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Editorial

Nutritional modulation of inflammation in knee osteoarthritis: integrating dietary bioactives into precision disease modification

Aavrati Rastogi[1], Roop B. Kalia[1]*, Pradeep K. Meena[1], Mukesh Bairwa[2]

[1]Department of Orthopedics, AIIMS Rishikesh, Uttarakhand, India
[2]Department of Internal Medicine, AIIMS Rishikesh, Uttarakhand, India

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***Correspondence:**
Dr. Roop B. Kalia,
E-mail: roop.orth@aiimsrishikesh.edu.in

Traditionally thought of as a degenerative joint condition, knee osteoarthritis (OA) is now more widely understood as a chronic low-grade inflammatory and metabolic disease, with systemic factors like food having a major impact on the disease's start, progression, and severity of symptoms. The growing notion of "metabolic OA " underscores how dietary inflammation affects joint health.[1,2-10]

At the molecular level, OA is characterized by the activation of inflammatory pathways driven by cytokines, including interleukin 1 beta (IL 1β), interleukin 6 (IL 6), and tumor necrosis factor alpha (TNF α), which promote synovial inflammation and cartilage destruction.[3,6] The nuclear factor kappa B (NF κB) signaling system, a master regulator of inflammatory gene expression and matrix-degrading enzymes, is essential to this process.[6,4]

Dietary habits largely determine systemic inflammatory state. Elevated inflammatory biomarkers, such as C-reactive protein (CRP), are associated with diets high in ultra-processed foods, refined carbohydrates, and saturated fats, which contribute to OA progression.[5] On the other hand, diets high in bioactive substances such as antioxidants, polyphenols, omega-3 fatty acids, and vitamins have chondroprotective and anti-inflammatory properties.

Punicalagin, curcumin, and resveratrol are examples of polyphenols that have been shown to reduce pro-inflammatory cytokine production and inhibit NF-κB activation.[4,6] Additionally, these substances slow degenerative processes and maintain the integrity of the cartilage extracellular matrix by downregulating matrix metalloproteinases (MMPs), especially MMP-13.[6,7] Furthermore, polyphenols enhance cellular antioxidant defenses, thereby reducing chondrocyte apoptosis induced by oxidative stress.[7]

Another essential nutrient with anti-inflammatory qualities is omega-3 fatty acids. Omega-3 fatty acids attenuate joint inflammation by reducing the synthesis of pro-inflammatory mediators and increasing the production of specific pro-resolving lipid mediators through the modulation of eicosanoid pathways.[1,8] This process is especially important in OA because prolonged inflammation sustains tissue damage.

A key factor in the pathophysiology of OA is oxidative stress, characterized by an excess of reactive oxygen species (ROS) and compromised antioxidant defenses. Vitamins C and E, as well as polyphenolic chemicals, are examples of nutritional antioxidants that are essential for preventing ROS, protecting chondrocyte viability, and keeping joint homeostasis.[7]

Crucially, the metabolic milieu of joint tissues is also influenced by nutrition. Dietary habits are intimately linked to the pathogenesis of OA because obesity-related adipokines and metabolic mediators increase inflammatory signaling in the joint.[2,10] This emphasizes the necessity of viewing diet as a key element of illness modification rather than just a supporting treatment.

The role of dietary treatments in OA therapy is supported by clinical evidence. Following anti-inflammatory eating

habits, such as the Mediterranean diet, has been linked to decreased knee OA prevalence, better physical function, and less pain.[9,11] Additionally, nutraceutical supplements have shown promise in improving function and reducing symptoms, which supports the clinical significance of dietary modification.[4,6]

The incorporation of nutrition into the treatment of OA aligns with the emerging paradigm of precision and personalized medicine, in which therapies are tailored to each patient's metabolic, inflammatory, and biomechanical profiles. Pharmacological therapies, rehabilitation, and nutritional methods may work in concert to improve both structural and functional results.

There are still issues, such as response heterogeneity across individuals, inconsistent dietary guidelines, and a lack of long-term randomized controlled trials. Furthermore, a crucial subject for further research is the relationship between inflammatory pathways, gut bacteria, and nutrition.

In the treatment of knee OA, nutritional modulation of inflammation is a physiologically sound and clinically significant approach. Dietary bioactives can shift therapy paradigms from symptomatic relief to disease management and metabolic regulation by targeting key inflammatory and oxidative pathways. Current OA management approaches may be insufficient if they don't address the systemic inflammatory milieu induced by food. A key component of integrated, precision-based musculoskeletal therapy will soon involve nutrition.

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