Cellular Nutrition and Its Influence on Age-Associated Cellular Decline (AACD)



Researchers have identified several molecular pathways and cellular processes that appear to underlie both aging and age-related chronic disease.

Cellular changes associated with aging are cumulatively referred to as age-associated cellular decline (AACD) and include defects in mitochondrial function.

Many risk factors are associated with AACD.

Identifying AACD risk factors and intervening with cellular nutrients earlier in the aging process, before major mobility disabilities and disease-driven limitations emerge, could help improve overall healthy aging.

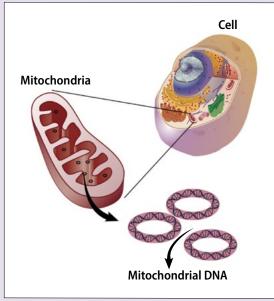
Risk Factors Associated With AACD

Clinical Risk Factors	Behavioral/Environmental Risk Factors
 Clinical conditions (e.g., cancer, cardiovascular, renal, or metabolic disease) 	SmokingSedentary lifestyle
• Obesity	 Low physical activity
 Unfavorable genetic background 	 Persistent physical or psychological stress
Insulin resistance	 Low socioeconomic status
 Low physical capacity (e.g., slow gait speed, 	Alcohol abuse
muscle weakness)	 Inadequate nutrition
	Air pollution

Mitochondria

- Mitochondria are organelles that produce cellular energy in the form of adenosine triphosphate (ATP).²
 - Other functions of mitochondria include regulating cellular metabolism, regulating apoptosis (programmed cell death), and signaling by producing reactive oxygen species.
- Mitochondria are dynamic and can change size, shape, and position throughout their life cycle. They regularly undergo fission (division of a single mitochondrion into multiple mitochondria) or fusion (the combination of two or more mitochondria into a single mitochondrion).³
- Mitochondrial homeostasis is maintained through a balance of fusion, fission, mitochondrial biogenesis (creation of new mitochondria), and mitophagy (the selective degradation of mitochondria).

Mitochondria and Mitochondrial DNA



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Learn more about Cellular Nutrition and Its Influence on AACD

Mitochondrial Function and AACD

- Declining mitochondrial function results in AACD. These changes include:
 - Decreased mitochondrial biogenesis.⁴
- Increased mitochondriamediated apoptosis.⁴
- Decline in mitochondrial DNA (mtDNA).⁴
- Increased mtDNA mutations.⁴
- Abnormalities in mitochondrial function are associated with many diseases such as:
 - Cancer, cardiovascular diseases, and neurodegenerative disease.

Source: Reference 2.

AACD Associations With Health and Physical Function

- AACD is often associated with fatigue, reduced strength and daily energy, and low physical activity.^{5,6}
- Changes associated with AACD act as triggers for age-associated diseases and conditions. For example:
 - -The age-related decline in nicotinamide adenine dinucleotide (NAD⁺) has been associated with the development of diabetes, nonalcoholic fatty liver disease, atherosclerosis, Alzheimer's disease, retinal degeneration, chronic fatigue syndrome, and depression.^{7,8} Conversely, research has shown that NAD⁺ intermediates, such as nicotinamide mononucleotide and nicotinamide riboside, may potentially be effective for preventing and treating age-associated pathophysiology.⁷
 - -Glutathione levels are lower and oxidative stress is higher in conditions associated with mitochondrial dysfunction, including aging, HIV infection, diabetes, neurodegenerative disorders, cardiovascular disorders, neurometabolic diseases, cancer, and obesity.

Nutritional Interventions to Address AACD Emerging research indicates that nutritional components that target specific mechanisms associated with AACD hold promise for improving the health and well-being of adults.⁶ Adoption of healthful eating patterns and - Dietary supplementation with these components may exercise has been shown to improve markers be an alternative approach to lifestyle interventions of age-associated diseases and attenuate targeting AACD. biological aging. Nutritional interventions may slow Emerging research also indicates that some nutritional compounds can support healthy AACD and have the potential to aging by influencing mitochondrial repair and extend human healthspan—the period of life in which people are preservation, quality control, and signaling.⁶ in good health. - Compounds that have been shown to address mitochondrial damage and clinical disease states include Szeto-Schiller (SS) peptides, coenzyme Q_{10} (Co Q_{10}), MitoQ, and glycine and N-acetylcysteine (GlyNAC).11-14 For example, calorie restriction (CR) may - Compounds that may address mitochondrial modulate cellular function and increase longevity. quality control include sirtuins, mitochondrial - CR combined with exercise has some beneficial synergistic division inhibitor (mdivi), urolithin A, and effects, such as improving insulin sensitivity, lowering systemic epicatechin.15-18 inflammation, and improving body composition. However,

 Compounds that have been shown to address mitochondrial signaling include nicotinamide riboside and nicotinamide mononucleotide.^{19,20}

Developed by



remains a challenge.

added to CR.9,10



exercise may not provide an additive benefit for longevity when

 CR appears to improve markers of disease risk in humans, but its acceptability and feasibility particularly over the long term

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