Supplemental Information

Supplementary Figures

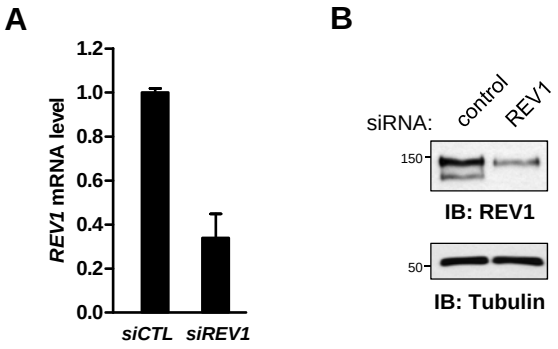

Figure S1. Confirmation of siRNA-mediated depletion of endogenous REV1. MCF7 cells were transfected with non-targeting control or *REV1* siRNAs. 48 h following siRNA transfection, cell extracts were prepared and analyzed by qRT-PCR (A) and Western blot (B).

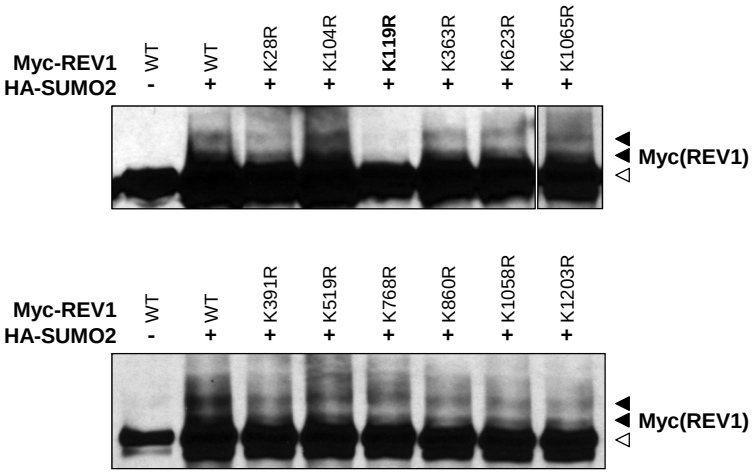

Figure S2. Mouse REV1 protein is SUMO2-modified at the K119 residue *in vivo*. HEK293 cells were co-transfected with wild-type or mutated Myc-REV1 and HA-SUMO2, and cell extracts were immunoblotted with anti-Myc antibody. Arrowheads indicate the SUMOylated form (filled) and the unmodified form (open).

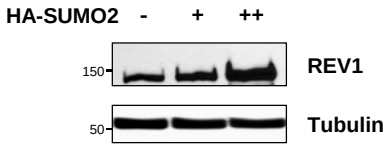

Figure S3. SUMOylation increases the stability of endogenous REV1. B16 cells were transfected with different amounts of HA-SUMO2 expressing vectors for 18 h, lysed and immunoblotted with anti-REV1 antibody. A tubulin blot was presented as a loading control.

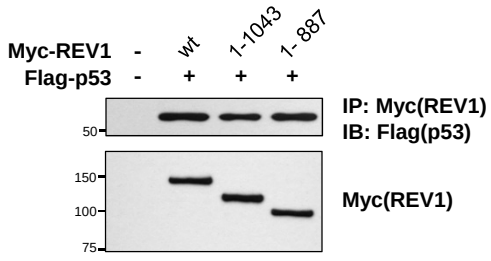

Figure S4. Pull-down assays of the interaction between p53 and deletion mutants of REV1. Myc-tagged full-length or deletion mutants (residues 1-1043 and 1-887) of REV1 were co-transfected with Flag-tagged full-length p53 in HEK293 cells. 24 h after transfection, cell lysates were immunoprecipitated with anti-Myc-agarose beads. The immune complexes were subjected to immunoblot with the indicated antibodies.

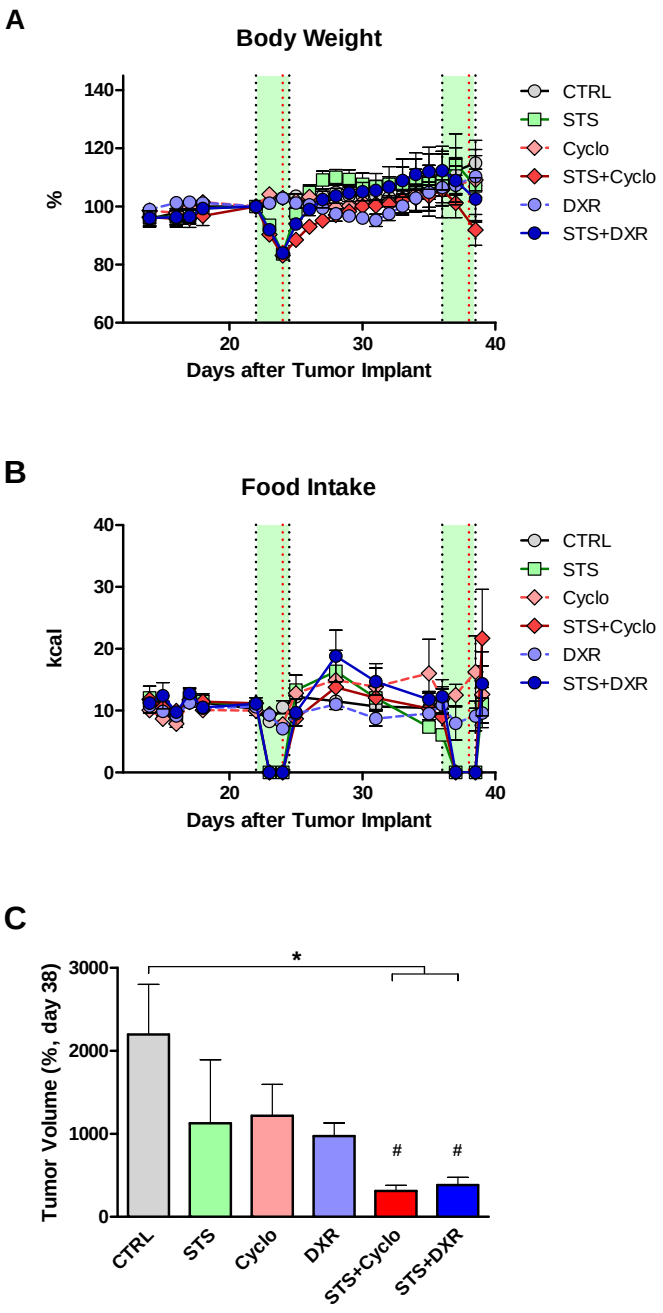

Figure S5. Effect of fasting and chemotreatment on body weight, food intake, and tumor volume in allografted mouse model. (A, B) Effect of fasting on body weight (A) and food intake values (B). Body weight and food intake of tumor-bearing mice were determined periodically, and points and bars represent the mean ±S.E.M (n = 5). (C) Tumor volumes on day 38 were measured. Data shown are the mean +/- S.E.M. *, $P < 0.05$, ANOVA with Tukey's multiple comparison test; $^{\#}$, $P < 0.05$, t test, two tailed, compared to chemotreatment alone.

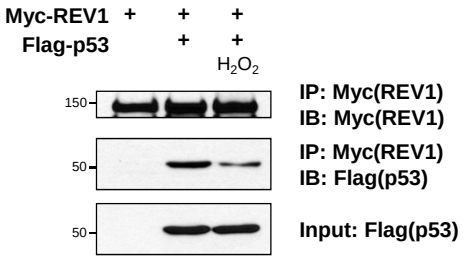

Figure S6. H_2O_2 treatment disrupts REV1-p53 interaction. The cells co-transfected with Flag-p53 and Myc-REV1 expression vectors were treated with H_2O_2 (200 μM) for 30 min. The whole cell lysates were subjected to Co-IP assay.

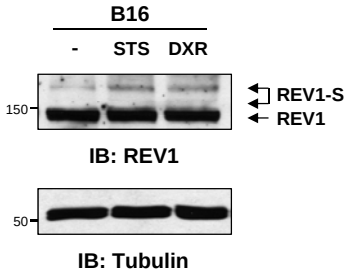

Figure S7. REV1 SUMOylation upon starvation and chemotreatment. B16 cells were incubated with starvation medium or treated with doxorubicin (1 μM) for 24 h, lysed and immunoblotted with anti-REV1 antibody. A tubulin blot was presented as a loading control.

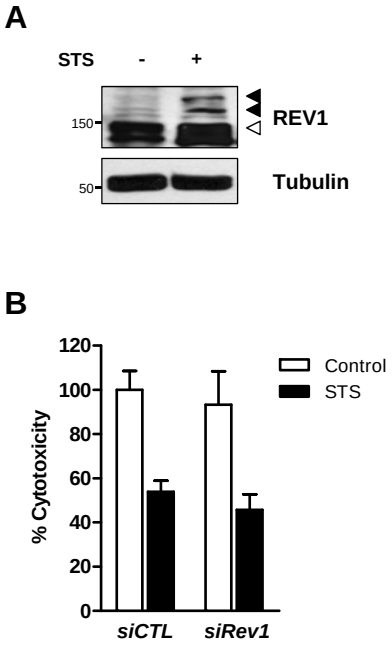

Figure S8. The effect of REV1 SUMOylation on normal cells. (A) Primary MEFs were treated with starvation medium for 24 h, lysed and immunoblotted with anti-REV1 antibody. A tubulin blot was presented as a loading control. (B) The cytotoxicity of primary MEFs after exposure to DXR treatments. Primary MEFs were transfected with *siCTL* or *siRev1*. 24 h following siRNA transfection, cells were incubated in normal or starvation conditions for additional 24 h. Cytotoxicity were analyzed 24 h treatment of 10 μM DXR. Data was plotted as the percentage of *siCTL*-transfected cells treated with DXR. Results represent mean ±SD from three independent experiments.

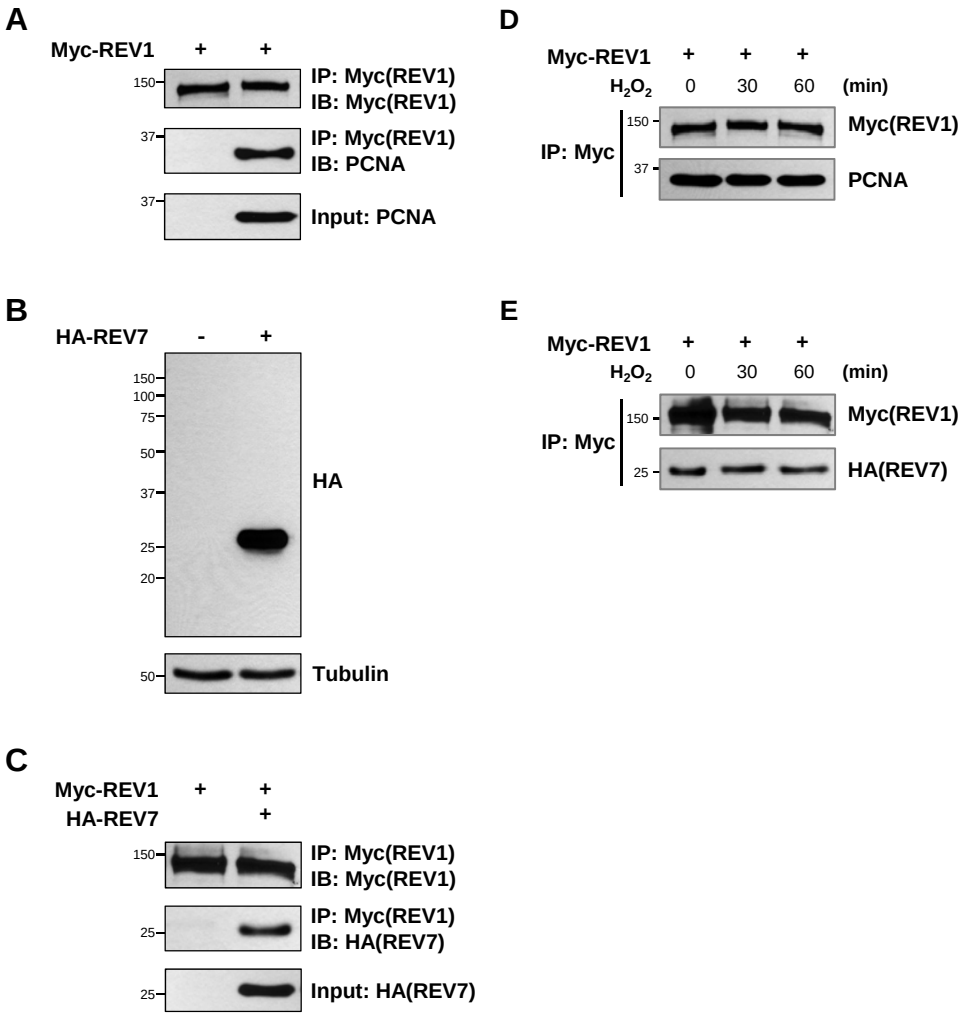

Figure S9. REV1 associates with PCNA and REV7, an accessory subunit of Polζ. (A) Myc-REV1 was transiently expressed in HEK293 cells, and cell lysates were immunoprecipitated with anti-Myc antibody, and the immune complex were analyzed by immunoblotting with anti-Myc and anti-PCNA antibodies. Whole cell lysates were immunoblotted for PCNA as input control. (B) Mouse REV7 tagged with HA epitope was expressed in HEK293 cells and immunoblot analysis using anti-HA antibody confirmed the expression of HA-REV7 construct. (C) Cells were co-transfected with Myc-REV1 and HA-REV7, and cell lysates were immunoprecipitated with anti-Myc antibody, followed by Western blot with anti-Myc and anti-HA antibodies. (D, E) Cells transiently transfected with Myc-REV1 and/or HA-REV7 were treated with H_2O_2 (200 μM) for the indicated time and immunoprecipitated with anti-Myc antibody. The immune complex were analyzed by immunoblotting with anti-Myc, anti-PCNA, and anti-HA antibodies.