Short Report

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Ultra-Processed Diets as Accelerators of Inflammaging: A Hypothesis Linking Diet to Premature Biological Aging

Falah Hasan Obayes AL-Khikani*[1,2]

*[1] Department of Medical Laboratory Technology, College of Medical Technology, The Islamic University, Najaf, Iraq
[2] Department of Microbiology, Al-Shomali General Hospital, Babylon Health Directorate, Babylon, Iraq

*Corresponding author: Falah Hasan Obayes AL-Khikani ; falahgh38@gmail.com

Aging is increasingly recognized as a biological process driven not only by genetic determinants but also by chronic environmental and lifestyle exposures. Among the most influential exposures is diet. The concept of inflammaging defined as a persistent, low-grade, systemic inflammatory state that develops with advancing age has emerged as a central mechanism linking aging to immune decline and chronic disease. This letter proposes the hypothesis that chronic consumption of ultra-processed foods may act as a potent accelerator of inflammaging, thereby promoting premature biological aging and immune dysfunction.

Ultra-processed diets are characterized by high levels of refined sugars, industrial fats, sodium, emulsifiers, and artificial additives, combined with a relative absence of dietary fiber and bioactive micronutrients. Accumulating evidence indicates that such dietary patterns promote sustained activation of inflammatory pathways, including NF-κB signaling and inflammasome activation, leading to chronic cytokine production. Persistent elevation of pro-inflammatory mediators such as interleukin-6 and tumor necrosis factor-α closely mirrors the inflammatory phenotype observed in biological aging [1].

Inflammaging is tightly linked to immune system remodeling, particularly immunosenescence. Chronic dietary inflammation may accelerate age-related shifts in immune cell populations, including reduced naïve T-cell pools, expansion of senescent T cells, and impaired regulatory immune mechanisms. These changes compromise host defense, reduce vaccine responsiveness, and increase susceptibility to infections and chronic inflammatory diseases, even in younger populations exposed to long-term processed food consumption [2].

Mitochondrial dysfunction represents another critical axis connecting processed diets to accelerated aging. Ultra-processed foods have been associated with increased oxidative stress and impaired mitochondrial bioenergetics, leading to excessive reactive oxygen species production. Mitochondrial damage amplifies inflammatory signaling and contributes to cellular senescence, a hallmark of aging characterized by irreversible cell cycle arrest and pro-inflammatory secretory phenotypes. Senescent cells further propagate inflammaging through the senescence-associated secretory phenotype (SASP), creating a self-reinforcing inflammatory loop [3].

The gut microbiome plays a pivotal intermediary role in diet-driven inflammaging. Diets rich in ultra-processed foods disrupt microbial diversity and promote dysbiosis, favoring pro-inflammatory microbial metabolites while reducing short-chain fatty acid production. These microbial alterations compromise gut barrier integrity, increase systemic endotoxin exposure, and sustain chronic immune activation, all of which are recognized contributors to aging-related inflammation [4].

Notably, inflammaging is not restricted to advanced chronological age. Emerging data suggest that dietary patterns may shift biological aging trajectories independently of chronological time. Ultra-processed diets may therefore induce a state of "accelerated aging," in which younger individuals exhibit inflammatory, metabolic, and immune features typically associated with older age. This hypothesis aligns with recent observations linking ultra-processed food intake to increased risk of cardiovascular disease, metabolic disorders, and all-cause mortality [5].

Despite growing recognition of inflammaging as a driver of chronic disease, the specific contribution of ultra-processed diets to premature immune aging remains underexplored. Integrative studies combining dietary exposure assessment, inflammatory biomarkers, immune phenotyping, and biological aging markers such as epigenetic clocks are urgently needed. Such investigations

may clarify whether dietary modification can decelerate inflammaging and restore immune resilience.

In conclusion, this hypothesis suggests that chronic consumption of ultra-processed foods may accelerate inflammaging through sustained immune activation, mitochondrial dysfunction, microbiome disruption, and cellular senescence. Recognizing ultra-processed diets as modulators of biological aging may provide novel preventive strategies to preserve immune function and delay the onset of age-related diseases.

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