during the epithelial-mesenchymal transition (EMT).Herein, we demonstrated that the ectopic expression of *TWIST1*, the EMT regulator, in HNSCC FaDu cells triggered EMT and resulted in the acquisition of a mesenchymal phenotype. Moreover, FaDu-pFLAG-*TWIST1* cancer cell populations that were induced to EMT displayed an increased proportion of cells with the CD44 marker, which is associated with tumour initiation. Interestingly, we found that emodin treatment reduced the tumour-initiating abilities and inhibited cell migration and invasion in FaDu-pFLAG-*TWIST1* cells. Emodin directly inhibited TWIST1 expression, upregulated E-cadherin mRNA and protein expression, and downregulated vimentin mRNA and protein expression. Moreover, we found that emodin inhibited TWIST1 binding to the *E-cadherin* promoter and repressed *E-cadherin* transcription activity. We also found that emodin inhibited *TWIST1*-induced EMT by inhibiting the β-catenin and Akt pathways. Moreinterestingly, emodin significantly inhibited *TWIST1*-induced invasion *in vivo*. Therefore, emodin might be applicable to anticancer therapy and could be a potential new therapeutic drug for HNSCC.

O48. **Garlic essential oil and its active compound attenuate high fat diet-induced lipid accumulation in C57BL/6J mice**

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Garlic (*Allium sativum* L.) is a traditional edible food used to treat obesity, hyperlipidemia, hypertension and cancer. All features of hepatoprotective effect and related molecular mechanisms of garlic essential oil (GEO) and its active compounds on high fat diet induced nonalcoholic fatty liver disease (NAFLD) are lacking. The present study investigated the hepatoprotective mechanisms of GEO and its major active compound (diallyl disulfide, DADS) in a high fat diet induced NAFLD mice model. We found that GEO and DADS significantly attenuated the increased level of hepatic triglyceride accumulation. Furthermore, GEO and DADS treatments also inhibited the expressions of SREBP-1c, ACC and FAS, which are the major biosynthetic enzymes for fatty acid synthesis in the liver. In contrast, the lipolytic enzyme expression of PPARα was increased. These results suggested that GEO had the potential to reduced lipid accumulation by suppresseing fatty acid synthesis and stimulating fatty acid oxidation. The protective effects may be associated with its major compound DADS.