Figure S1. The inhibition of Cav1.2 subunit of L-type calcium channel by RNAi have the potential inhibitory effects for cellular EMT and osteogenic/chondrogenetic differentiation in PRECs. (A) The relative expression of E-cadherin mRNA significantly increased in a time dependent-manner after Cav1.2 subunit depletion in PRECs. Additionally, relative expression of Zeb1; (B) Col2A1; (C) Runx2; (D) were also detected, and showed the decreasing trend in a time dependent-manner, and reached to the statistical minimum at 48 and 96 h. All data from three independent experiments ($n = 3$) were analyzed, significant difference (* $p < 0.05$) relative to control.
Figure S2. The IP3-mediated store-operated calcium channels (SOCs) have potential roles in EMT process and relevant differentiation in PRECs. The protein expressions of STIM1 and ORAI1 were analyzed by western blot respectively. (A–C) The levels of STIM1/ORAI1 in PRECs were significantly upregulated comparing with those of NRK cells (control) ($n = 3$); (D,E) The expression of E-cadherin in PRECs increased significantly when cells were incubated with 2-APB (100 μmol/L) and SKF96365 (250 μmol/L), the osteogenic/chondrogenetic markers including Runx2 and Sox9 were also inhibited by 2-APB and SKF96365 respectively ($n = 3$), the level of mensenchymal marker Snail1 in PRECs decreased statistically when cells were treated by 2-APB ($n = 3$). In addition, Double immunofluorescence staining showed Orai1 gene silencing attenuated the potential EMT and chondrogenetic differentiation in PRECs, and the dynamic changes of E-cadherin/Zeb1 and Col2A1/Snail1 were shown in (F) (Original magnification $\times 400$). Significant difference ($^* p < 0.05$) relative to control (NRK cells).
Figure S3. Quantitative analysis of RT-PCR showed the mRNA expression of EMT (A,B) and osteochondral (C,D) markers changed significantly with treatment of nifedipine (10 μmol/L) or/and Wnt11 depletion in PRECs. * p < 0.05 vs. the control.