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Editorial

Note On Aging Reasons

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EDITORIAL NOTE

Aging is due to entropy, the natural phenomenon towards disorders and randomness in any system. Everything ages both non living and living organisms. Isolated systems can achive a local reversal of entropy by using energy from outside the system, but only at the cost of greater chaos in the larger environment. Biological aging is a progressive degradation if function and coordination of internal processes leading to diminution of fitness of the organism. The net outcome of the balance between processed of damage and self repair/regeneration. Aging is heterogenous. Different species age at different rates. Individuals of the same species age at different rates. Systems within an individual age at different rates. Specific theories regarding the aging process include accumulation of damage to cell components like proteins, lipid membranes and DNA by free radicals. Generated by internal metabolic processes, generated by external exposures, opposed by antioxidant enzymes and nutrients. Deterioration of ability to carry out cell replacement and tissue repair, stem cell depletion or inactivation, Immunosenescenece, Telomere shortening leading to cell senescence, Accumulation of senescent cells which produce toxic by-products that have and repair. All of the previously discussed may interact to produce organismal aging including changes in function of the endocrine systems.

Senescent cells and senolysis or cellular senescence with aging in mice, monkeys and humans and interventions that increase lifespan in animals, including caloric restriction and mutations in the growth hormone axis, are associated with decreased senescent cell abundance. Cellular senescence is characterized by irreversible cell cycle arrest accompanied by a senescent associated secretory phenotype like cytokines, tissue damaging proteases and others. Senescent cell anti-apoptotic pathways which make cells resistant to death. All those mechanisms harm activity of local normal cells that display increased senescent cell numbers. Important markers of senescent cells include SASP factors. Senescent cells are associated and with age related pathologies in animal models experimental studies have shown that young milieu is capable of protecting aging tissues form cellular senescence. Senolysis is based on targeting senescent cells to be eliminated by genetic or pharmacological approaches. Senolytc drugs are agents that selectiviely induce apoptosis of senescent cells acting on SCAPS. These agents exhibit a high degree of cell type specificity. Brief disruption of scaps can kill senescent cells. Thus senolytics could theoretical be administered intermittently.

Proof of principle studies have showed that selective depletion or elimination of senescent cells can prevent or delay chronic conditions in animal models as frailty cardiac dysfunction, vascular hyperactivity and calcification, diabetes mellitus, liver steatosis, osteoporosis, vertebral disk regeneration, pulmonary fibrosis and radiation induced damage.

Early phase clinical trials are needed to know if chronic diseases as a group or one at a time might be prevented or delayed as in animal models has been observed. Cautions must be emphazied, particulary until clinical trials are completed and the potential adverse effects of senolytic drugs. Rate of regression is equivalent to the rate of aging and depends on the balance of organic wear and repair. Exogenous factors that favour the production of free radicals such as tobacco products as well as the consumption of peroxides etc. appear to accelerate aging.

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