Online supplemental material

FIGURE S1. **Characterization of hTERT-immortalized HFFs.** A, Immuno-blotting. The top panel shows the expected expression of Myc epitope-tagged ectopic GpIbα in the GpIbα and shp53+ GpIbα cell lines (37). The second panel shows the expected up-regulation of p53 in GpIbα but not in shp53+GpIbα cells in response to ongoing DNA damage as previously described (37). The third panel shows that $p21^{WAF1/CIP1}$ levels mirror those of p53. The lower panel is a β-tubulin control for protein loading. B, Failure to up-regulate p53 and $p21^{WAF1/CIP1}$ in response to adriamycin in HFF cells expressing shp53. Each of the cell lines shown in (A) was exposed to 25 ng/ml adriamycin for 16 hr and then immuno-blotted for p53 and $p21^{WAF1/CIP1}$ as previously described (37). Note the repeat failure to up-regulate p53 in both cell lines expressing shp53. C, Equivalent growth rates for HFF lines. Each of the lines was plated into 12 well plates at 2 x 10^3 cells/well. At the indicated times thereafter, total viable cell counts were performed on triplicate wells as previously described (37). Bars= 1 standard error (SE).

FIGURE S2. **Differential protein expression in hTERT-immortalized shp53 cells versus shp53+GpIbα cells.** The indicated cell lines were lysed and the proteins were fluorescently labeled in vitro according to protocols supplied by the Kinexus Bioinformatics Corp (http://www.kinexus.ca/services/kinex/antibody_microarrays.php). Equivalent amounts of each lysate were then used to probe identical membranes containing >650 antibodies directed against a variety of human proteins. After this initial screen, we confirmed the differential expression of 15 of the proteins by quantitative western blotting of total cell lysates. Note specifically the 2.57-fold down-regulation of Aurora B in response to enforced GpIbα expression.

FIGURE S3. **Additional images of shp53+GpIbα and shp53+GpIbα+Aurora B cells demonstrating various numerical and structural abnormalities of nuclei in the former cells and their**

normalizaton in the latter cells. Arrows indicate dysmorphic nuclei (panels 1,4 and 5), multiple nuclei (panel 3), and micro-nuclei (panels 2&4)

FIGURE S4. **Karyotypes of shp53 and shp53+GpIbα cells**.

A-C, Examples of typical HFF-shp53 cells demonstrating low incidence of non-clonal abnormalities.

Karyotype: 41~46,XY[12]/43~45,XY,+mar[2]/85,XXYY,+mar[1]

Results: Twenty G-banded metaphase cells were analyzed from the cytogenetic harvests of this specimen. Twelve of these cells had a normal male karyotype with a diploid or random hypodiploid chromosome complement. The remaining eight cells had numerical and or structural abnormalities. The only clonal aberration is a small unidentifiable marker chromosome seen in two diploid cells and one copy in a near-tetraploid cell. Three cells had a near-tetraploid chromosome number. Of the near-tetraploid cells, one cell had the marker chromosome mentioned above and additional chromatin material of unknown origin attached to two copies of chromosome 1, one of had five copies of chromosome 9 and loss of one copy of chromosome 13, and the other near-tetraploid cell had random loss of several chromosomes. The other three near-diploid cells had non-clonal abnormalities. One cell had a deletion of the distal long arm of chromosome 10, another had a deletion of the long arm of chromosome 20, and the third cell had an interstitial deletion of the long arms of chromosomes 6 and 13.

D-F, Examples of typical HFF-GpIbα+shp53 cells demonstrating a high incidence of non-clonal abnormalities.

Results: Sixteen G-banded metaphase cells were analyzed from the cytogenetic harvests of this specimen. Ten of these had a hypodiploid to near-triploid chromosome number ranging from 34 to 63. The remaining six cells had a hypotetraploid to near-tetraploid chromosome number ranging from 82 to 92. Four of these cells were completely analyzed. All four of these cells showed both numerical and

complex structural abnormalities, which differ from cell to cell. No two cells were identical. The remaining twelve metaphase cells observed could not be karyotyped, but also contained a wide range in chromosome number. All cells also appear to have multiple structural rearrangements that differ from cell to cell. The cells observed in this sample have a greater variability in chromosome number and structural aberrations compared to HFF-shp53 cells.

FIGURE. S5. **Additional images of cleavage furrow-localization or mis-localization of GpIbα, actin, filamin, RhoA, and Aurora B in shp53+GpIbα cells.** shp53 cells are again included as controls.