Aging and chronic DNA damage response activate a regulatory pathway involving miR-29 and p53

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Supplementary information

Supplementary Figure 1. (A) miR-29 expression analysis by qPCR of two independent p53\^-/- fibroblast cell lines at passages 2 and 8, and of mouse ATM\^-/- and ATR\^-/- fibroblasts treated with two different concentration of doxorubicin. (n=2 biological replicates). *Significantly different from untreated cells, p<0.05. (B) Table showing the mRNA levels of validated miR-29 target genes in whole transcriptome analysis of Zmpste24\^-/- muscle samples. Data represent the normalized fold change in deficient mice. (C) qPCR analysis of Narf, Ppm1d and Hbp1 mRNA levels in muscle from wild-type and mutant mice (n = 6 mice of each genotype). * Significantly different from wild-type mice, p<0.05.

Supplementary Figure 2. (A) Wild type (left panel) or Zmpste24\^-/- (right panel) primary mouse fibroblasts were seeded in 96-well plates and transfected with miR-29 or control precursor or inhibitor molecules, respectively. Cell proliferation was assessed by MTT assay at different time points (n = 3 biological replicates). (B) MTT assay of U2OS over-expressing miR-29. U2OS cells transduced with a lentiviral vector expressing the miR-29b-2\/-29c cluster were seeded in 96-well plates and transfected with control or miR-29 inhibitor molecules and cell proliferation was assessed by MTT assay at different time points (n = 3 biological replicates). * significantly different from empty vector transduced cells, p-value < 0.05. # significantly different from control transfected cells, p-value < 0.05.
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