

Abstract

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Nicotinamide extends replicative lifespan of human cells.

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BACKGROUND: We found that an ongoing application of nicotinamide to normal human fibroblasts not only attenuated expression of the aging phenotype but also increased their replicative lifespan, causing a greater than 1.6-fold increase in the number of population doublings.

RESULTS: Although nicotinamide by itself does not act as an antioxidant, the cells cultured in the presence of nicotinamide exhibited reduced levels of reactive oxygen species (ROS) and oxidative damage products associated with cellular senescence, and a decelerated telomere shortening rate without a detectable increase in telomerase activity. Furthermore, in the treated cells growing beyond the original Hayflick limit, the levels of p53, p21WAF1, and phospho-Rb proteins were similar to those in actively proliferating cells. The nicotinamide treatment caused a decrease in ATP levels, which was stably maintained until the delayed senescence point. Nicotinamide-treated cells also maintained high mitochondrial membrane potential but a lower respiration rate and superoxide anion level.

CONCLUSION: Taken together, in contrast to its demonstrated pro-aging effect in yeast, nicotinamide extends the lifespan of human fibroblasts, possibly through reduction in mitochondrial activity and ROS production.

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