B5 Regulation of epithelial mesenchymal transition (EMT) by *Serum Amyloid A 1* (*SAA1*) is dependent on integrin in esophageal squamous cell carcinoma (ESCC)

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Esophageal Squamous Cell Carcinoma (ESCC) is the predominant type comprising more than 90% of esophageal cancer, which is a highly metastatic and fatal cancer, and is ranked the eighth in mortality rate in Hong Kong cancer patients (Hong Kong Cancer Registry, Hospital Authority, 2010). Using a functional complementation approach, *SAA1* was identified as one of the tumor suppressor gene candidates. The *SAA1* is an acute phase protein, which is highly expressed in response to inflammation by the liver. It is also present as a secretary protein in histologically normal human epithelial tissues. The expression of *SAA1* was found to be down-regulated in ESCC. Interestingly, the gene expression of *SAA1* and the mesenchymal marker N-cadherin was found to be inversely correlated in a panel of ESCC and the immortalized esophageal epithelial cell lines. Therefore, we want to determine whether *SAA1* could regulate EMT in ESCC.

The mesenchymal markers such as N-cadherin and fibronectin were significantly up-regulated when using *SAA1* knockdown assays by both lentiCRISPR and pLKO shRNA systems in the immortalized esophageal cell lines. At the same time, the expression of another group of cell adhesion molecules, integrin alphaV was found to be concomitantly up-regulated. To elucidate the role of integrin alphaV in regulating EMT-associated genes in ESCC, the knockdown assay of integrin alphaV in ESCC cell line was performed and showed that the expression of mesenchymal markers N-cadherin, fibronectin, and vimentin and EMT-activating transcription factors Twist and Slug, were down-regulated. As a summary, the down-regulation of *SAA1* in ESCC could lead to the up-regulation of integrin and its subsequent induction of EMT. Further investigation will be taken to understand how the regulation of EMT *SAA1* would affect metastasis in ESCC.