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„Regulation of Nuclear and Mitochondrial Epigenetics through Exercise and Nutrients for Healthy Aging“

Abstract

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In an era of rapid scientific advancement, we almost everyday hear incredibly exciting breakthroughs claiming to extend human lifespan or even reverse aging (rejuvenation). Personally, I believe that we do not truly understand what aging is, and therefore all claims of anti-aging breakthroughs are unreliable. First, although the average human life expectancy has risen from 40 to 80 years, this is primarily attributable to improvements in public health—such as the reduction of epidemics and infant mortality—rather than a breakthrough in biological limits. Second, results from animal trials basically only marginally extend average lifespan, not maximum lifespan. Most importantly, these results likely stem from animals whose lifespans were already shortened for various reasons, rather than from normal, long-lived animals. Although we don't know why humans age and whether we can delay or even reverse aging, we are all familiar with the historical anecdote of " A life-threatened person’s hair turned gray overnight," which demonstrates that stress can accelerate aging. Why does stress accelerate aging? The theory that oxidative damage caused by mitochondrial free radicals leads to aging has brought mitochondria into the forefront of aging research. Psychological and physical stresses have been identified as a catalyst for accelerated aging. Mitochondria, known for their dynamic nature and adaptability, function as a highly sensitive stress sensor and central hub in the process of accelerated aging. In this review, we explore how mitochondria as sensors respond to stress and contribute to the molecular processes in accelerated aging by viewing mitochondria as hormonal, mechanosensitive and immune suborganelles. This understanding of the key role played by mitochondria and the close association with accelerated aging helps us to distinguish normal aging from accelerated aging, correct misconceptions in aging studies, and develop strategies such as exercise, stem cell, mitochondrial transfer and mitochondria-targeting

nutrients/ drugs for regulating mitoepigenetics to delay accelerated aging and prevent/treat age-related diseases.

Short CV

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Dr. Jiankang Liu received his BS from Xi'an Jiaotong Unviersity and PhD of Medical Science from Okayama University School of Medicine, Japan. He completed post-doc training in Dr. Bruce Ames laboratory at University of California, Berkeley and worked as a faculty at University of California at Berkeley, Children Hospital Oakland Research Institute, University of California at Irvine, University of Kentucky College of Medicine, and University of Southern California. Since 2008, Dr. Liu returned China and worked at Xi'an Jiaotong University as Professor and Dean of the School of Life Science and Technology. Currently, he is a Professor and First and Honorary Dean of the School of Life Sciences, University of Health and Rehabilitation Sciences at Qingdao and Xi'an Jiaotong University at Xi'an, China. Dr. Liu's research interests include molecular and cellular mechanisms of aging, stress, and age-/stress-associated diseases with a focus on redox-mitochondrial metabolism and mitoepigenetics. He has published more 280 papers（H-index 87）and was elected as one of the Most Cited Chinese Researchers for 12 years (2014-2025) and the World Top Scientists from 2019-2026.