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Therapeutic role of ketogenic diet in neurological disorders: epilepsy to alzheimer's – A review

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Abstract
The ketogenic diet (KD), a high-fat, low-carbohydrate, moderate-protein dietary intervention, has gained renewed attention for its therapeutic potential in various neurological disorders. By inducing ketosis and shifting the brain's energy metabolism from glucose to ketone bodies, KD modulates neuronal excitability, neurotransmitter balance, mitochondrial function, oxidative stress, and inflammation. This review summarizes the mechanisms and clinical evidence supporting the use of KD in epilepsy, neurodegenerative diseases including Alzheimer's disease (AD), Parkinson's disease (PD), and other neurological conditions such as autism spectrum disorder (ASD), migraine, and traumatic brain injury (TBI).

Keywords: Ketogenic diet, epilepsy, alzheimer, human health, therapeutic diet

Introduction

Neurological disorders are among the leading causes of morbidity worldwide, with rising prevalence due to increased life expectancy and lifestyle changes (Deuschl *et al*., 2020; Feigin *et al*., 2024; GBD 2021 Nervous System Disorders Collaborators, 2024) [6, 9, 12]. Conventional pharmacological therapies often offer limited efficacy or cause adverse effects (Rubio *et al*., 2025; Pietrzak *et al*., 2022) [28, 32]. The ketogenic diet, originally developed in the 1920s as a treatment for refractory epilepsy, has re-emerged as a promising metabolic therapy for various brain disorders (Wheless, 2008; Wilder, 1921) [37, 38]. Ketone bodies—β-hydroxybutyrate (BHB), acetoacetate, and acetone—not only serve as alternative fuels but also exhibit neuroprotective, anti-inflammatory, and anti-oxidative effects (Gano *et al*., 2014; Maalouf *et al*., 2009; Olson *et al*., 2023) [11, 20, 26]. These properties have stimulated growing research into the broader therapeutic applications of KD (Dyńka *et al*., 2023; Rubio *et al*., 2025) [7, 32].

Mechanism of Action of the Ketogenic Diet in the Brain

1. Modulation of Energy Metabolism

The ketogenic diet (KD) modifies cerebral energy metabolism by shifting the primary fuel source from glucose to ketone bodies such as β-hydroxybutyrate and acetoacetate, which cross the blood–brain barrier efficiently and are readily utilized by neurons and glial cells. These ketone bodies generate more ATP per unit of oxygen than glucose, thereby enhancing metabolic efficiency and compensating for impaired cerebral glucose metabolism, a hallmark of conditions such as Alzheimer's disease (AD) (Cunnane *et al*., 2016; Newman & Verdin, 2017) [3, 4, 24]. This metabolic shift not only improves energy availability but also reduces metabolic stress in neurons, ultimately contributing to enhance cognitive and neuroprotective outcomes.

2. Reduction in Neuronal Excitability

KD reduces neuronal excitability through multiple mechanisms, most notably by increasing GABAergic neurotransmission and reducing glutamatergic activity, thereby restoring the balance between inhibitory and excitatory signaling in the brain. This shift helps reduce seizure susceptibility in individuals with epilepsy (Rho, 2017) [31]. Additionally, KD activates ATP-sensitive potassium channels, which hyperpolarize neuronal membranes and dampen excessive neuronal firing, further contributing to seizure control and overall stabilization of neuronal networks (Ma *et al*., 2007) [19]. These combined effects make KD highly effective, especially in drug-resistant epilepsy.

3. Mitochondrial Biogenesis and Antioxidant Effects

Ketone bodies stimulate mitochondrial biogenesis and enhance mitochondrial efficiency, leading to increased ATP production and improved neuronal resilience. KD also reduces the generation of reactive oxygen species (ROS), owing to cleaner-burning ketone metabolism and upregulation of endogenous antioxidant systems such as superoxide dismutase and glutathione (Maalouf *et al*., 2009) [20]. By minimizing oxidative stress and preserving mitochondrial integrity, KD provides significant neuroprotective benefits, particularly relevant in neurodegenerative disorders characterized by mitochondrial dysfunction, including Parkinson's disease and Alzheimer's disease.

4. Anti-inflammatory Actions

Ketosis exerts potent anti-inflammatory effects, primarily through inhibition of the NLRP3 inflammasome, a key regulator of innate immune activation in the brain. The ketone body β-hydroxybutyrate directly suppresses NLRP3 activation, resulting in decreased production of pro-inflammatory cytokines such as IL-1β and IL-18 (Youm *et al*., 2015) [39]. By attenuating neuroinflammation—a major pathological component of Alzheimer's and Parkinson's diseases—KD helps reduce neuronal damage and supports healthier neural function.

5. Improved Synaptic Function and Gene Expression

KD enhances synaptic function and neuronal survival by influencing gene expression through epigenetic mechanisms. β-hydroxybutyrate functions as a histone deacetylase (HDAC) inhibitor, thereby promoting the expression of genes associated with stress resistance, neuroplasticity, and metabolic stability (Shimazu *et al*., 2013) [34]. This includes increased expression of brain-derived neurotrophic factor (BDNF), which supports synaptic growth, learning, and memory. Through these pathways, KD contributes to long-term neural adaptability and resilience.

Ketogenic Diet in Epilepsy

1. Historical Background

The ketogenic diet has a long-established history in epilepsy management, dating back to the 1920s when it was introduced as a dietary therapy for children with refractory seizures. Before the advent of antiepileptic drugs (AEDs), KD was one of the primary treatments for epilepsy and demonstrated remarkable efficacy in reducing seizure frequency (Wheless, 2008) [37]. Even today, despite advancements in pharmacotherapy, KD remains a gold-standard non-pharmacological intervention for drug-resistant epilepsy, particularly in pediatric populations, due to its ability to significantly reduce seizures when medications fail.

2. Clinical Efficacy

Clinical evidence consistently demonstrates that the ketogenic diet (KD) is highly effective in reducing seizure frequency, particularly in pediatric drug-resistant epilepsy. Studies report that approximately 30–60% of children treated with KD achieve at least a 50% reduction in seizure frequency, with a substantial subset achieving complete seizure control (Kossoff & Rho, 2009) [17]. Alternative KD-based approaches such as the Modified Atkins Diet (MAD) and the Low Glycemic Index Treatment (LGIT) have shown comparable therapeutic benefits while offering better tolerability and greater dietary flexibility, making them suitable for long-term adherence in children and adults (Kossoff *et al*., 2013) [18]. KD has also shown strong efficacy in specific epileptic syndromes, including Lennox–Gastaut syndrome, Doose syndrome, and GLUT-1 deficiency, where it is considered a first-line therapy due to its ability to bypass impaired glucose transport and stabilize neuronal metabolism (Klepper & Leiendecker, 2007; Cross, 2013) [2, 16]. Collectively, these findings highlight KD as a powerful non-pharmacological intervention with broad applicability across refractory epilepsy subtypes.

3. Proposed Mechanisms

The therapeutic efficacy of the ketogenic diet (KD) in refractory epilepsy is supported by several complementary mechanisms that influence neuronal stability and excitability. A key mechanism involves enhanced synthesis of gamma-aminobutyric acid (GABA), the brain's primary inhibitory neurotransmitter, which helps counteract excessive neuronal firing characteristic of epileptic seizures (Yudkoff *et al*., 2008) [40]. KD also stabilizes synaptic activity by improving mitochondrial energy production, regulating ion channel function, and reducing excitotoxicity, thereby supporting balanced neuronal signaling (Masino & Rho, 2012) [21]. Additionally, KD modulates the gut microbiota, increasing beneficial bacterial species that influence neuroinflammation and neurotransmitter pathways, resulting in improved seizure control through gut–brain axis interactions (Olson *et al*., 2018) [25]. Because these metabolic, synaptic, and microbial mechanisms work synergistically, KD remains the most well-established dietary intervention for refractory epilepsy compared with other neurological disorders.

Ketogenic Diet in Alzheimer's disease

1. Rationale

In Alzheimer's disease (AD), impaired cerebral glucose metabolism occurs decades before the onset of clinical symptoms, contributing to neuronal energy deficits and cognitive decline. The ketogenic diet provides ketone bodies—β-hydroxybutyrate and acetoacetate—which serve as efficient alternative energy substrates capable of bypassing impaired glucose pathways and restoring neuronal ATP production (Cunnane *et al*., 2016) [3, 4]. This improved metabolic support helps sustain neuronal viability and cognitive function in individuals with AD or mild cognitive impairment.

2. Mechanistic Benefits in AD

KD offers multiple neuroprotective mechanisms relevant to Alzheimer's pathology. Ketone bodies reduce amyloid-β accumulation and aggregation, decrease tau hyperphosphorylation, and enhance mitochondrial respiratory efficiency, all of which help preserve neuronal health (Kashiwaya *et al*., 2013) [15]. KD also lowers oxidative stress and neuroinflammation by enhancing antioxidant defenses and inhibiting inflammatory signaling pathways such as the NLRP3 inflammasome (Newman & Verdin, 2017) [24]. Additionally, ketosis increases brain-derived neurotrophic factor (BDNF) expression, which supports synaptic plasticity and cognitive performance. These combined effects demonstrate the strong potential of KD as a metabolic therapy for AD.

3. Clinical Evidence

Clinical studies demonstrate that both ketogenic diets and medium-chain triglyceride (MCT) supplementation improve cognitive outcomes in individuals with mild cognitive impairment and early-stage Alzheimer's disease. Short-term interventions consistently enhance memory, attention, processing speed, and overall cognitive function by raising circulating ketone levels and enhancing brain energy availability (Henderson *et al*., 2009) [13]. Evidence also suggests genetic influences on responsiveness, with APOE-ε4 negative individuals showing greater cognitive improvement following KD or MCT supplementation (Reger *et al*., 2004) [30]. These findings highlight KD as a promising therapeutic strategy for early cognitive decline.

Ketogenic Diet in Parkinson's disease

The ketogenic diet supports Parkinson's disease (PD) management through its effects on mitochondrial function, oxidative stress, and neuronal survival. By providing ketone bodies as alternative energy substrates, KD enhances mitochondrial ATP production in dopaminergic neurons, which are highly vulnerable to metabolic dysfunction in PD (Phillips *et al*., 2018) [27]. Ketosis also reduces oxidative stress in the substantia nigra and decreases neuroinflammatory signaling, both of which contribute to

dopaminergic neuron degeneration (VanItallie *et al*., 2005) [36]. Small clinical trials report improvements in motor symptoms, including tremor, rigidity, and bradykinesia, suggesting potential therapeutic benefits. Although the evidence remains preliminary and sample sizes are small, KD shows considerable promise as an adjunct metabolic therapy in PD.

Ketogenic Diet in Autism Spectrum Disorder (ASD)

1. Mechanisms

The ketogenic diet may benefit individuals with autism spectrum disorder through several interrelated mechanisms involving the gut–brain axis, inflammation, and neurotransmitter balance. KD alters gut microbiota composition, increasing species associated with improved metabolic and neural signaling, which may positively influence behavior and cognition (Newell *et al*., 2016) [23]. Ketosis also reduces neuroinflammation—an emerging hallmark of ASD—by lowering inflammatory cytokines and oxidative stress in neural tissues (Ruskin *et al*., 2013) [33]. Furthermore, KD restores neurotransmitter homeostasis by enhancing GABAergic signaling and modulating glutamate activity, promoting improved communication, reduced hyperactivity, and more stable behavioral patterns. These combined mechanisms highlight KD's therapeutic potential in ASD management.

2. Evidence

Several small studies indicate improvements in social behavior, communication, and hyperactivity in children with ASD following KD or MCT-based regimens.

Ketogenic Diet in Migraine

Ketosis stabilizes neuronal excitability and reduces cortical spreading depression—the primary mechanism behind migraine aura. Clinical trials show KD reduces migraine frequency and severity, particularly in chronic migraine sufferers.

Ketogenic Diet in Traumatic Brain Injury (TBI) and Neuroprotection

The ketogenic diet (KD) demonstrates significant neuroprotective potential in traumatic brain injury (TBI) by addressing the acute metabolic crisis that follows neuronal trauma. After TBI, glucose metabolism becomes impaired, leading to energy deficits that worsen neuronal dysfunction; KD compensates for this by supplying ketone bodies, which serve as efficient alternative substrates that enhance ATP availability to injured neurons (Prins, 2008). In addition to improving cellular energy supply, KD suppresses neuroinflammation by downregulating pro-inflammatory cytokines and inhibiting the activation of the NLRP3 inflammasome, thereby reducing secondary injury cascades (Paoli *et al*., 2014). Ketosis also minimizes oxidative stress through increased mitochondrial efficiency and enhanced antioxidant defenses, which help limit free radical damage in traumatized neural tissue (Greco *et al*., 2016). These combined mechanisms contribute to improved cognitive recovery, including memory, learning, and executive function, as reported across multiple animal studies demonstrating rapid and robust benefits (Prins & Matsumoto, 2014). While human clinical trials remain limited, early findings suggest that KD may offer a promising adjunct therapeutic strategy in TBI management.

Conclusion

The ketogenic diet has evolved from a niche therapy for refractory epilepsy to a promising metabolic treatment for a wide spectrum of neurological disorders. Its diverse mechanisms—including enhanced mitochondrial function, reduced oxidative stress, improved neurotransmission, and anti-inflammatory effects—support its therapeutic potential. While evidence for epilepsy is robust, growing research highlights benefits in Alzheimer's disease, Parkinson's disease, autism, migraine, and traumatic brain injury. Future large-scale, long-duration clinical trials are essential to establish standardized protocols, identify optimal patient populations, and clarify long-term safety. Nonetheless, KD represents a powerful, non-pharmacological, and metabolically targeted approach for improving neurological health.

Table 1: Therapeutic Role of the Ketogenic Diet in Neurological Disorders: A Summary of Evidence from Previously Published Studies

Neurological disorder	Key therapeutic findings from published studies	Proposed mechanisms	Level of evidence (summary)	Citation
Drug-resistant epilepsy (children & adolescents)	KD significantly reduces seizure frequency; many patients achieve ≥50% seizure reduction and some become seizure-free in paediatric cohorts; KD is an established option for drug-resistant epilepsy.	Metabolic shift to ketone bodies (stable brain energy), altered neurotransmitter balance (GABA↑), reduced neuronal excitability, anti-inflammatory effects.	Strong (esp. pediatric): multiple RCTs, meta-analyses and systematic reviews support efficacy.	Desli *et al*., 2022 [5]
Infant epilepsy (including GLUT1, refractory infantile epilepsies)	KD and variants (classical, modified) are effective and generally tolerable in infants with refractory seizures; special protocols exist (careful monitoring).	Same as above; additionally supplies alternative fuel when glucose transport/metabolism is impaired (e.g., GLUT1 deficiency).	Moderate–strong (growing infant-specific evidence but fewer large RCTs).	Falsaperla *et al*., 2020 [8]
Alzheimer's disease / Mild cognitive impairment (MCI)	Emerging clinical studies and trials report modest improvements in cognition, memory, and brain energy metabolism in some patients (esp. MCI or early AD); evidence is mixed and often short-term.	Ketones provide alternative neuronal fuel, reduce amyloid/tau-related pathology in animal models, improve mitochondrial function and reduce neuroinflammation.	Limited–moderate: promising small RCTs and pilot studies; larger, longer RCTs needed.	Morris and Cummings, 2005 [22]
Parkinson's disease (PD)	Small studies and feasibility trials report symptomatic improvements (motor and non-motor symptoms) and better quality of life in some participants; findings not	Improved mitochondrial function, reduced neuroinflammation, altered dopamine metabolism, improved energy availability to	Limited — pilot trials and small cohorts; more RCTs required.	Kapogiannis & Mattson, 2011 [14]

	yet definitive.	vulnerable neurons.		
Traumatic brain injury (TBI)	Preclinical work shows strong neuroprotection; human clinical work is preliminary but several trials (and trial registrations) are underway to test KD or ketone supplementation for acute/subacute TBI.	Neuroprotection via improved energy metabolism, reduced oxidative stress, dampened inflammation, and stabilized neuronal membranes.	Preclinical→early clinical (robust animal data; human evidence emerging).	Belmonte-Jimeno *et al*., 2023 [1]
Multiple sclerosis (MS)	Small clinical and preclinical studies suggest KD may improve fatigue, quality of life, and inflammatory markers; data are preliminary.	Anti-inflammatory effects, modulating immune cell function and microglial polarization, improved mitochondrial efficiency.	Limited / exploratory.	Pukoli and Vécsei, 2025 [29]
Migraine	Some trials and observational studies report reduced frequency/intensity of migraine attacks with carbohydrate reduction/ketosis, but trial sizes are small.	Stabilization of neuronal excitability and cortical spreading depression threshold; metabolic stabilization.	Limited / promising (small trials).	Torres-Ferrus *et al*., 2020 [35]
Autism spectrum disorder (ASD)	Sparse clinical data and case reports; some reports of behavioral and seizure improvements in ASD with comorbid epilepsy, but systematic evidence is insufficient.	Possible modulation of neurotransmitters, anti-inflammatory effects, microbiome changes.	Very limited / experimental.	Frye *et al*., 2013 [10]

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