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Mechanism and Research Progress of Anti-Inflammatory Dietary Intervention in Tic Disorders and Their Comorbidities

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Abstract: *Tic disorder represents a prevalent neurodevelopmental disorder, yet conventional pharmacotherapy frequently causes notable adverse effects. Anti-inflammatory diets show therapeutic promise for neuropsychiatric conditions through modulating gut microbiota and relieving neuro inflammation and oxidative stress. This paper reviews mechanisms and evidence of Mediterranean, ketogenic and elimination diets in tic disorders and comorbid ADHD, aiming to offer references for clinical practice.*

Keywords: *tic disorders; intestinal microbiota; gut-brain axis; anti-inflammatory diet; Mediterranean diet*

1. Introduction

Tic disorders (TD) refer to neurodevelopmental diseases with onset in childhood or adolescence, characterized by involuntary, abrupt motor or vocal tics [1]. According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), TD is classified into three categories based on clinical manifestations and disease course: transient tic disorder (TTD), chronic motor or vocal tic disorder (CTD), and Tourette syndrome (TS) [2]. Estimates suggest approximately 10 million children and adolescents in China suffer from TD annually, including around 2 million TS cases [3]. Conventional TD management mainly relies on pharmaceutical intervention. Though drug therapy achieves certain therapeutic effects, it carries obvious side effects such as sedation, aggravated depressive and anxious mood, and severe impairment of motor function [4]. Furthermore, nearly all antipsychotic medications induce weight gain, which may trigger series of metabolic complications including diabetes mellitus and hyperlipidemia, posing latent long-term health risks to patients [5].

With emerging evidence linking TD to gut dysbiosis, chronic inflammation and autoimmunity, safe and effective non-pharmacological alternatives have drawn increasing attention, shifting TD treatment strategies toward nutritional and anti-inflammatory interventions [6]. Studies have validated that specific nutrients contained in anti-inflammatory diets—especially the Mediterranean diet (MedDiet), ketogenic diet (KD), and Mediterranean-DASH Intervention for Neurodegenerative Delay (MIND) diet—can regulate nervous system function by modulating neuroinflammation and reshaping intestinal flora [7]. Nevertheless, TD-related research in this field remains scarce compared to its comorbidities such as autism spectrum disorder (ASD) and ADHD [8]. Hence, this review focuses on anti-inflammatory dietary interventions for TD and its comorbidities, particularly concurrent ADHD, establishes a theoretical bridge connecting intestinal health, chronic inflammation and TD, and expands non-pharmacological nursing strategies for TD.

2. Proposition of Anti-Inflammatory Diets and Relevant Inflammatory Mechanisms

Inflammation serves as a protective response of organisms against pathogens or irritants, playing an indispensable role in host defense, pathogen clearance and tissue injury repair. Appropriately regulated inflammatory responses facilitate pathogen elimination, tissue repair and homeostasis restoration [9]. When excessive triggers (free radicals, oxidative stress, exogenous pathogens) overactivate

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inflammatory cascades, uncontrolled chronic inflammation occurs under persistent stimulation, resulting in local or systemic tissue damage [10].

Dietary adjustment constitutes one major intervention to mitigate inflammation. Diet can be either pro-inflammatory or anti-inflammatory, determined by hormonal responses induced by food ingredients and interactions between specific nutrients and the innate immune system during inflammatory reactions [11]. Anti-inflammatory diets barely activate inflammatory cascades, whereas pro-inflammatory diets trigger elevated circulating inflammatory biomarkers including cytokines, chemokines and prostaglandin E2 (PGE2).

The concept of anti-inflammatory diets was first proposed by Dr. Barry Sears in The Zone Diet in 1995. Major well-studied categories include the MedDiet, KD and Dietary Approaches to Stop Hypertension (DASH), among which the MedDiet has accumulated the most abundant research data [12]. Although a unified definition of anti-inflammatory diets has not yet been established, they are recognized as lifestyle regimens composed of natural food patterns (Mediterranean, ketogenic, etc.), rather than simple fixed recipes. Anti-inflammatory diets prioritize plant-based foods rich in fruits, vegetables, whole grains, olive oil, seafood, legumes and nuts; red meat, processed foods, refined sugar and saturated fat are restricted. Fresh, seasonal and local ingredients are advocated, combined with healthy cooking, physical activity, relaxation and mindfulness as integrated sociocultural components.

The Dietary Inflammatory Index (DII) quantifies the pro-or anti-inflammatory properties of food components to categorize dietary patterns. High DII scores correspond to elevated systemic inflammatory potential, while low DII scores indicate anti-inflammatory capacity [13]. The DII has been correlated with multiple chronic illnesses including tumors, cardiovascular diseases, diabetes and osteoporosis [14].

3. Mechanisms Underlying Anti-Inflammatory Dietary Interventions for TD

3.1 Modulation of Intestinal Microecology and the Gut-Brain Axis

The gut microbiota consists of trillions of microorganisms and their collective genomes, exerting pivotal roles in human development, physiological homeostasis and disease progression. It directly participates in synthesizing key neurotransmitters including γ-aminobutyric acid , serotonin (5-HT), glutamate and dopamine (DA), all closely implicated in TD, ADHD and obsessive-compulsive disorder. The bidirectional molecular signaling channel linking gut microbiota and the central nervous system is defined as the microbiome-gut-brain axis (mGBA) [15]. Recent research confirms inflammatory factor alterations also participate in mGBA signaling. Gut dysbiosis, a pathological imbalance of intestinal microbial communities, impairs homeostatic function through multiple pathways: production of toxic metabolites, disrupted intestinal barrier integrity and aggravated inflammation [16].

Gut dysbiosis damages intestinal epithelial cells, widens intercellular gaps and increases intestinal permeability, a pathological state termed leaky gut syndrome [17]. Pro-inflammatory mediators permeate intercellular gaps into systemic circulation to trigger pan-inflammation; pro-inflammatory cytokines further modulate CNS function by altering cerebral 5-HT concentrations [18].

Structural alterations of gut microbiota have been identified in children with TD. Two existing studies comparing gut microbiota between TD patients and healthy controls detected no significant differences in alpha diversity [19].Wang [16] reported distinct beta diversity profiles: TD patients exhibited depleted Bifidobacterium and Collinsella, alongside enriched unclassified Ruminococcaceae, Prevotella, Faecalibacterium, Coprobacillus and Odoribacterium. Elevated Ruminococcaceae and reduced Prevotella represent characteristic microbial signatures in pediatric TD, potentially mediating disease pathogenesis.

Diet acts as a core regulator of gut microbiota diversity, abundance and immune responses [20]. Dietary nutrients including vitamins, minerals, polyunsaturated fatty acids and amino acids are essential for maintaining cerebral structure and function, participating in diverse metabolic cascades, cellular signal transduction, glucose/lipid metabolism and neurotransmitter synthesis [21] providing a safe, low-cost therapeutic strategy for TD management. Adherence to the MedDiet correlates with favorable gut microbial remodeling: reduced Escherichia coli, elevated Bifidobacterium/E. coli ratio, suppressed Candida albicans, increased total bacterial load and higher short-chain fatty acid concentrations [22].

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3.2 Regulation of Neurotransmitter Homeostasis

TD pathogenesis involves multifactorial etiologies including genetics, neurotransmitter imbalance, immune dysfunction and gut dysbiosis, among which neurotransmitter disturbance represents the most widely recognized pathological mechanism, manifesting as dysfunction within the cortico-striatal-thalamic-cortical circuit. Norepinephrine and dopamine mediate neural regulation in TD-associated cerebral regions and circuits . Recent neuromodulation studies further confirm that modulating striatal dopamine release markedly alleviates TD symptoms, providing direct evidence for dopamine's central role in TD pathogenesis [23].

Moreover, neuroinflammation drives dopaminergic dysfunction in TD. Elevated pro-inflammatory cytokines (IL-6, TNF-α, C-reactive protein (CRP)) disrupt the synthesis and release of serotonin, dopamine and norepinephrine.Specific nutrients mitigate TD manifestations by attenuating neuroinflammation, reshaping gut microbiota and directly interfering with cerebral dopamine metabolism. For instance, gastrodin reduces stereotypical abnormal behaviors via downregulating D2 receptor density and upregulating dopamine transporter expression, while decreasing serotonin transporter density to indirectly suppress dopamine release [24]. Animal experiments demonstrate dietary patterns profoundly regulate the cerebral reward system: long-term high-calorie diets desensitize dopaminergic neuronal responses to standard food without impairing sensitivity to high-fat meals. Longitudinal recordings indicate this adaptive remodeling occurs at the hypothalamic agouti-related protein neuron and midbrain peripheral dopamine signaling levels [25].

3.3 Alleviation of Neuroinflammation

Neuroinflammation refers to inflammatory reactions occurring within the CNS (brain and spinal cord). Cumulative evidence identifies neuroinflammation as a key pathological driver of TD, triggered by pathogenic stimuli including group A streptococcus (supporting the PANDAS hypothesis), COVID-19, enteroviruses and allergens. These stimuli activate adaptive immunity to induce T-cell secretion of pro-inflammatory cytokines or B-cell production of anti-neuronal antibodies, ultimately causing neuronal injury and tic exacerbation [26]. TD is mediated by host T-cell autoimmunity: pathogens crossing the blood-brain barrier aberrantly activate autoreactive T lymphocytes to initiate autoimmune responses with massive pro-inflammatory cytokine secretion [27]. On one hand, pro-inflammatory cytokines disrupt blood-brain barrier integrity to infiltrate the CNS; on the other hand, inflammatory mediators interfere with astrocyte-neuron metabolic coupling, dysregulate gut-brain axis signaling and overactivate the kynurenine pathway (KP), triggering imbalances in dopamine, glutamate and other neurotransmitter systems to directly induce or aggravate TD [28].

Micronutrient deficiencies also exacerbate neuroinflammation: magnesium insufficiency induces oxidative stress, disrupts essential fatty acid metabolism and alters serotonin, dopamine and norepinephrine concentrations ; vitamin D deficiency exacerbates neuroinflammation and oxidative stress , both accelerating TD progression. Diets rich in antioxidants (vitamin C, vitamin E, polyphenols) and omega-3 fatty acids eliminate free radicals, suppress neuroinflammation and inhibit pro-inflammatory mediator release, reducing TD incidence and mitigating clinical manifestations[29] .

3.4 Improvement of Oxidative Stress Status

Oxidative stress arises from imbalance between oxidative and antioxidant systems, generating excessive reactive oxygen species (ROS) that induce cellular damage, and serves as a critical contributor to TD progression [30]. Neuroinflammation activates cerebral immune cells to release toxic oxidative metabolites, triggering chronic oxidative stress , which further causes protein/lipid peroxidation, membrane damage, neuronal degeneration and impaired cerebral function to facilitate TD onset [31].

M. Sarchioto [32] first identified glutathione (GSH) imbalance in the right frontal white matter of adult TS patients via magnetic resonance spectroscopy, linking neuronal oxidative stress severity to tic intensity. In TS rat models, Guo Lixian [33] verified that gastrodin alleviates motor and stereotyped behaviors by activating the BDNF/NF-κB pathway and regulating oxidative stress-related factors. Extra virgin olive oil (EVOO), the primary lipid source of the MedDiet, exhibits potent antioxidant capacity, conferring protective effects against cardiovascular disease, malignancies, neurodegeneration and diabetes mellitus .

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4. Application of Major Anti-Inflammatory Dietary Patterns in TD

Anti-inflammatory dietary interventions are promising for TD,but high-quality clinical trials recruiting TD patients are still limited,and their definite efficacy and mechanisms need further validation.TD and ADHD share overlaps in genetics, neural circuits and neuroinflammatory pathophysiology,thus dietary evidence from ADHD studies can inform TD management.This section reviews anti-inflammatory dietary research in ADHD for translational implications in TD.

4.1 Research on Mediterranean Diet Application

The MedDiet originated as a traditional dietary pattern documented in the late 1950s–1960s among olive-growing populations in Crete, Greece and southern Italy. It is defined as a plant-centered regimen featuring abundant fruits, vegetables, whole grains, legumes, nuts and seeds with EVOO as the primary lipid source; moderate fermented dairy, fish and seafood consumption; restricted red meat, processed meat, butter and ultra-processed foods [34].

Existing epidemiological data demonstrate inverse correlations between MedDiet adherence and ADHD risk, yet high-quality interventional evidence supporting MedDiet for ADHD symptom management remains limited [35]. In 2016, Ríos-Hernández [36] conducted the first pediatric cohort study investigating associations between low MedDiet adherence and ADHD diagnosis among 120 children, reporting that poor MedDiet compliance increased ADHD risk sevenfold, with insufficient fruit/vegetable intake and excessive sugar/fast food as key risk factors. While this cross-sectional study cannot establish causal relationships, it pioneered a paradigm focusing on holistic dietary patterns rather than isolated nutrients. Subsequent Iranian pediatric research replicated these findings, further identifying a significant dose-response relationship between MedDiet adherence and ADHD risk after adjusting for confounders including socioeconomic status and family history, advancing causal inference [37]. To acquire interventional evidence, San Mauro Martin [38] implemented the first randomized controlled trial enrolling 60 ADHD children receiving combined MedDiet and omega-3 intervention, observing trending improvements in impulsivity. However, limited sample size restricted statistical power, requiring larger cohorts to validate conclusions.

Beyond childhood dietary exposure, prenatal and perinatal environments profoundly shape neurodevelopment. Maternal infection, gestational smoking and other prenatal/perinatal triggers activate microglia-mediated neuroinflammation, elevating TD susceptibility in genetically vulnerable offspring; secondary stimulation during critical developmental windows reactivates peripheral immune responses to drive TD pathogenesis [39]. Based on this mechanism, Cendra-Duarte [40] shifted research focus to fetal development, discovering that maternal MedDiet adherence during pregnancy significantly reduces behavioral disturbances (including ADHD manifestations) in 4-year-old children, providing novel insights into early nutritional prevention of neurodevelopmental disorders.

4.2 Research on Ketogenic Diet Application

The ketogenic diet (KD) is characterized by high fat, extremely low carbohydrate and moderate protein intake. KD rewires cellular energy metabolism to replace glucose with ketone bodies, protecting multi-organ physiological function [41]. Its therapeutic efficacy in neurological disorders including epilepsy, Alzheimer's disease, migraine and Parkinson's disease has been partially validated . Emerging hypotheses link KD benefits to the microbiome-gut-brain axis, whereby intestinal microbiota modulate CNS function to regulate neurobehavioral disorders [42]. Existing studies confirm KD alleviates colitis by remodeling gut microbiota and suppressing group 3 innate lymphoid cells in the colon; in murine ASD models, KD mitigates neurological manifestations via gut microbial remodeling [43]. Nevertheless, research exploring KD-mediated TD improvement through the microbiome-gut-brain axis remains preliminary, with current evidence limited to animal experiments. A prospective, randomized, double-blind, controlled canine study observed KD-induced amelioration of hyperactive behaviors [44], yet interspecies discrepancies in diagnostic criteria, subjective experience and core pathology between canine ADHD-like behaviors and human neurodevelopmental disorders restrict direct translational value for human clinical practice. A rat study reported comparable efficacy between KD and pharmaceutical intervention in alleviating hyperactivity in spontaneously hypertensive rats (SHR), alongside significantly elevated gut microbial alpha diversity and restored dysbiotic profiles [45]. These findings imply KD may relieve ADHD symptoms via gut-brain axis regulation, yet translation to human pediatric TD requires rigorous high-quality clinical trials to verify efficacy, safety and specific molecular mechanisms, including identification of KD-modulated bacterial taxa and their signaling

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pathways (vagus nerve, immunity, metabolism) regulating cerebral attention and impulse control.

4.3 Research on Elimination Diet Application

The elimination diet concept was first proposed by Albert Rowe in 1926 to manage food allergies. In 1976, Hall[46] documented elimination diets for neurological hypersensitivity-related behavioral disorders including tics, learning disabilities, schizophrenia and depression. Concurrently, the Feingold diet regimen was developed for hyperactivity, establishing longstanding clinical recognition of diet-behavior interactions for emotional, attentional and behavioral disturbances.

The core principle of elimination diets involves excluding potential adverse food triggers: natural allergensand artificial additives (synthetic pigments, flavorings, sweeteners, preservatives). Regimens range from single-food exclusion to strict oligoantigenic "few-food diets"[47]. The Feingold diet specifically eliminates artificial colorants and preservatives hypothesized to exacerbate hyperactivity. One trial reported behavioral improvements in 59 out of 78 hyperactive children following oligoantigenic diet intervention, supporting elimination diets as viable therapeutic approaches for such populations [48]. However, a systematic review of 12 double-blind, placebo-controlled trials by Krummell[49] concluded sugar and chocolate additives are not primary ADHD triggers, sparking controversy that drove rigorous follow-up research. Pelsser [50] demonstrated statistically significant attenuation of hyperactive symptoms in 62% of ADHD children after short-term elimination diet intervention. A landmark 2011 randomized controlled trial enrolling 100 ADHD children provided high-level evidence validating elimination diet efficacy [51]. Subsequent trials compared short- and long-term outcomes alongside cost-effectiveness between elimination diets and general healthy dietary regimens, emphasizing personalized interventions accounting for individual food sensitivity[52] . Recent mechanistic research highlights the microbiome-gut-brain axis, proposing complex crosstalk between diet, metabolism, immunity and endocrine signaling to modulate cerebral function.

5. Conclusion

D incidence has risen globally, causing lifelong impacts and substantial public-health burdens.This review elaborates pathological mechanisms of anti-inflammatory diets for TD and summarizes research progress of major dietary patterns in TD with comorbid ADHD.Dietary factors promote TD pathogenesis via gut dysbiosis, intestinal barrier damage, systemic inflammation and oxidative stress, with inflammatory cascades occurring in early-life critical developmental windows.Bidirectional interactions between ADHD and diet obstruct definite causal interpretation of diet-disease associations.Future clinical work requires personalized nutritional strategies to reduce inflammation, balance immunity and optimize gut health for neurological rehabilitation.While the Mediterranean diet and its bioactive components regulate gut microbiota and inflammatory responses, specific TD-oriented trials are still insufficient.Large-scale long-term randomized controlled trials are essential to validate nutritional benefits for TD and reveal relevant molecular mechanisms.These findings will support the integrated use of dietary therapy and conventional medical treatments.

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