CORRIGENDUM

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Telomere dysfunction induces metabolic and mitochondrial compromise

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In this Article, references[1–3] reporting an association between telomere dysfunction and mitochondrial impairment or TERT deficiency and mitochondrial impairment were inadvertently omitted.

1. Passos, J. F. *et al.* Feedback between p21 and reactive oxygen production is necessary for cell senescence. *Mol. Syst. Biol.* **6,** 347, doi:10.1038/msb.2010.5 (2010).
2. Haendeler, J. *et al.* Mitochondrial telomerase reverse transcriptase binds to and protects mitochondrial DNA and function from damage. *Arterioscler. Thromb. Vasc. Biol.* **29,** 929–935 (2009).
3. Kovalenko, O. A. *et al.* A mutant telomerase defective in nuclear-cytoplasmic shuttling fails to immortalize cells and is associated with mitochondrial dysfunction. *Aging Cell* **9,** 203–219 (2010).