



Bradley A. Stohr Supp. Figure 2











Supplementary Fig. 1. MT-hTer-47A treatment activates ATM. LOX melanoma cells were infected with lentivirus expressing scramble #1 or ATM #2 shRNAs on day -2. The cells were subsequently infected with lentivirus expressing WT-hTER or MT-hTer-47A on day 0. Cells were harvested on day 6, and protein lysates were analyzed by Western blot. Samples were probed for ATM or phosphorylated ATM (pS1981 ATM), with both actin and nonspecific (NS) bands serving as loading controls.

Supplementary Fig. 2. WT-hTER overexpression does not significantly affect LOX cell growth. At day 0, LOX cells were mock infected (circles) or infected with lentivirus expressing GFP alone (squares) or GFP plus WT-hTER (triangles) or MT-hTer-47A (diamonds). Cells were counted at indicated time points by hemocytometer. Error bars indicate the standard deviation from three separate infections.

Supplementary Fig. 3. ATM depletion does not block hTER overexpression. LOX melanoma cells were infected with lentivirus expressing scramble #1 or ATM #2 shRNAs at day -2, followed by lentivirus expressing WT-hTER or MT-hTer-47A at day 0. Cells were harvested at day 4 and day 6, and total RNA was isolated. The hTER expression levels were determined by real-time RT-PCR. Error bars indicate the standard deviation for three independent experiments. Because the magnitude of hTER overexpression varied from experiment to experiment, the data within each experimental time point were normalized by dividing each data point by the mean of all four data points. The difference in MT-hTer-47A expression between the scramble and ATM #2 shRNA treated groups at day 6 is statistically significant (p = 0.01).

Supplementary Fig. 4. Short-term ATM depletion does not affect UM-UC-3 telomere length. UM-UC-3 cells were mock infected or infected with lentivirus expressing scramble #1 or ATM #2 shRNAs. Cells were harvested ten days later, genomic DNA was isolated, and Southern blotting for wild-type telomeric DNA was performed.

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