中文題目：IL-17A加速間質幹細胞所誘發的黑色素瘤細胞的移動性

英文題目：IL-17A enhances mesenchymal stromal cells (MSCs)-induced cell motility in melanoma cells

作　　者：王耀廣[1]，劉玉森[1]，劉忠榮[1,2]，王聖雯[1]，林忠成[1,3]，盧建宇[1]

服務單位：高雄醫學大學附設醫院 胃腸內科[1] 癌症中心[2]
行政院署立屏東醫院 內科[3]

Background/Aim: It is becoming increasingly evident that Tumor Microenvironment such as mesenchymal stromal cells (MSCs), Th17 cell associated with tumors significantly contribute to tumorigenesis such as interact with cancer cells migration and proliferation in tumor tissues.

Methods: We applied the Transwell migration assay from coculture with to mesenchymal stromal cells (MSC) successfully evaluated the migration of B16F10 cell and matrigel invasion assay has been used to observed the expression of invasion by DAPI stain. We measured the B16F10 cell survival by MTT assay; and observed the secretion of coculture with MSC and B16F10 cell by ELISA array. We also used MMP3 inhibitor to indentify the molecular mechanism behind B16F10 cell migration.

Results: In this study, we showed that MSCs increase proliferation of B16F10 murine melanoma cells and promoter B16F10 cell migration Our data show that B16F10 migration ability , but not invasion by mesenchymal stem cell can be upregulated by IL-17A , a cytokine produced by Th17 cells. Furthermore, throughout this process maybe upregulation of MMP3，TIMP1， PAI1，MCP1，and inhibition of OPN1. among these angiogenesis proteins,MMP3 influence this result most greatly.

Conclusion: Our data show that IL-17A can promote B16F10 migration ability via MMP-3 secretion by B16F10 murine melanoma cells and MMP-3 inhibitor can effectively reduce the ability.