Molecular Medicine

Supplemental Data

Activated Protein C Inhibits Proliferation and Tumor Necrosis Factor α–Stimulated Activation of p38, c-Jun NH2-Terminal Kinase (JNK) and Akt in Rheumatoid Synovial Fibroblasts

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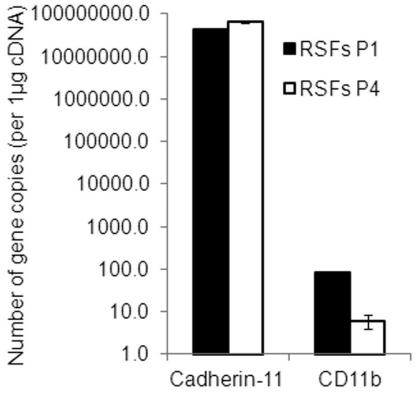
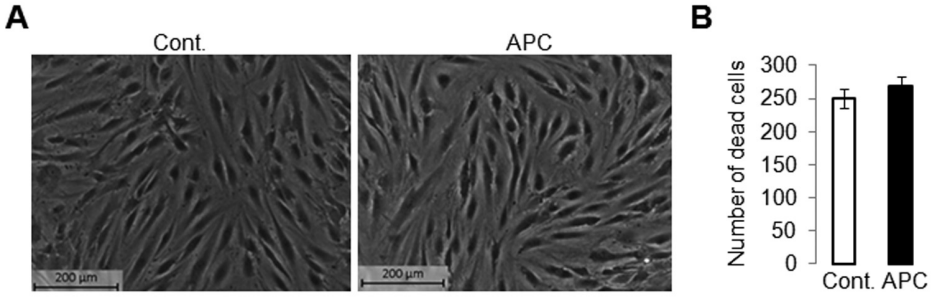
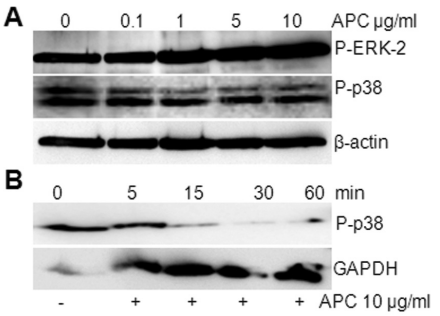


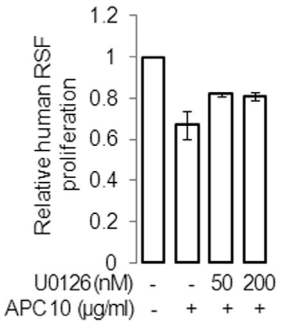
Supplementary Figure S1. Expression of cadherin-11 and CD11b in cultured RSFs by quantitative reverse transcriptase-polymerase chain reaction (qRT-PCR). Passage depicted as P.



Supplementary Figure S2. Similar pattern of RSFs morphology (A) and cell death (B) between control and APC treated groups. Scale bar, 200 µm. Cont., control.



Supplementary Figure S3. Effects of APC on phosphorylated (P) ERK-2 and P-p38. A). RSFs were treated with increasing doses of APC for 30 min and P-ERK-2 and P-p38 were assessed by western blotting. β-actin was used as a loading control. B) APC (10 µg/ml) was added to RSFs for up to 60 min and P-p38 measured by western blotting. GAPDH was used as a loading control.



Supplementary Figure S4. Blockade of antiproliferative effects of APC on RSFs by U0126. RSFs were pre-treated for 1 h without or with U0126 (50 and 200 nM, non toxic to RSFs), then treated with or without APC (10 µg/ml) for 24 h. Proliferation was measured by crystal violet assay. Controls were defined as 1. Values shown are mean ± SD, n=2, 4 wells for each patient in each group.