Supporting Information

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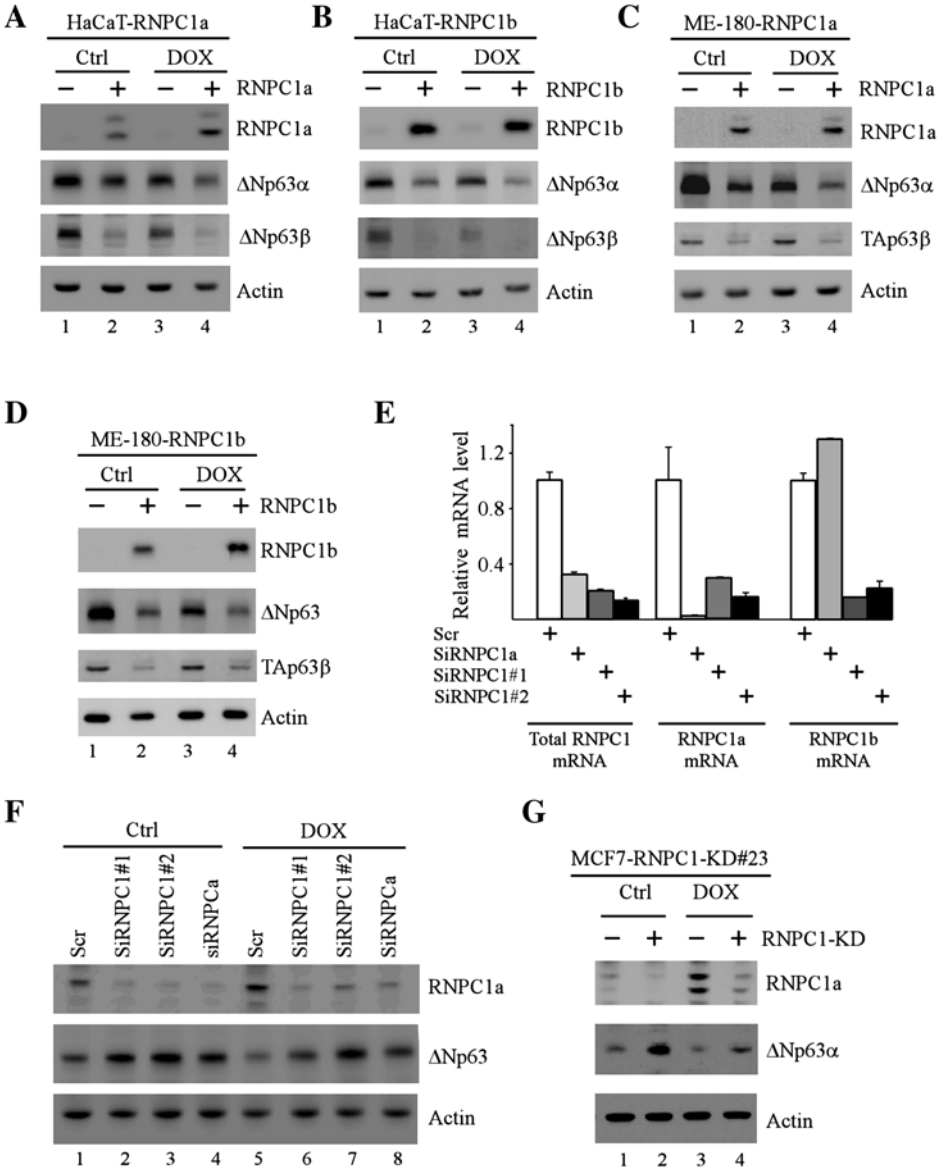

Fig. S1. Overexpression of RNPC1 inhibits, whereas knockdown of RNPC1 increases, p63 expression. (*A* and *B*) HaCaT cells uninduced or induced to express RNPC1a (*A*) or RNPC1b (*B*) for 24 h, followed with or without doxorubicin treatment for 12 h. The level of RNPC1a, RNPC1b, ΔNp63α, ΔNp63β, and actin was measured by Western blot analysis. (*C* and *D*) The experiment was performed as in (*A* and *B*) except that ME-180 cell line was used. (*E*) HaCaT cells were transfected with scrambled siRNA, siRNA against RNPC1a, siRNA#1 or #2 against RNPC1 for 3 d. Total RNAs were purified and used to measure the level of RNPC1a, RNPC1b, and total RNPC1 transcripts by quantitative RT-PCR. (*F*) HaCaT cells transfected with scrambled siRNA or siRNAs against total RNPC1 or RNPC1a for 3 d, followed with or without doxorubicin treatment for 12 h. The level of RNPC1a, ΔNp63α, and actin was measured by Western blot analysis. (*G*) MCF7 cells were uninduced or induced to knock down RNPC1 for 3 d, followed with or without doxorubicin treatment for 12 h. The level of RNPC1a, ΔNp63α, and actin was measured by Western blot analysis.

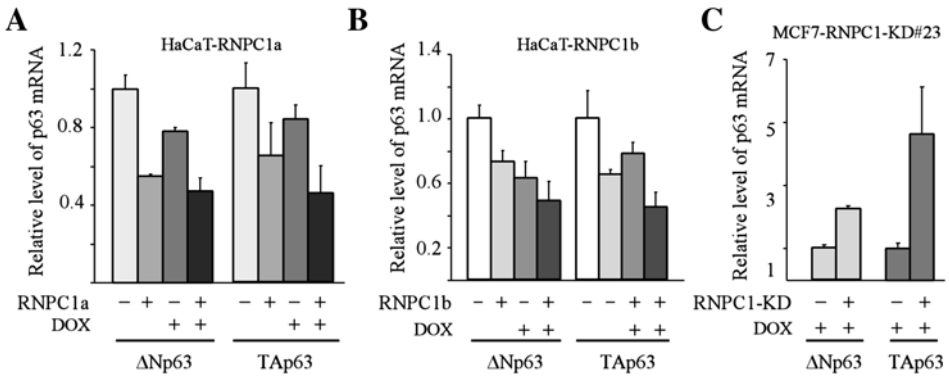

Fig. S2. The steady-state level of p63 transcript is decreased by overexpression, but increased by knockdown, of RNPC1. (*A* and *B*) HaCaT cells were uninduced or induced to express HA-tagged RNPC1a (*A*) or RNPC1b (*B*) for 24 h, followed with or without doxorubicin treatment for 12 h. The level of TA and ΔN p63 transcripts was measured by qRT-PCR. The level of GAPDH transcript was measured as an internal control. (*C*) MCF7 cells were uninduced or induced to knock down RNPC1 for 3 d, followed by doxorubicin treatment for 12 h. The level of TA and ΔN p63 transcripts was measured by qRT-PCR.

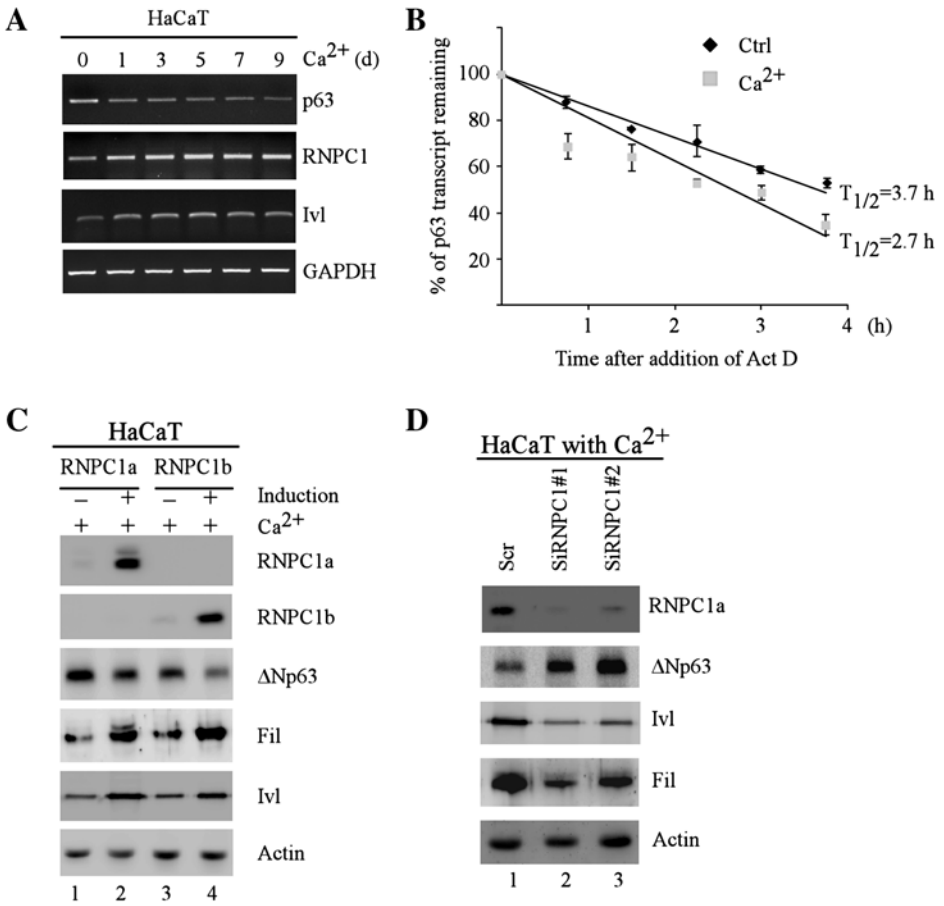

Fig. S3. Terminal differentiation of HaCaT cells by calcium is accompanied with increased expression of RNPC1 and decreased expression of p63. (*A*) HaCaT cells grown at confluence were treated with or without 1.5 mM calcium for 0–9 d. At each time point, total RNAs were isolated from HaCaT cells and analyzed for the indicated gene expression by semiquantitative RT–PCR. GAPDH was measured as an internal control. (*B*) HaCaT cells were treated with or without calcium for 3 d, followed by treatment with actinomycin D for various times. The level of p63 transcript was measured by qRT-PCR. (*C*) Confluent HaCaT cells were uninduced or induced to express RNPC1a or RNPC1b for 24 h, followed by treatment of 1.5 mM calcium for 9 d. The level of RNPC1a, RNPC1b, ΔNp63, involucrin, filaggrin, and actin was determined by Western blot analysis. (*D*) HaCaT cells were transfected with scramble siRNA or siRNA against RNPC1 for 3 d, followed by treatment of 1.5 mM calcium for 11 d. The level of RNPC1a, ΔNp63, involucrin, filaggrin, and actin was determined by Western blot analysis.