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Effects of the ketogenic diet on neurodegenerative diseases and drug-resistant epilepsy – a literature review

Wpływ diety ketogenicznej na choroby neurodegeneracyjne i padaczkę lekooporną – przegląd literatury

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Abstract

The ketogenic diet, a high-fat, low-carbohydrate dietary approach, is increasingly used as a therapeutic strategy for the treatment of drug-resistant epilepsy and neurodegenerative diseases such as Alzheimer's and Parkinson's disease. The primary mechanism of the ketogenic diet is the induction of a state of ketosis, during which the brain begins to use ketone bodies. Not only do ketones replace glucose as a fuel but also exhibit a number of neuroprotective effects, such as reducing oxidative stress, stabilising mitochondrial function, mitigating excitotoxicity, reducing neuroinflammatory signalling pathways, and stimulating autophagy. In drug-resistant epilepsy, the ketogenic diet can significantly reduce seizure frequency, especially in children with metabolic syndromes (glucose transporter type 1 deficiency syndrome, pyruvate dehydrogenase complex deficiency). In Alzheimer's disease, the ketogenic diet improves cerebral metabolism, reduces β -amyloid deposition, and supports cognitive function, especially in the early stages of the disease. In Parkinson's disease, a reduction in non-motor symptoms, improvement in mood and mitochondrial function, and potential modulation of the gut microbiota have been observed. However, it should be noted that the ketogenic diet is not without side effects, which include gastrointestinal disorders, hypoglycaemia, lipid abnormalities, kidney stones, micronutrient deficiencies, and possible stunted growth in children. Contraindications, on the other hand, include metabolic diseases, liver and kidney failure, type 1 diabetes, and pregnancy. In conclusion, the ketogenic diet is a promising non-pharmacological therapeutic option in neurology. However, its use requires individual assessment, specialised supervision, and further multicentre studies to confirm long-term efficacy and safety.

Keywords: ketogenic diet, drug-resistant epilepsy, neurodegenerative diseases

Streszczenie

Dieta ketogeniczna, będąca dietą wysokotłuszczową i niskowęglowodanową, znajduje coraz szersze zastosowanie jako strategia terapeutyczna w leczeniu padaczki lekoopornej i chorób neurodegeneracyjnych, takich jak choroby Alzheimera i Parkinsona. Podstawą działania diety ketogenicznej jest indukcja stanu ketozy, podczas którego mózg zaczyna wykorzystywać ciała ketonowe. Ketony nie tylko zastępują glukozę jako paliwo, ale wykazują też wiele działań neuroprotekcyjnych, takich jak redukcja stresu oksydacyjnego, stabilizacja funkcji mitochondriów, zmniejszenie ekscytotoksyczności, ograniczenie neurozapalnych szlaków sygnałowych oraz stymulacja autofagii. W padaczce lekoopornej dieta ketogeniczna może znacząco zmniejszyć częstość napadów, zwłaszcza u dzieci z zespołami metabolicznymi (zespołem niedoboru transportera glukozy typu 1, niedoborem kompleksu dehydrogenazy pirogronianowej). W chorobie Alzheimera dieta ketogeniczna poprawia metabolizm mózgowy, ogranicza odkładanie β -amyloidu i wspiera funkcje poznawcze, zwłaszcza w początkowych stadiach

choroby. W chorobie Parkinsona zaobserwowano zmniejszenie objawów niemotorycznych, poprawę nastroju i funkcji mitochondrialnej oraz potencjalny wpływ na mikrobiotę jelitową. Należy jednak zaznaczyć, że dieta ketogeniczna nie jest pozbawiona działań niepożądanych, do których należą zaburzenia żołądkowo-jelitowe, hipoglikemia, zaburzenia lipidowe, kamica nerkowa, niedobory mikroelementów i możliwe zahamowanie wzrostu u dzieci. Do przeciwwskazań do jej stosowania należą między innymi choroby metaboliczne, niewydolność wątroby i nerek, cukrzyca typu 1 oraz ciąża. Podsumowując, dieta ketogeniczna stanowi obiecującą, nefarmakologiczną opcję terapeutyczną w neurologii. Jej stosowanie wymaga jednak indywidualnej kwalifikacji, specjalistycznego nadzoru oraz dalszych, wieloośrodkowych badań potwierdzających długoterminową skuteczność i bezpieczeństwo tej interwencji.

Słowa kluczowe: dieta ketogeniczna, padaczka lekooporna, choroby neurodegeneracyjne

INTRODUCTION

Neurological diseases, including drug-resistant epilepsy (DRE) and neurodegenerative disorders such as Alzheimer's disease (AD) and Parkinson's disease (PD), represent major clinical challenges worldwide. Despite advances in diagnosis and treatment, effective methods to completely cure or halt the progression of these conditions remain lacking (Bohnen et al., 2023). A particular challenge remains the group of patients with refractory epilepsy, for whom conventional therapeutic methods are ineffective. In the face of these difficulties, there is growing interest in nutritional interventions, among which the ketogenic diet (KD) is gaining particular prominence (Martin-McGill et al., 2020). The KD, by significantly reducing carbohydrate intake and increasing fat supply, leads to a state of ketosis in which ketone bodies become the primary source of energy for the brain. It has been documented that ketones, such as β -hydroxybutyrate or acetoacetate, exhibit a number of neuroprotective effects in addition to their energy functions (Maalouf et al., 2009). These include improved mitochondrial function, reduced oxidative stress, modulation of the immune and inflammatory systems, regulation of glutamate-GABAergic neurotransmission, and effects on autophagy and the gut microbiota (Gasior et al., 2006; Santangelo et al., 2023). The KD is particularly effective in the treatment of DRE, reducing seizure frequency in both children and adults (Manral et al., 2023; Yilmaz et al., 2022). Promising results are also obtained in the context of neurodegeneration, where ketogenic interventions may slow down degenerative processes in the early stages of AD and PD (Bohnen et al., 2023). The aim of this paper is to analyse in detail the current scientific evidence on the mechanisms of action of the KD and to evaluate its application in the treatment of DRE and neurodegenerative disorders.

MATERIALS AND METHODS

This paper reviews the available scientific literature analysing the effects of the KD on the development of neurodegenerative diseases and DRE. The most recent clinical trials, systematic reviews, and meta-analyses published in

recognised medical journals between 2006 and 2025 were included.

ANALYSIS OF THE LITERATURE

The ketogenic diet – general information, types

The KD is a high-fat, low-carbohydrate diet leading to ketogenesis and an increase in ketone bodies, which provide an alternative energy source for the brain. In addition to its use in DRE, it shows potential neuroprotective effects in AD and PD (Bohnen et al., 2023).

The classic KD provides approximately 90% of energy from fat, with minimal carbohydrate intake and moderate protein supply, facilitating rapid induction of ketosis. A diet enriched with medium-chain triglycerides (MCTs) allows increased production of ketone bodies with less carbohydrate restriction, due to the rapid absorption and efficient metabolism of fatty acids with chain lengths of 6 to 12 carbon atoms. The modified Atkins diet (MAD) limits carbohydrates to 10–20 g per day with fewer restrictions on other macronutrients, which may enhance adherence. Low-glycaemic index treatment (LGIT) reduces the glycaemic load of the diet, allowing moderate ketosis to be maintained with milder carbohydrate restriction. These options enable the KD to be tailored to individual clinical needs and patient tolerance (Tao et al., 2022).

Mechanisms of action of the ketogenic diet in the brain

The KD is an alternative approach for managing neurodegenerative diseases and DRE. Its efficacy stems from a number of molecular and cellular mechanisms that affect brain metabolism, mitochondrial function, neurotransmitter balance, and the inflammatory response. The key mechanisms of action of the KD in the brain are outlined below.

Ketones as an alternative energy source for neurons

Adherence to the KD mimics the physiological metabolic changes that occur under conditions of limited glucose

availability, such as starvation. Under these conditions, the body is forced to use fats as the main energy source, leading to increased production of the ketone bodies β -hydroxybutyrate (β -HB), acetoacetate, and acetone. β -HB provides more ATP per molecule than glucose, making it a more efficient energy source for tissues with high energy requirements, such as the brain. Acetoacetate is one of the precursors for the synthesis of brain structural lipids, including sphingolipids and phospholipids, which are necessary for the proper formation of myelin sheaths and neuronal cell membranes. In addition, ketones efficiently cross the blood-brain barrier via diffusion or monocarboxylate transporters activated in the ketosis state, allowing rapid and efficient delivery of energy and anabolic substrates to the developing brain. These properties underlie the use of ketone bodies as part of the supportive treatment of neurodegenerative disorders (Gasior et al., 2006).

Reduction of oxidative stress and excitotoxicity

Ketone bodies decrease the activity of mitochondrial respiratory chain complex I, leading to reduced production of reactive oxygen species (ROS). This decrease in ROS reduces oxidative stress in nerve cells, thereby protecting protein, lipids, and DNA structures from damage. Protection from oxidative damage increases the survival and functionality of neurons, particularly those exposed to calcium overload and excitotoxicity. In addition, ketones can enhance the expression of uncoupling proteins, which reduce the mitochondrial membrane potential and thus further limit free radical generation. These mechanisms improve mitochondrial stability and increase neuronal resistance to damaging factors such as ischaemia, injury, or toxins. Thanks to these properties, ketones are used as a therapeutic adjunct in the treatment of neurodegenerative diseases, including DRE, AD, and PD. Ketogenic therapies support brain function under conditions of cellular stress, prolonging the metabolic activity of neurons and slowing down their degeneration (Hasan-Olive et al., 2019; Maalouf et al., 2009).

Modulation of inflammatory pathways (NLRP3, TNF- α)

Following the KD results in decreased activation of microglia in the brain, which reduces the production of pro-inflammatory cytokines, such as IL-1 β , IL-6, TNF- α , and nitric oxide, leading to the inhibition of chronic neuroinflammation. Reducing the chronic activation of M1-type microglia and A1-type astrocytes prevents neurotoxic neuronal damage and promotes neuronal survival. The increase in ketone bodies promotes the transition of glial cells into anti-inflammatory phenotypes (M2 and A2), which release neuroprotective cytokines such as IL-10 and TGF- β . This helps restore homeostasis in the neuronal environment, slowing down neurodegenerative processes. The KD also modulates systemic inflammatory factors, reducing peripheral immune system activation, which can otherwise exacerbate inflammation in the central nervous system (Jiang et al., 2022).

Regulation of autophagy

The KD, by increasing β -hydroxybutyrate levels, stimulates autophagy in the brain, leading to the removal of damaged proteins and organelles, and reducing inflammation, which has a neuroprotective effect. Since β -HB inhibits the mTORC1 pathway and activates sirtuin-1 and HIF-1, macroautophagy is upregulated, promoting more efficient clearance of neuronal cells. Additionally, β -HB promotes chaperone-mediated autophagy, which enhances the removal of oxidised and damaged proteins, reducing neurotoxicity. Consequently, these processes may slow the progression of neurodegenerative diseases, such as AD, by reducing the deposition of toxic β -amyloid proteins (Jiang et al., 2022).

Increased GABA production and decreased glutamate activity – antiepileptic effects

The KD may have antiepileptic effects by putting the brain into a state of ketosis, which increases the tricarboxylic acid cycle. This results in increased glutamine production in astrocytes, which capture excess glutamate from the extracellular space and convert it into non-toxic glutamine. The process prevents overstimulation of glutamate receptors and protects neurons from excitotoxicity, reducing epileptic seizures.

In addition, increased levels of acetyl-CoA promote increased availability of glutamate for conversion to GABA, thereby increasing the activity of the anti-excitatory neuronal system. Together, these metabolic changes contribute to stabilising brain function and reducing seizure frequency in individuals with epilepsy (Yudkoff et al., 2008).

Effects of the ketogenic diet on the gut microbiome

The KD modifies the composition of the gut microbiota by increasing the abundance of bacteria with anti-inflammatory properties, such as *Akkermansia muciniphila*, *Parabacteroides*, and *Lactobacillus*, while reducing populations of pro-inflammatory microorganisms from the genera *Desulfovibrio* and *Proteobacteria*. These changes contribute to a reduction in inflammation and increased production of fermentative metabolites, particularly short-chain fatty acids (SCFAs), especially by bacteria in the *Firmicutes* and *Akkermansia* families. SCFAs help maintain a state of ketosis, which contributes to the diet's anti-convulsant and neuroprotective effects. However, the KD is not exclusively associated with positive effects, as a decrease in the abundance of *Bifidobacterium*, bacteria considered beneficial for human health, has been observed. The variation in the composition of the gut microbiota in patients responding well and poorly to the KD suggests that the individual bacterial profile may determine the efficacy of the therapy, giving it potential prognostic significance. The exacerbation of neurological symptoms after antibiotic therapy and their subsequent improvement after restoration of the gut microbiota suggest an important role

of the microbiota in modulating the course of epilepsy. Thus, the mechanisms of action of KD extend beyond classical metabolic effects, such as an increase in GABA, to include effects on the gut-brain axis via bacterial metabolites (Santangelo et al., 2023).

Ketogenic diet and drug-resistant epilepsy

DRE is defined as the failure to control seizures with at least two appropriately selected anti-epileptic drugs. It is estimated to affect approximately 20–30% of patients with epilepsy. An effective non-pharmacological therapeutic alternative is the KD, especially in patients with genetically confirmed epilepsy syndromes (Martin-McGill et al., 2020).

The anticonvulsant effects of the KD include improved mitochondrial function, increased GABA activity, reduced neuronal excitability, and anti-inflammatory effects (Jiang et al., 2022).

The KD is the treatment of choice for glucose transporter type 1 deficiency syndrome (GLUT1DS) and pyruvate dehydrogenase complex deficiency (PDCD), as it bypasses metabolic defects that prevent the efficient use of glucose as an energy source for the brain. However, it is important to note that the KD is contraindicated in patients with pyruvate carboxylase defects and *SLC22A5* mutations, due to the risk of severe metabolic decompensation. For this reason, qualification for KD therapy should include a detailed metabolic and genetic assessment (Na et al., 2025).

The efficacy of the KD in the paediatric population is confirmed by a prospective study involving 91 children with DRE who were treated with the KD for at least 12 months. The effect was a reduction in seizures to $\geq 50\%$ in 70.3% of patients, of whom 35.2% achieved complete seizure remission (Yilmaz et al., 2022).

In the adult population with DRE, the efficacy of the KD is supported by both randomised trials (Manral et al., 2023) and a meta-analysis (Manral et al., 2024), demonstrating a correspondingly significant reduction in seizure frequency $\geq 50\%$ and improvement in 25–34% of patients, with a more favourable therapeutic profile for the MAD.

Preliminary reports also indicate a potential benefit of the KD in epileptic encephalopathies such as Lafora body disease, Rett syndrome, Landau-Kleffner syndrome, and subacute sclerosing panencephalitis, expanding its possible therapeutic applications (Kossoff et al., 2018).

The response to KD treatment is modified by a number of clinical and biological factors, which may determine its efficacy in the treatment of DRE. Retrospective studies have shown that later age of onset of epileptic seizures, female sex, higher fat-to-protein and carbohydrate ratios, and omission of the pre-fasting phase increase the likelihood of achieving $\geq 50\%$ seizure reduction (Agarwal et al., 2017; Na et al., 2025).

The gut microbiome is also emerging as an important factor. The presence of bacteria, such as *Akkermansia muciniphila* and *Parabacteroides*, may correlate with the anticonvulsant

effect of the KD, suggesting a role for the microbiota as a potential predictive biomarker of treatment efficacy. Therefore, individual genetic characteristics and the composition of the gut microbiota should be taken into account when adjusting dietary therapy for patients with DRE (Olson et al., 2018).

Side effects and safety of long-term use

Despite its proven efficacy in the treatment of DRE, the KD carries a risk of side effects, especially during the initial adaptation phase.

Among the most common are gastrointestinal symptoms such as diarrhoea, constipation, nausea, vomiting, and gastro-oesophageal reflux, which are usually alleviated with symptomatic treatment. Lipid profile abnormalities and hypoglycaemia, especially after a previous fast, are also common and can lead to neurological symptoms.

Prolonged use of the KD can lead to stunted growth rates, especially in younger children, and carnitine, selenium and potassium deficiencies.

In addition, prolonged use of the KD is associated with an increased risk of nephrolithiasis, but prophylactic use of potassium citrate (2 mmol/kg/day) significantly reduces this risk by counteracting metabolic acidosis and preventing the accumulation of calcium salts in the urinary tract.

Rare adverse effects (<0.5%) include pancreatitis, vascular changes, and cardiac abnormalities, hyperuricaemia, hypomagnesaemia, hyponatraemia, hypoproteinaemia, hepatitis, and metabolic acidosis.

Given the risk of adverse effects, the International Ketogenic Diet Study Group recommends a comprehensive clinical and biochemical assessment before starting treatment and systematic monitoring during therapy. Prior to the introduction of the KD, the following should be performed: complete blood count with platelets, electrolytes (with bicarbonate, calcium, total protein), liver and kidney function tests (albumin, creatinine, urea nitrogen), lipid profile, acylcarnitine profile, vitamin D level, and general urine examination. In cases of diagnostic ambiguity, urinary amino acid and organic acid analyses should be performed. In addition, levels of free and total carnitine, selenium, beta-hydroxybutyrate, zinc, copper, and renal indices (calcium, urinary creatinine) should be monitored during treatment.

Most adverse effects subside when the KD is discontinued. Long-term complications, such as kidney stones, bone fractures, or cardiovascular disease, are no more frequent than in the general population, except in patients with pre-existing risk factors (Guerreiro et al., 2024; Wells et al., 2020).

Ketogenic diet in neurodegenerative diseases

Alzheimer's disease

AD is the most common form of dementia in the elderly population. Key pathophysiological mechanisms of AD

Side effect	Possible causes	Possible mitigation/prevention strategies
Nausea, vomiting, diarrhoea, constipation, abdominal pain	Sudden dietary change, excess fat intake	Gradual introduction of KD, selection of easily digestible fats, increased intake of fibre and fluids
Headaches, fatigue, irritability, dehydration	Adaptation to ketosis, electrolyte loss	Electrolyte supplementation (Na, K, Mg), adequate hydration, adaptation period
Hypoglycaemia, acidosis, pancreatitis, hepatitis	Excessive carbohydrate restriction, comorbidities	Continuous glucose monitoring, metabolic status control, careful patient selection for KD
Reduced appetite	Effect of ketone bodies on hunger centre	Desirable in obesity treatment, contraindicated in individuals at risk of malnutrition (e.g. oncology patients)
Vitamin and mineral deficiencies (B, Ca, Se, Zn, Mg)	Limited dietary diversity	Supplementation (e.g. multivitamins, omega-3, minerals), proper product selection
Increased LDL, total cholesterol, ApoB	High intake of saturated fats	Increase intake of unsaturated fats, individual cardiovascular risk assessment
Decreased bone mineral density	Calcium deficiency	Calcium and vitamin D supplementation, regular bone density monitoring
Increased risk of colorectal cancer	Fiber deficiency	Inclusion of fibre sources (e.g. low-carb vegetables, fibre supplements)
Kidney stones	Increased excretion of calcium and acids	High fluid intake, kidney function monitoring, possible potassium citrate supplementation
Optic neuropathy, anaemia, cardiomyopathy	Vitamin and nutrient deficiencies, long-term restrictions	Regular blood parameter monitoring, appropriate supplementation
Ketoacidosis (especially in type 1 diabetes or breastfeeding women)	Excessive ketogenesis without glycaemic control	Exclusion of contraindications prior to diet initiation, close medical supervision

Tab. 1. Most common side effects of the ketogenic diet and possible strategies to minimise them. Own elaboration based on literature (Watanabe et al., 2020)

include β -amyloid ($A\beta$) accumulation, hyperphosphorylation of tau protein, neurotoxic damage, and impaired glucose metabolism in the brain resulting from, among other factors, a deficiency of the GLUT1 transporter. In this context, the KD, by providing an alternative energy source in the form of ketone bodies, may compensate for glucose hypometabolism, improve mitochondrial function, reduce oxidative stress and inflammation, and promote neurotransmission and neuronal survival. The clinical response to ketogenic therapy depends on several factors including the severity of the disease, genetic status, and the efficacy of ketosis induction, which determines the neuroprotective impact of ketones (Oliveira et al., 2023).

Other studies focusing on the APO ϵ 4 genotype stage have shown that patients carrying the APO ϵ 4 allele had a poorer clinical response and required a more restrictive KD regimen or a combination of glycaemic restriction and direct supplementation with ketone esters than patients without this mutation to achieve an improvement in cognitive function. In some patients, the lack of efficacy may have been due to insufficient ketosis, inadequate supplement formulation, or an advanced stage of the disease in which the prevalence of degenerative mechanisms limits the effect of metabolic interventions (Bohnen et al., 2023).

The KD may also have an effect on AD biomarkers. Studies have shown that adherence to the KD improves clearance of β -amyloid from the brain and reduces its deposition as plaques. Such an effect indicates a potential neuroprotective effect of this diet, which may slow down the progression of AD or delay its development in at-risk individuals. The increase in $A\beta$ concentration in cerebrospinal fluid indicates that it may promote normal metabolism and transport of $A\beta$, counteracting its toxic accumulation in the central nervous system (Ramezani et al., 2023).

The KD, by providing an alternative energy source, may be a promising therapeutic strategy for the treatment of AD, especially in the early stages of the disease when glucose hypometabolism is still reversible. However, further clinical trials are needed to fully evaluate the efficacy and safety of this intervention in the long-term treatment of AD.

Parkinson's disease

PD is a progressive neurodegenerative disorder whose pathophysiology includes oxidative stress and the degeneration of dopaminergic neurons within the substantia nigra. The KD, by inducing a state of ketosis, provides an alternative energy source for neurons in the form of ketone bodies, such as β -hydroxybutyrate, which can be utilised by neurons in place of glucose. Preclinical studies indicate that the KD may protect dopaminergic neurons from neurotoxicity by increasing antioxidant levels and reducing inflammation (Bohnen et al., 2023). In addition, the KD modulates the gut microbiota, potentially affecting brain metabolism and cognitive function (Jiang et al., 2022; Santangelo et al., 2023). Ketogenic interventions, such as high-fat dietary regimens or MCT supplementation, may contribute to the improvement of selected non-motor symptoms in patients with PD, including reduced fatigue, improved sleep quality, and mood stabilisation, although the available evidence is still limited (Bohnen et al., 2023).

In the case report presented by Tidman (2022), KD implementation led to a marked reduction in depressive and anxiety symptoms, which was accompanied by favourable metabolic changes – a reduction in glucose, insulin, and HbA_{1c} levels and a reduction in body weight. These improvements in metabolic parameters may have influenced the patient's overall somatic state, which indirectly translated into a long-term improvement in quality of life (Tidman, 2022).

Side effects and contraindications to the ketogenic diet

Side effects

Given the potential side effects associated with the KD, Tab. 1 outlines the most common side effects and possible strategies to minimise or prevent them (Malinowska and Żendzian-Piotrowska, 2024).

A careful assessment of the patient – taking into account metabolic and organ function status and pharmacotherapy history – is essential before implementing the KD. The diet should only be followed under the supervision of a specialist, who will carry out appropriate tests and ensure that parameters such as electrolytes, liver function, kidney function, and lipid metabolism are monitored.

Contraindications

The introduction of the KD is not recommended in patients with liver failure, as it may exacerbate existing fat metabolism disorders and lead to the deterioration of organ function. Also, in patients with advanced kidney disease, there is a risk of acid-base imbalances and difficulty in eliminating ketones. In patients with type 1 diabetes, the KD without close medical supervision may increase the risk of ketoacidosis and severe hypoglycaemia, despite potential metabolic benefits. Concomitant intake of SGLT-2 inhibitors in type 2 diabetes may further predispose patients to the development of euglycaemic ketoacidosis. The diet is also contraindicated in rare disorders of fatty acid metabolism, such as carnitine or β -oxidation enzyme deficiency, due to the potential for hypoglycaemia and impaired consciousness. Pregnant and lactating women should avoid the KD, as there are no sufficient data to support its safety during these periods. Caution should also be exercised in patients with active infections, malignancies (especially melanoma and renal cell carcinoma), severe mental illness, or substance use disorders, as these may affect the body's metabolic tolerance. In patients with recent cardiovascular incidents or cardiac arrhythmias, the use of the KD should be carefully reviewed, especially in the context of metabolic stress related to treatment or surgery (Watanabe et al., 2020).

CONCLUSION

The KD shows multidirectional therapeutic effects in neurological disorders, including modification of energy metabolism, reduction of oxidative stress and inflammation, improvement of mitochondrial function, and regulation of neurotransmitters and the gut microbiota. In the treatment of DRE, the KD is an effective non-pharmacological therapy, especially in children, but also increasingly in adults. For neurodegenerative diseases such as AD and PD, promising results have been reported, although further clinical trials are needed to confirm the long-term efficacy and safety of this intervention. Due to the potential for side effects, the implementation of the KD requires careful monitoring and ongoing follow-up by qualified healthcare professionals.

Conflict of interest

The authors do not report any financial or personal connections with other persons or organisations which might negatively affect the content of this publication and/or claim authorship rights to this publication.

Author contribution

Original concept of study; collection, recording and/or compilation of data; analysis and interpretation of data; writing of manuscript; critical review of manuscript; final approval of manuscript: PSK, WK.

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