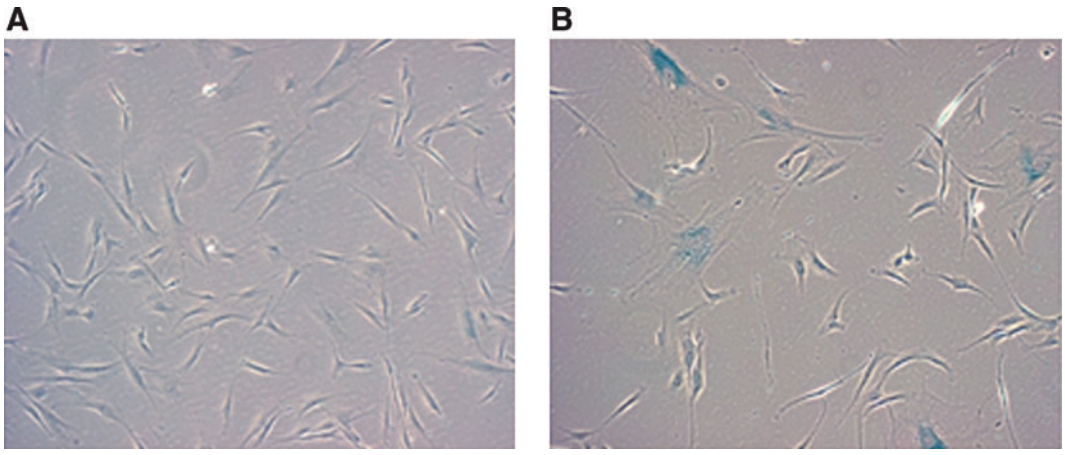
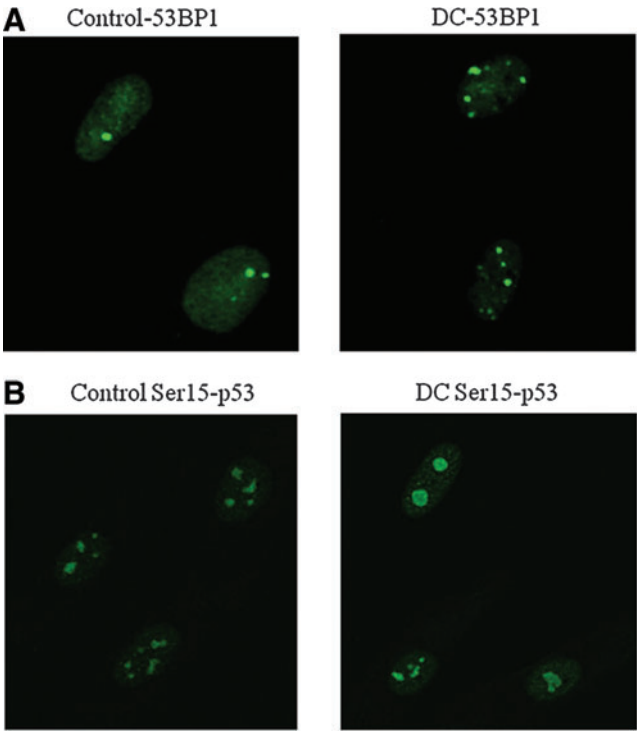
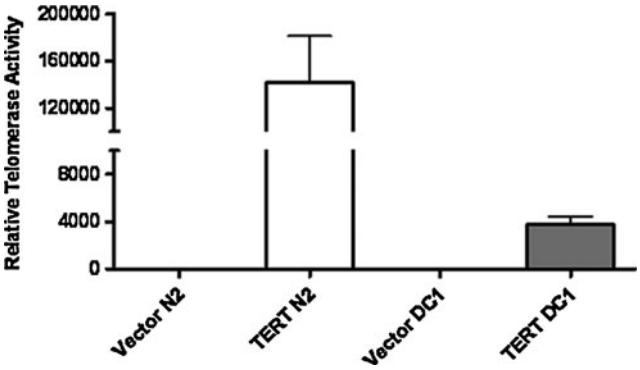
Supplementary Data



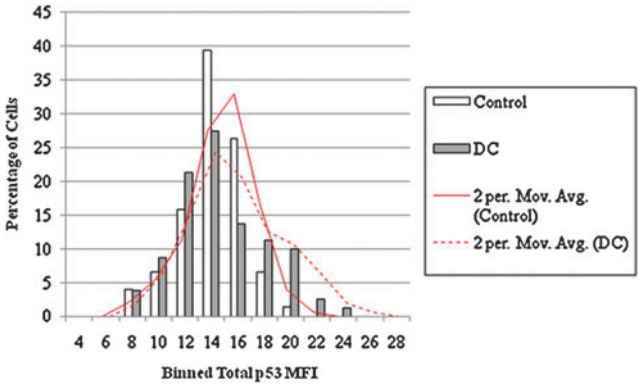
SUPPLEMENTARY FIG. S1. Representative micrographs of dyskeratosis congenita (DC)-1 and normal control cells stained for senescence-associated β-galactosidase (β-gal). (A, B) The assay was performed as described in the Materials and Methods section. Shown are micrographs taken at 100×. Actual counts were made from micrographs taken at 40×.



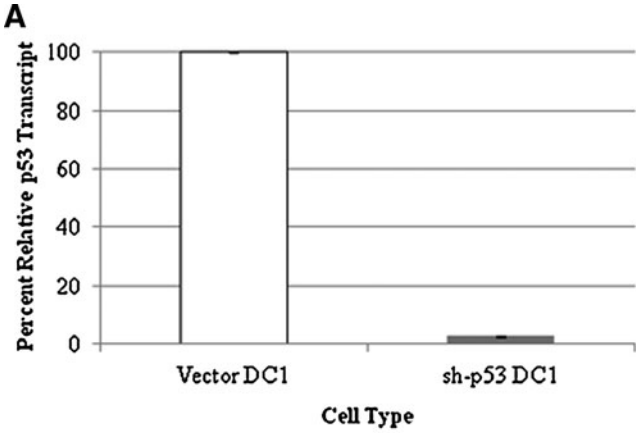
SUPPLEMENTARY FIG. S2. Representative immunofluorescence images of p53 binding protein 1 (53BP1) foci and phospho-ser15 p53 levels in DC and normal control fibrobasts. Assays were performed as described in the Materials and Methods section. **(A)** 53BP1 foci, **(B)** phospho-ser15 p53 levels.



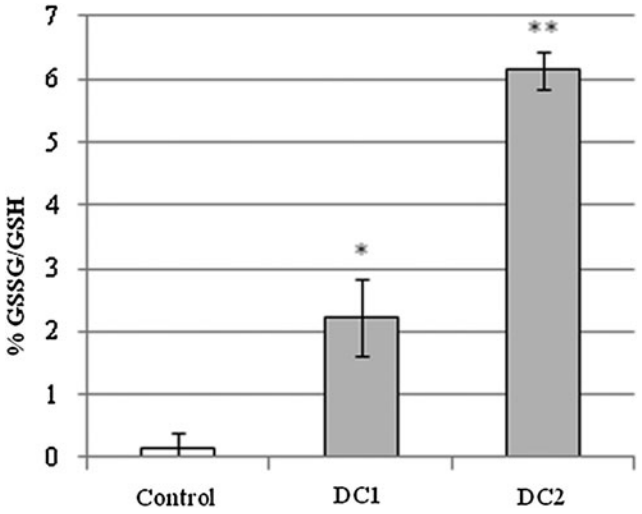
SUPPLEMENTARY FIG. S3. Telomerase activity in telomerase reverse transcriptase (TERT)-expressing DC-1 and normal control cells. The telomerase assay was performed as described in the Materials and Methods section using equivalent cell numbers. Telomerase was barely detectable in vector control DC and normal fibroblasts.



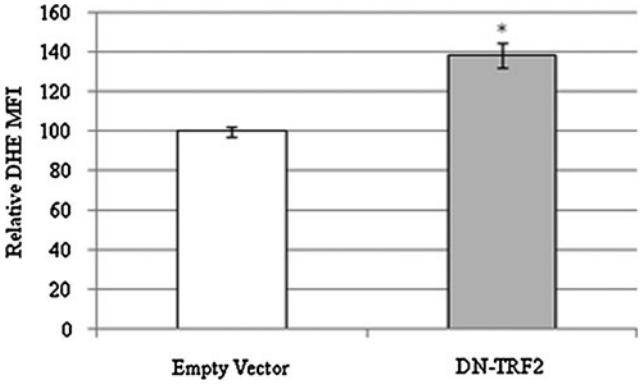
SUPPLEMENTARY FIG. S4. Similar total p53 levels in DC and control cells. Passage 8 DC-1 and control normal cells were grown under routine conditions, fixed, and stained with an antibody assessing total p53 levels. Images were captured using Zeiss 510 multiphoton confocal microscope and the intensity of nuclear signals was quantified using ImageJ and binned. *p*-Values were generated based on the Student's *t*-test ($p < 0.23$).



SUPPLEMENTARY FIG. S5. Downregulation of p53 and protein 21 wild-type p53 activation factor/Cdk-interacting protein by short-hairpin RNA. Cells were transduced with retroviral vectors expressing short-hairpin RNA to **(A)** p53 or **(B)** p21 or a scrambled vector control, and QRT-PCR was performed as described in the Materials and Methods section.



SUPPLEMENTARY FIG. S6. Oxidative stress in DC cells as determined by glutathione disulfide (GSSG)/glutathione (GSH). Passage 8 DC-1 and control normal fibroblast samples were analyzed for glutathione disulfide and GSH to determine the percent of total GSH that was in the disulfide state (%GSSG). Statistical significance based on the Student's *t*-test ($*p < 0.05$; $**p < 0.001$).



SUPPLEMENTARY FIG. S7. Dysfunctional telomeres *via* expression of dominant negative telomere repeat binding factor 2 (TRF2) leads to increased dihydroethidium (DHE) oxidation. Passage 6 normal cells were infected with a retroviral vector expressing a dominant-negative form of TRF2 (DN-TRF) as well as an antibiotic resistance gene. Post-infection, the cells were selected for a pure population and analyzed *via* DHE-FACS. Statistical significance was assessed *via* the Student's *t*-test. $*p < 0.005$.