ACTN4 promotes pancreatic cancer metastasis by enhancing the focal adhesion dynamic

Speaker : Qin-Ling Hong
Advisor : Ching-Chieh Weng
Date :2022/9/16

儘管在診斷和外科藥物治療方面上取得了相當大的進展，但胰腺癌的高度致死率仍然影響了病患的存活。α-Actinin 4 (ACTN4) 是一種肌動蛋白結合蛋白，屬於 Spectrin 家族成員，在許多的癌症類型中均被認為是致癌基因。 儘管 ACTN4 已經被發現參與子宮頸癌的致腫瘤性及影響上皮-間質轉化，但 ACTN4 在胰腺癌中的作用卻仍然未知。在我們初步的成果中發現在胰腺癌小鼠模型中 ACTN4 表現會隨著胰腺癌的發展過程而增加。並且發現 ACTN4 剔除會降低胰腺癌細胞的增殖及細胞爬行能力。在這項研究中我們將證明 ACTN4 基因如何通過點狀黏著促進胰腺癌 (PDAC)的轉移能力。

Pancreatic Adenocarcinoma (PDAC) is still associated with high mortality and morbidity for affected patients notwithstanding considerable progress in diagnosis and surgical pharmacological therapy. α-Actinin-4 (ACTN4) is an actin-binding protein belonging to the Spectrin gene superfamily that acts as an oncogene in various cancer types. Although ACTN4 is involved in cervical cancer tumorigenesis and epithelial-mesenchymal transition (EMT), the role of ACTN4 in pancreatic cancer remains unknown. In our preliminary data, we found that ACTN4 expression levels were increased during the pancreatic cancer progression in the PDAC mouse model. ACTN4 knockdown leads to reduce proliferation and motility of pancreatic cancer cells. This study will demonstrate how the ACTN4 gene promotes metastasis in pancreatic ductal adenocarcinoma (PDAC) through focal adhesion.