CCN5 negatively regulates TGF-β-induced endometriosis associated fibrosis through Wnt/β-catenin signaling via Smad3-dependent mechanism

Mian Liu[1, #], Yi Gong[2, #], Hong Cai[3, #], Rui Hua[1], Hong Li[1], Zhe Wang[1], Yao Zhou[1], Yanlin Ma[4, *], Song Quan[1, *]

[1]Center for Reproductive Medicine, Department of Obstetrics and Gynecology, Nanfang Hospital, Southern Medical University, Guangzhou, China.

[2]Henan Provincial Key Laboratory of Genetic Diseases and Functional Genomics, Medical Genetic Institute of Henan Province, Henan Provincial People's Hospital, People's Hospital of Zhengzhou University, Zhengzhou, Henan, China.

[3]Department of Obstetrics and Gynecology, Shenzhen Hospital of Southern Medical University, Shenzhen, China.

[4]Hainan Provincial Key Laboratory for Human Reproductive Medicine and Genetic Research, Hainan Provincial Clinical Research Center for Thalassemia, Key Laboratory of Reproductive Health Diseases Research and Translation (Hainan Medical University), Ministry of Education, Department of Reproductive Medicine, the First Affiliated Hospital of Hainan Medical University, Hainan Medical University, Haikou, Hainan, China.

*Co-correspondence authors: mayl1990@foxmail.com and QuanSongnffc@163.com.

[#]Theses authors contributed equally to this work.

Figure S1

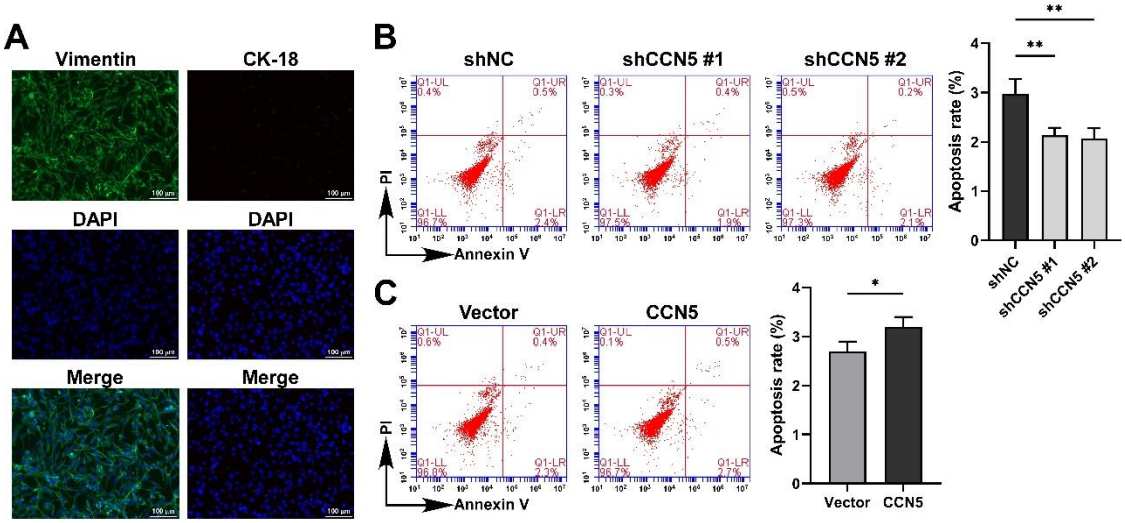

Figure S1. Purity identification of HESCs and the impact of CCN5 on their apoptosis. A. The expression levels of Vimentin and CK-18 in HESCs were assessed through immunofluorescence analysis, yielding positive results. Scale bar represents 100 μm. B, C. Annexin V and propidium iodide (PI) double staining were utilized to evaluate the effects of CCN5 knockdown (B) and overexpression (C) on the apoptosis of HESCs. Data are presented as mean ± standard deviation (SD). Statistical significance is indicated as *$p < 0.05$ and **$p < 0.01$.