Figure S1. GCIP represses CBP-mediated transcription. (A) Nuclear extracts from RA-derived FLS expressing HA-GCIP were immunoprecipitated with anti-HA antibodies and immunoblotted with anti-CBP antibodies. (B) Reporter activity was induced by cotransfection of either wt or mutant PKA (PKAwt and PKAmut, respectively) in RA-derived FLS. The luciferase activity of cells transfected with empty vector and PKAmut was set to 1. (C) RA-derived FLS were cotransfected with NFKB-Luc and GCIP. After transfection, cells were treated with 10 ng/ml TNF-α. Data were analyzed by performing a Tukey-Kramer post hoc analysis and expressed as mean ± SD. *P<0.05. These experiments were repeated at least three times. GCIP, grap2 cyclin D interacting protein; RA, rheumatoid arthritis; FLS, fibroblast-like synoviocytes; CBP, cAMP-response element-binding protein-binding protein. S1.