S10-5 Analysis of aging-related oxidative stress status in normal aging animals and development of anti-aging interventions

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Since Harman proposed the “free-radical theory of aging” in 1956, oxidative stress is postulated to be a major causal factor of senescence. Accumulation of oxidative stress-induced oxidatively modified macromolecules including protein, DNA, and lipid, were found in tissues during the aging process. However, it is not necessarily clear which factor is more critical an increase in endogenous reactive oxygen and/or decrease in antioxidative defense, to the age-related increase in oxidative damage. To clarify the production of reactive oxygen increasing with age, we examined reactive oxygen-dependent chemiluminescent (CL) signals in ex-vivo brain slices prepared from different aged animal brain during hypoxia-reoxygenation treatment using a novel photonic imaging method. CL signal was intensified during reoxygenation. The signals in SAMP10 (short life strain) and SAMR1 (control) brain slices was increased with aging. The slope of increase of CL intensity with age in P10 was steeper than those in R1. Age-dependent increase of CL intensity was also observed in C57BL/6 mouse, Wistar rat, and pigeon. However, SOD activity in brain was not changed with age. These results suggest that reactive oxygen production itself is increased with aging. The rate of age-related increases of CL intensity was inversely related to the maximum life span of the animals. We speculate that reactive oxygen may be a kind of signal for aging and its levels in tissue may determine the aging process and life span. To decelerate the in age-related increases of reactive oxygen production is expected as a potent strategy for anti-aging interventions.