Can We Increase Our Health Span?

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KEYWORDS

• Aging • Health span • Inflammation • Chronic disease • Lifestyle

KEY POINTS

- The deterioration of physical and mental capabilities is inevitable with aging.
- Even though some hereditary factors cannot be changed, many other external factors can be manipulated to provide our body with better weapons to have a better quality of life as we age.
- Different cellular pathways that lead to cell deterioration and aging usually act through excessive oxidative damage and chronic inflammation.
- Suppression of inflammation is the most important driver of successful longevity and increases in importance with advancing age.
- Lower inflammation is associated with prolonged physical functionality and cognitive abilities.

Aging brings a cascade of ills and health problems leading to the deterioration of physical, mental, emotional, and social dimensions of life.[1] It is influenced by hereditary and environmental factors. The hereditary factors, like physical and mental abilities, will inevitably deteriorate, but the external, environmental factors like level of physical activity, nutrition, and lifestyle can be controlled and regulated.[1] Life expectancy has been increasing and is projected to continue this trend and reach more than 80 years in most industrialized countries by 2030 with some reaching up to 90 years.[2] As important and exciting as this is, an increased lifespan without corresponding quality of life may not be very meaningful. In this article, we review the theories of aging, some of its physiologic processes, and possible ways to increase our health span and decrease morbidity by limiting progression or development of some of the most common risks of aging, including cardiovascular diseases, diabetes, cerebrovascular disorders, cancer, and neurodegenerative disorders.

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THEORIES OF AGING

There are different theories and factors to try to explain aging and its changes, although it is well understood that it is a multifactorial process.[3] Some of these will follow.

1. Wear and tear: compares humans to machines. Accumulation of excessive consumption of fat, sugar, and UV radiation will cause cell damage and death. Disregards the cell capacity for self-repair.
2. Stochastic theory sees aging as the result of inevitable, random, small changes that accumulate over time.
3. Evolutionary theory: Specific genes are implicated in longevity and in maintenance of the cells. These genes are susceptible to mutations with age.
4. Rate of living theory: organisms that metabolize oxygen more rapidly have a higher energy expenditure and shorter lifespan.
5. Oxidative stress theory: most frequently cited lately and possibly the most important one with regard to increasing health span. Age-associated functional losses are due to the accrual of oxidative damage to macromolecules, such as lipids, DNA, proteins by free radicals, and that the progressive oxidant/antioxidant imbalance leads to the consequent disruption of redox-regulated signaling mechanisms.

CAUSES OF AGING

1. Failure of cellular metabolism, decreasing secretion of body hormones, and environmental factors, such as quality of diet, lack of physical activity. Discrepancy between biological damage and the rate of repair leads to accumulation of damage, especially through oxidation.[1]
2. Cell determinants of aging include free radical damage, mitochondrial dysfunction, decreased autophagy, alterations of glucose and cholesterol metabolism, telomere shortening, and increased apoptosis[4]
3. Mitochondria dysfunction.
   - Mitochondria are responsible for most of the useful energy derived from the breakdown of carbohydrates and fatty acids, which are converted to ATP by the process of oxidative phosphorylation.
   - Mitochondria are a major source of reactive oxygen species (ROS) and reactive nitrogen species (RNS) that can regulate mitochondrial activity through different mechanisms, including modulation of O_2 consumption and oxidative phosphorylation, induction of mitochondrial membrane permeability transition, regulation of mitochondrial biogenesis, mitochondrial dynamics, and autophagy/mitophagy.[5]
     - Excessive ROS production is usually compensated by the antioxidant defense system, thus maintaining the redox balance. Cellular antioxidants, including enzymes, such as superoxide dismutase and catalase (CAT) and low-molecular-weight antioxidants, such as vitamins C and E, and glutathione peroxidase (GPX), are the main orchestrators of this defensive barrier.
   - Mitochondria are a primary target of the aging process, as evidenced by a decline in mitochondrial oxidative capacity in both skeletal and heart muscle with age.
     - Aging has been associated with excessive oxidative stress and overproduction of RNS. Indeed, oxidative/nitrosative stress is thought to be an important contributor to the degeneration of long-lived post-mitotic cells, such as cardiomyocytes and neurons. This explains in part the relation between acquired cardiac and neurodegenerative diseases and aging.