

Supplementary Materials: In Vitro and in Vivo Antifibrotic Effects of Fraxetin on Renal Interstitial Fibrosis via the ERK Signaling Pathway

Yi-Hsien Hsieh, Tung-Wei Hung, Yong-Syuan Chen, Yi-Ning Huang, Hui-Ling Chiou, Chun-Wen Cheng, Chu-Che Lee and Jen-Pi Tsai

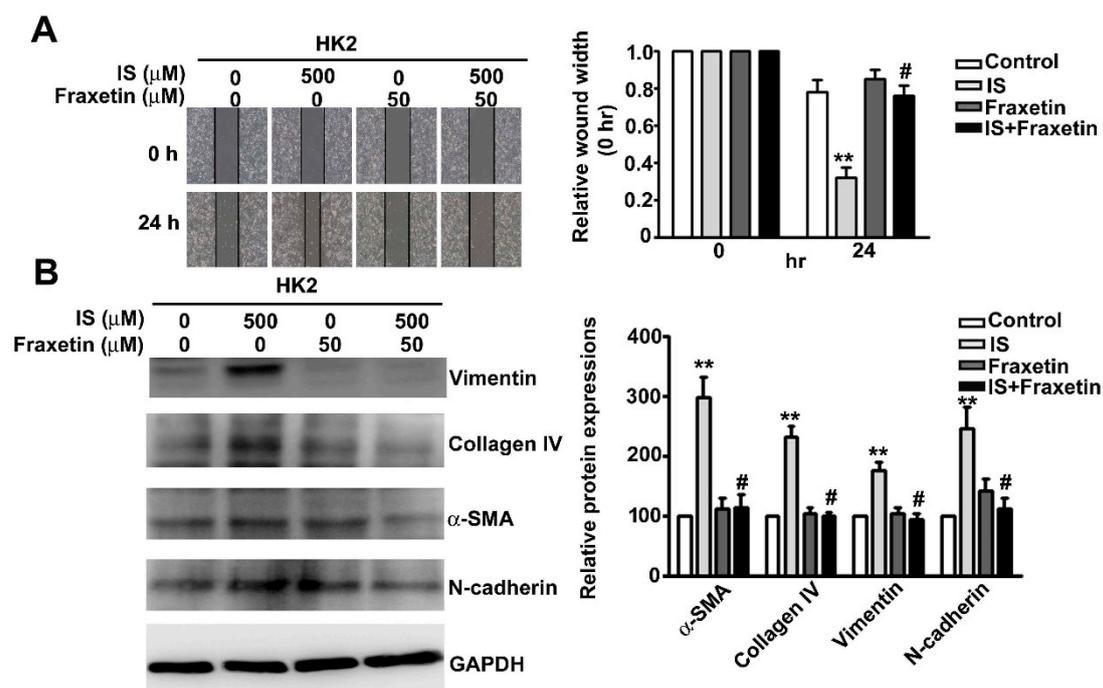


Figure S1. Fraxetin inhibit IS-induced cell motility and fibrosis/EMT-related protein expression in HK2 cells. (A) HK2 cells were pre-treated with fraxetin (50 μM) for 2 h and then treated with IS (500 μM) for 24 h. Cell motility was determined by wound healing assay. Cells were photographed under a light microscope at 400 \times magnification. (B) Total cell lysates were analyzed by western blot to determine the expression of vimentin, collagen IV, α -SMA, and N-cadherin. GAPDH was used as an internal control for protein loading. Data are presented as the mean \pm SE of at least three independent experiments. ** $p < 0.01$ vs. control cells; # $p < 0.01$ vs. IS-treated cells.

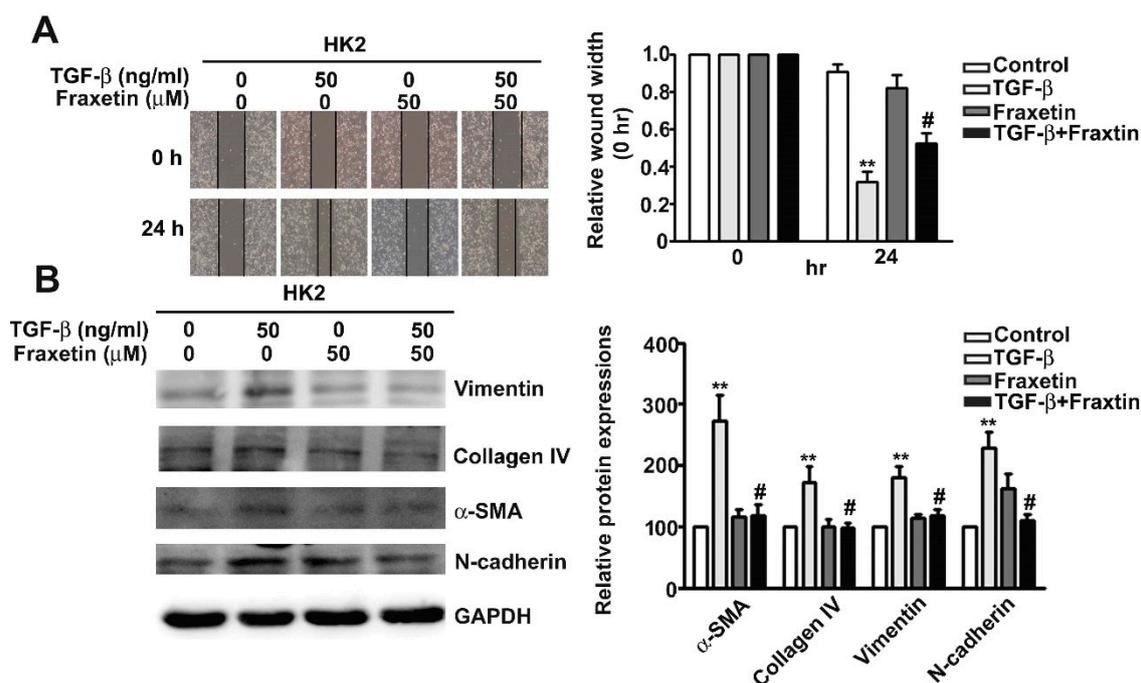


Figure S2. Fraxetin inhibit TGF- β -induced cell motility and fibrosis/EMT-related protein expression in HK2 cells. (A) HK2 cells were treated fraxetin (50 μ M) for 2 h, then with or without TGF- β (50 ng/ml) treatment. Cell motility were showed by using the wound healing assay and photographed under a light microscope at 400 \times magnification. (B) The expression of vimentin, collagen IV, α -SMA, and N-cadherin were detected by western blotting. GAPDH was used as an internal control for protein loading. Data are presented as the mean \pm SE of at least three independent experiments. ** $p < 0.01$ vs. control cells; # $p < 0.01$ vs. TGF- β -treated cells.