Editorial

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Metabolic Syndrome and Longevity: A Framework of Situation

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ABSTRACT

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Editorial

Dietary intake, physical activity, and waste elimination choreograph anabolic and catabolic processes that govern development, maturity, and aging in the human superorganism (which includes the host and its microbiome). Metabolism research is experiencing a renaissance after years of being eclipsed by advancements in cellular and molecular biology. Metabolites are becoming increasingly important in all biological processes, including physiological and pathological aging. Several organ metabolic features have been linked to lifespan in recent cross-species research [1]. Several studies have found a relationship between sphingomyelin levels and longevity [2]. On the other hand, triacylglycerols with polyunsaturated fatty acid (PUFA) side chains and inflammatory by-products are inversely related to longevity [3]. Sphingomyelin levels in the blood and PUFA-containing triacylglycerol levels in the blood have both been linked to female familial lifespan in humans [2]. Longevity is negatively proportional to hepatic levels of enzyme cofactors involved in amino acid metabolism as well as hepatic concentrations of tryptophan breakdown products across mammalian species. Reducing dietary amino acids, notably tryptophan and methionine, has been shown in animal experiments to extend lifespan [4]. As a result, long-lived mammals may consume less energy per day per unit of body mass (mass-specific basal metabolism).

Using proxies like telomere length, gene methylation (which would reflect a "epigenetic clock"), and transcriptional fingerprints (which would demonstrate "transcriptomic aging"), some studies have attempted to determine human biological age [5]. It has also been investigated to assess metabolic features associated with aging. Researchers developed a "metabolic age score" founded on Nuclear Magnetic Resonance methods as a consequence of this finding, that could indicate longevity regardless of the physical maturity or other risk factors [6]. The blood metabolome and lipidome can regularly detect age-related changes and some early indications of age-related illnesses in people [7]. Many human metabolic gene variations have been linked to living to be 100 years old, including those in forkhead box O3 (FOXO3) and other PI3K/AKT1 signaling genes [8]. Male children of centenarian families have lower abdominal visceral fat, suggesting that a healthy metabolic profile is related to family longevity [9]. Many genetic flaws that cause rapid aging in humans, on the other hand, are linked to metabolic difficulties. Cutis laxa (proline biosynthesis defects), Ehlers-Danlos syndrome (proteoglycan synthesis defects), Lenz-Majewski hyperostotic dwarfism (phosphatidylserine synthesis defects), SHORT syndrome (PIK3R1 hypomorphic mutations), and progressive external ophtalmoplegia (mitochondrial DNA instability) are all examples of these disorders [10]. Even though

the bulk of genetically characterized progeroid illnesses are caused by mutations in genes that preserve genomic integrity and hence damage metabolism secondarily, these examples highlight the possible significance of metabolic deficits in aging [11].

As people age, maintaining cellular and organismal metabolic balance becomes more difficult, favoring a metabolic imbalance that self-amplifies and eventually manifests clinically [12]. As a consequence, all of the foregoing anti-aging treatments could be paired with metabolic restructuring, which improves

(1) Food efficiency and

(2) Stress resistance [13].

There are certain commonalities in metabolic reprogramming, despite the fact that it can be very wide and hence difficult to treat pharmacologically. Signal-transduction cascades and metabolic circuitries that remodel as humans age may work in the context of a small number of modules that shift nutrients and other resources from anabolism to non-toxic catabolism, preserving homeostasis [14]. A notable example is the longevity-extending advantages of various techniques that inhibit "insulin and IGF1 signaling" (IIS) or activate autophagy [15,16]. When considering aging and longevity in the context of a systemic rewiring of intermediate metabolism, a number of cautions should be carefully considered [17]. To begin with, many of the studies described above were done on C. elegans or D. melanogaster and have yet to be confirmed in mammals [18]. Second, because there are no well-defined biological biomarkers of aging, determining the short- and long-term impacts of metabolic therapy on the aging process is difficult [19]. Third, none of the longevity-prolonging medicines discussed above has been proved to prevent the development or progression of age-related disorders in humans [17]. We are sure that combining aging and metabolism research will reveal new insights into the aging process, assisting in the development of therapeutically useful healthspan and lifespan extension medicines [20]. In this environment, organizations such as the National Institute on Aging Interventions Testing Program (NIA-ITP), which is attempting to standardize the experimental models and procedures used to research longevity around the world, are likely to play a vital role [21].

Although there is little doubt that a combination of regular exercise and a healthy diet can delay the onset and progression of all indicators of aging, our current understanding of metabolic changes that can improve elderly health and hence extend longevity is still in its infancy. Dietary advice is difficult to come by, and specialized advice from a nutritionist may be advantageous in some circumstances. However, we believe that extending food-free intervals, reducing overall caloric and animal protein intake, and moving to a Mediterranean diet rich in fibers and complex carbohydrates, when paired with regular physical activity, could have considerable anti-aging effects [22,23]. The worldwide trend toward a westernized lifestyle is introducing new health hazards that must be addressed through public awareness efforts. Positive affect and subjective well-being are surprisingly connected to positive neuroendocrine, cardiovascular, and inflammatory indices [24], implying a biological foundation for the long-suspected link between happiness and health [25]. As a result, policies that promote and democratize high-level education across socioeconomic strata, as well as policies that prioritize peaceful cooperation over vicious competition among individuals, may promote healthy aging and thus comprise the "ultimate preventative medicine".

Disclosure Statement

The authors declare that there are no conflicts of interest.

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