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Effects of Ketogenic Diet on Brain Functions and Behaviors

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Abstract

The ketogenic diet, a high-fat, low-carb diet, is being researched for treating conditions like epilepsy, cancer, neurological conditions, metabolic syndromes, and cognitive impairments. Ketone salts and exogenous ketone salt supplementation are also being studied for treating neurologic conditions like gliomas, Alzheimer's, and seizure disorders. The diet may also improve depression and brain health by reducing inflammation and oxidative stress. KDs have neuroprotective properties in epilepsy, induction of aging, neurodegenerative and dementia in animals. Treatment with ketosis decreases the induced seizures and anxiety-like behaviors through handling, according to Morris water maze tests. Mood and cognitive function are positively impacted by the ketogenic diet. Blood glucose levels are stabilized and the inflammatory response is improved by a ketogenic diet. The ketogenic diet has a significant impact on body weight as well as hunger reduction and satiety stimulation. Improved problem-solving abilities, quicker thinking, and better recall. Long-term brain health and a lower chance of cognitive decline brought on by ageing.

Keywords: Ketogenic Diet, Blood Glucose, Inflammation, Neurodegenerative Disorders

1 Introduction

In nutritional research, the ketogenic diet (KD) is undergoing a "renaissance." KD is not frequently used in therapeutic settings, even though it has been shown to be effective in treating pharmacologically resistant childhood epilepsy (Walczyk & Wick, 2017). KD is useful in treating pyruvate dehydrogenase deficiency (PHDH) and glucose transporter 1 deficiency syndrome (Glut1DS), two conditions affecting the brain's energy metabolism, in addition to childhood epilepsy. Each of the aforementioned conditions have neurobiological characteristics in common with epilepsy (Kossoff et al., 2018). The ketogenic diet is also used to treat cancer, neurological conditions like Parkinson's and Alzheimer's disease, metabolic syndromes, and cognitive impairments (Cavaleri & Bashar, 2018). The number of yearly publications on the ketogenic diet has increased by 300% over the past ten years, according to a PubMed search for the term (Grigolon et al., 2020). KD is a low-carb dietary plan with a high-fat content and a specific ratio of macronutrients containing 90% fat, 8% protein, and 2% of carbs (McDonald & Cervenka, 2018). The suggested macronutrient ratio of the diets that target healthy individuals is 45-65% carbohydrates, 10-15% protein, and 20-30% fat. KD causes ketosis through the beta-oxidation of

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fatty acids of serum that give the brain energy molecules (Cavaleri & Bashar, 2018). It involves the necessary nutrients, dietary supplements without carbohydrates, minerals (such as calcium) supplements, consultation of specialists, and laboratory control, which is now recommended (Grigolon et al., 2020). KDs as well as exogenous ketone salt supplementation have been researched in a number of preclinical trials for the therapy of particular neurologic conditions, such as gliomas, Alzheimer's disease, and seizure disorders. When used isolated, KDs reduce tumor weight, serum insulin-like growth factor, and serum glucose in rodent glioma models (McDonald & Cervenka, 2018). A moderate protein proportion (6–15%), typically adapted to the patient's needs, is what determines the standard KD (Bostock et al., 2020). Lipids make up 80–90% of the diet, with carbohydrates being substantially decreased (5–10%). Changes in cellular energy metabolism brought on by these particular dietary needs may have a major impact on oxidation-reduction reactions and inflammation. In a KD, adipocytes release glycerol, triacylglycerols (TAG), and free fatty acids (FFA) as a result of a decline in insulin levels and an increase in adrenaline concentration. After being stimulated, they are utilized for synthesis of ketone bodies (KBs), first as acetoacetate (AcAc), which is then transformed into acetone and beta-hydroxybutyrate (Barry et al., 2018; Rubio et al., 2025). In a supplemental treatment for this neurodegenerative condition, the ketogenic diet has been shown to relieve both cognitive and motor symptoms (Frank & Scolnick, 2024). Five clinical studies have demonstrated the important benefits of the ketogenic diet, including a significant correlation between a low-carb, high-fat diet and a reduction in the severity of Parkinson's disease, as well as improvements in cognitive function, including a significant decrease in body weight and an improvement in voice quality as determined by the Voice Handicap index (Rubio et al., 2025). For a number of years, drug-resistant epilepsy has been treated with a ketogenic diet (KD) therapy. According to current research, KD therapy may provide neuroprotective and cognitive-enhancing benefits for both healthy people and those with non-epileptic illnesses (IJff et al., 2016). KD may therefore be a viable non-invasive nutritional therapeutic option for challenging-to-manage ailments like mood disorders or neurodegenerative diseases (Chinna-Meyyappan et al., 2023). Research of rats has indicated that KD elevates the concentration of the inhibitory neurotransmitter GABA, thus reducing neurological hyperactivity and preventing seizures. One reliable method of treating epilepsy is the ketogenic diet (Jiang et al., 2022; Parent et al., 2022). Over the last seven years, randomized controlled trials and clinical trials have demonstrated the efficacy of the ketogenic diet in the therapy of drug-resistant epilepsy (Operto et al., 2020). Research has shown that both adults and children may see improvements in their risk of seizures, and may even achieve seizure-free status (Kasperek et al., 2022). Additionally, to being utilized as an alternative medicine for numerous other illnesses, KD has been used for than a century to treat seizures in children caused by refractory epilepsy. Despite having a high fat content, KD has been shown in recent studies to improve blood glucose levels in diabetic patients and reduce body weight in obese patients (Gumus, H et al., 2022). The KDs have neuroprotective properties in animal models of epilepsy, aging, dementia and neurodegenerative diseases. Two of the four studies (4548) that utilized animal models of traumatic brain injury reported that KD has beneficial effects on cognition, although one study did not (49). In comparison to the injured animals fed the control diet, young adults (4748-years) and male adolescents (4547-years) fed kD reportedly exhibited significantly improved spatial memory and trauma damage recovery in the variety of behavioral tests (MWM, NOR, or Y-maze) (Grabowska et al., 2024). Four to ten % of children might suffer at least one critical period over their lifetime, causing seizure disorders the most common neurological ailment in developmental age. Compared to their peers in good health, children with epilepsy are also more likely to experience emotional and behavioral issues (Kasprowska-Liśkiewicz et al., 2017). More precisely, 8–35% of instances of depression and 15–36% of cases of anxiety disorders are documented in teenagers (Matricardi et al., 2020). Depression and anxiety are more common in clinical studies (2736 and 2029% respectively) as opposed to population-based studies (5 and 713, respectively). Nevertheless, the risk of psychiatric comorbidity, including tic disorders,

oppositional-provocative disorder, attention deficit and impulsivity or hyperactivity disorder, anxiety and depressive disorders is much higher in children with newly diagnosed epilepsy than in healthy controls (Coppola, G. et al., 2020). It is unclear how exactly the ketogenic diet acts. The changes in mitochondrial activity, reduction in the mammalian target of rapamycin, the impact of ketone bodies on the brain activity and release of neurotransmitters, the anti-epileptic effects of fatty acids, or stabilization of glucose are among the suggested mechanisms. Ketones have the potential to increase membrane potential which may lead to hyperpolarization causing γ-aminobutyric release, and decreased flow of other neurotransmitters such as glutamate, norepinephrine or adenosine (Matricardi et al., 2020).

2 Behavioral effects of KD Treatment Cognition

The effects of nutrition ketosis include the promotion of mitochondrial activity and mitigation of oxidative stress and neuroinflammation. All these processes influence neurocognitive functioning and brain health to a considerable extent. The neuroprotective properties of KDs had been demonstrated in animal models of epilepsy, aging, dementia, and neurodegenerative disorders (Masood et al., 2022). Cognition encompasses many facets of intellectual activity and is a very complex concept. Therefore, findings from animal research may only capture a portion of this term's values (Annamaraju et al., 2022). Morris Water Maze (MWM) is the most popular cognitive impairment behavioral test. MWM model is used to test spatial learning and long-term memory by recording and monitoring the escape latency, thigmotaxis duration, distance covered, and the velocity during which the rodents are supposed to find the escape route to the platform by remembering the visual stimuli that mark where the platform is located. The unconventional object recognition test (NOR) is another popular assessment tool used for assessing memory and learning, especially recognition memory (Pietrzak et al., 2022). With respect to recognized things (such as blocks or balls), the NOR test makes use of an individual's instinctive desire to interact with unknown objects (Masood et al., 2022). Variations of maze tasks, such as the Y-maze, T-maze, V-maze, Hebb Williams Maze, or Barnes Maze, are used in other behavioral tests in animal research. The rats' learning, spatial learning, and short- and long-term memory can all be evaluated using the previously mentioned methods (Majid et al., 2022). The most frequent conditions in which KD impairs cognitive abilities are Alzheimer's disease (AD), epilepsy, traumatic brain injury (TBI), and healthy animals, especially in old age. Numerous studies on ageing, lifespan, and health found that KD-fed mice performed better cognitively. Regardless of gender, KD therapy improves cognition throughout life when given at both young ages (4 months) and old ages (20 months). It was linked to improved performance on a cognitive dual task involving working memory as well as the elevated maze alternation challenge. (Gentile et al., 2018) These results demonstrate that the KD could positively affect cognitive performance in both male and female rodents particularly in middle aged and old aged animals after several weeks of treatment and in conjunction with persistent dietary exposure. Nonetheless, the postnatal exposure of laboratory mice to KD at postnatal day (P) 20 or later to a minimum of 5 weeks might lead to adverse outcome on cognition later in life. Miles and Skelton have discovered that exposure of nutritional ketosis in the early life (P21-young adulthood P90) can influence memory and learning (Uppaluri et al., 2022). When treated one and twelve months with KD, the issue of cognitive impairment of female mice in a sleep deprivation-induced AD model was resolved in the APP/PS1 mice study (Yousufzai et al. 2024). Although KD reduced A 240 and 242 by 25% it was insufficient to improve cognitive performances in female mice with the London APP mutation (APP/ V717I) (Kraeuter et al., 2019). Nutritional ketosis did not enhance memory impairment in a variety of experimental conditions in other studies using genetic (APP/PS1, Tg4510) or pharmacological models of AD (Gibson et al., 2015).

2.1 Depressive like Behavior

A KD can possibly alleviate depressive symptoms via the glutamate-glutamine cycle, monoamines levels, and the gamma-aminobutyric acid (GABA) level of neurotransmission. In the meantime,

the food has an effect on the makeup of the gut flora and provides nutrients such as the ω-3 fatty acids, which could help in reducing depression. KD could enhance the dysbiosis of the gut, decrease the production of cytokines, and lower systemic inflammation associated with depression through alterations in gut bacteria and their metabolites (Kong et al., 2022). The metabolic and behavioural evaluation of Dravet mice on KD is another significant piece of evidence supporting the therapeutic potential of dietary ketosis (Khodabakhshi et al., 2020). According to four studies, nutritional ketosis may have a positive effect on depressive-like behaviors as evaluated by the tail suspension test (TST) and the forced swim test (FST), two traditional behavior paradigms used to gauge depression levels. These paradigms also include changes seen in response to acute stress. Depression-related behavior is estimated using the extended immobility in TST and FST. When evaluating behavioral signs of feeling hopeless but not anhedonia, it appears that these models function similarly (Zilberter & Zilberter, 2017). The latest study by Guan et al. reports that KD therapy increased sucrose preference in anhedonia-based SPT and reduced the duration of immobility in TST and FST in constant social defeat stress (R-SDS) and lipopolysaccharide (LPS) depression models. In line with this finding, the most prominent goal of the current work from Gumus et al. was to assess if voluntary exercise during early to late adulthood (between 1 month and 8 months-old, when voluntarily allowed) could effectively reduce depressive-like behaviours in adult male mice fed with a continuous diet of KD (Gumus et al., 2022). This correlated with both a increase in BHB levels and decrease in glucose, insulin or LDL/HDL ratio. Nutritional ketosis may positively affect depressive-like behaviour, according to experimental research findings (Zhu et al., 2022).

2.2 Anxiety-like Behaviors

There are several factors contributing to this including GABA imbalance and increased neuronal activity. The therapeutic effect of KD on anxiety disorders may, at least to some degree, be attributed to the effects of the diet on gut microbiota, intestinal barrier integrity, anti-inflammatory properties, and decreased production of reactive oxygen species (ROS). Zhu et al. (2022) performed a systematic review of the currently available clinical data and meticulously examined the mechanisms underpinning the potential relevance of KDs in anxiety disorders (Brietzke et al., 2025). To evaluate rodent fear, a variety of behavioral analyses have been created. The determination of anxiety in tests such as elevated plus maze (EPM), open field, and dark/light compartment tests is based on the observation of laboratory mice prefer enclosed, dark conditions to open, light ones. “Conflict" tests, such as the Geller-Seifter or Vogel test, are other common types of tests that are mostly used for anxiolytic screening. In these tests, an animal that is hungry or thirsty is given the choice to receive food or water by pressing a knob that can also cause an electric shock (Zhu et al., 2016).

2.3 Nutritional Behaviors

KD is being used more and more to treat obesity in addition to a supplemental treatment for type 2 diabetes (T2DM). Meta-analyses that evaluate the effectiveness of KDs to low-fat diets consistently reveal improved triacylglycerol (TAG), HDL cholesterol, and other cardiometabolic measures, as well as slightly greater weight loss, but also higher LDL cholesterol. Although the exact process determining the effectiveness of weight loss in nutritional ketosis has not been widely agreed on, appetite suppression is thought to represent a major factor (Chinna-Meyyappan et al., 2023). A typical consequence of diet-induced weight loss is a creased feeling of hunger, which gradually impairs patient adherence, threatening the therapy's effectiveness and ultimately encouraging weight gain (Zarifkar et al., 2022). Thus, knowing how a KD reduces appetite can greatly aid in the creation of new medications as well as lifestyle modifications that improve weight loss treatments. Animal models are nearly always used to study the neuroendocrine network that regulates appetite at the molecular and cellular level (Breda et al., 2022). After the period of habituation, mice fed a KD typically consume the same number of calories but less food in grams than those fed normal chow. In addition to several illness models such as the T2DM

model, the AD model in the stress model, the glaucoma model in both males and females, the models of hepatic enzyme abnormalities, and the acute alcohol withdrawal symptoms, that pattern emerged in wild-type animals fed KD for up to two months or more. However, in long-term treatment, feeding with KD frequently leads to enhanced body mass and metabolic health regardless of the comparable caloric consumption. In many trials, mice fed KD ingested more calories than controls fed chow. This was likely due to the fact that weight gain was not increased (Brietzke et al., 2025). A large number of studies conducted in a variety of illness models demonstrate that KD has beneficial impacts on cognition, particularly in models of epilepsy, traumatic brain injury, and aging, as well as in naive animals. According to precisely all of the studies, the KD has little to no impact on behaviors linked to depression (Davis et al., 2021). Most research indicates that the KD therapy has no effect on behaviors related to anxiety. Seldom was it noted that the KD had a negative impact on behaviors related to depression, anxiety, and cognition (Davis et al., 2021).

3 Ketogenic Diet in the Therapy of Migraine

Most common neurological condition is migraine. It affects 12% of the global population, while 1% to 2% of persons worldwide suffer from chronic migraine (CM). 2.5% of people who suffer from acute migraines move on to acquire chronic migraines (Pavón et al., 2021). It is characterized by common, often quite severe paroxysmal headaches. As a result, it is a condition that severely lowers a patient's quality of life and capacity to carry out everyday tasks. Other symptoms, such as psychological disorders, sleep disturbances, or cardiovascular signs, are more likely to occur in migraineurs (Kondziella et al., 2022).

Numerous recent clinical trials have shown that the ketogenic diet has great potential benefits for patients with migraines. Disorders in mitochondrial activity and associated issues with ATP synthesis are thought to be among the potential mechanisms of migraine, even if the precise reasons have not been determined. Since ketone bodies give the brain an alternate source of energy, they appeared to be a possible solution in these situations, where the cause could involve a disruption in the cerebral cells' metabolism that limits their ability to use glucose as fuel. However, significant conclusions can be made by analyzing the actual findings of research conducted on migraine patients rather than the hypothesis. An advantage of a very low-calorie ketogenic diet was demonstrated in a randomized controlled study that contrasted its effects with those of a very low-calorie non-ketogenic diet (Dyńka et al., 2022). Thirty-five migraine and overweight individuals participated in the study. Participants on the ketogenic diet demonstrated a typical decrease of −3.02 migraine attacks and −3.73 migraine days per month when compared to those on the non-ketogenic diet. Only 8.57% of patients in the non-ketogenic group experienced at least a 50% decrease in the mean number of migraine days, but 74.28% of patients in the ketogenic group did. It was proposed that nutrition may be an effective migraine treatment (Dyńka et al., 2022). Additionally, these patients had decreased body mass, fatty tissue mass, and consequently, BMI levels. The positive impact extends beyond weight loss, and the researchers came to the conclusion that the therapeutic action must be supported by additional mechanisms. Another study in 2022 aimed to determine how the ketogenic diet affected treatment-refractory migraine in 31 individuals (in the second trial) and 22 patients (in the first study). Crucially, a low-carbs diet and the ketogenic diet were contrasted (Dyńka et al., 20222).

The frequency of migraine attacks, the severity of the headaches, and the quantity of medications taken were all substantially reduced in the initial group of 22 patients following the ketogenic diet. Conversely, no significant alterations were observed in the group following a low-carbs diet. Similar outcomes were seen in the trial of the second group of 31 patients, demonstrating the efficacy of the ketogenic diet in treating migraines that are resistant to treatment. The synthesis of ketones and their impact on headaches were also found to be related (Dyńka et al., 2022).

2.4 The Range and Mechanisms of the Ketogenic Diet Effect in Neurological Diseases

The ketogenic diet may work through a variety of processes. In a 2021 publication, the researchers thoroughly analyzed 170 studies examining the wide range of its effects on neurological diseases in 14 different aspects, including neuroprotection, neuroplasticity, neuroinflammation, neurotransmitter function, epigenetics, nociception, changes in cell energetics and metabolism, and other aspects (Dynka et al., 2022).

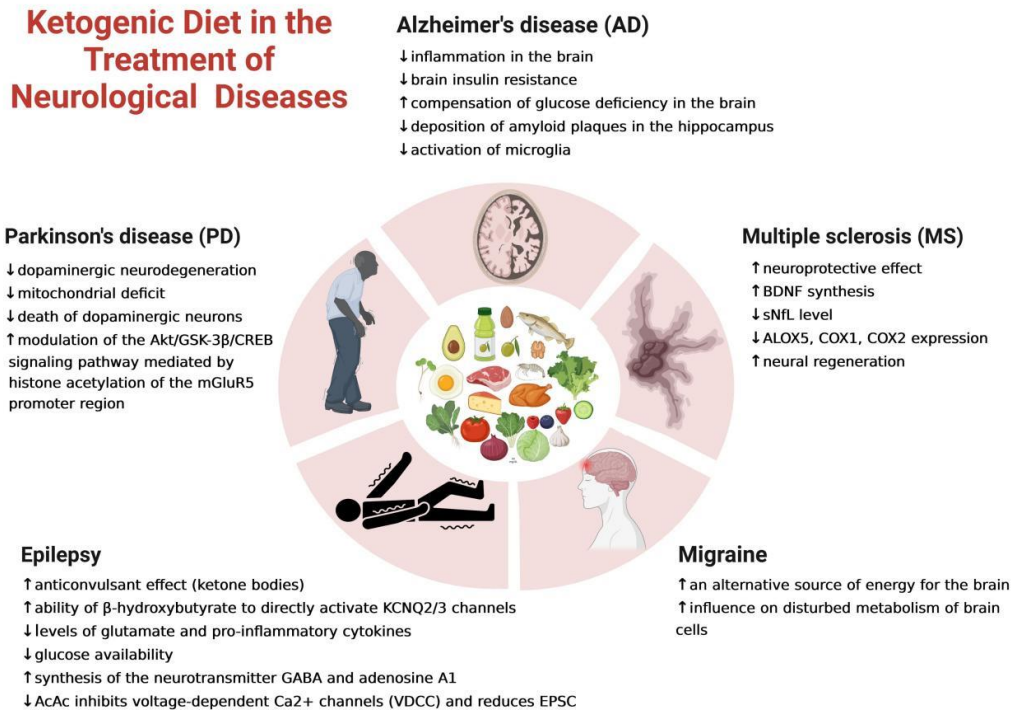

Figure 1: Ketogenic Diet in the Treatment of Neurological Diseases

The fact that the brain can perform effectively using ketone bodies as the energy source is a crucial piece of knowledge. When glucose is not easily accessible, that is often the sole way to provide energy. In fact, as many other body cells, the brain cells have monocarboxylate transporters (MCTs) that facilitate the movement of ketone molecules to produce energy. In 2021, a comprehensive meta-analysis based on 49 animal research from 1979 to 2020 revealed the broad and, first and foremost, positive range of the ketogenic diet effect (Dynka et al., 2022). In actuality, it raises the quantity of neuroprotective factors, such as molecular chaperones, neutrophin-3 (NT-3), glial cell line-derived neurotrophic factor (GDNF), and brain-derived neurotrophic factor (BDNF) (Kumar et al., 2024). Additionally, the deficiency enhances mitochondrial activity, which lowers the generation of reactive oxygen species (ROS) and improves energy production efficiency. It also exhibits anti-inflammatory properties due to, among other things, blocking the production of proinflammatory interleukins (IL-1β, IL-2, IL-4, and IL-6) and tumor necrosis factor alpha (TNFα) and inhibiting the activities of cyclooxygenase-2 (COX-2) and inducible nitric oxide synthase (iNOS). In addition, it lowers the level of transcriptional nuclear factor kappaB (NFκB), an important player in the inflammatory process (Dynka et al., 2022). One of the many possible ways that the ketogenic diet may affect neurological disorders and the nervous system is through its multiple anti-inflammatory effects. Trauma, ischemia, degeneration, or infection are some of the causes of a neurological inflammatory disease (Dynka et al., 2022). The influence of the ketogenic diet on the prevention and treatment of neurological illnesses, particularly

neurodegenerative ones, through a change in intestinal microbiota is another mechanism that is being highlighted more and more in publications. This is because intestinal dysbiosis may have a role in the emergence of many illnesses, but a ketogenic diet is known to have a major impact on the intestinal microbiota's remodeling (Ling et al., 2019). As a result, there is growing interest in the ketogenic diet's possible indirect beneficial impact on neurological disorders by altering gut microbiota (Dynka et al., 2022).

4 Conclusion

A high-fat, low-carb diet called the ketogenic diet is being researched as a treatment for glucose transporter 1 deficiency syndrome, pyruvate dehydrogenase deficiency, and pharmacologically resistant childhood epilepsy. Cancer, neurological disorders, metabolic syndromes, and cognitive impairments can all be effectively treated with it. The diet has seen a 300% increase in yearly publications over the past decade. Ketogenic diets (KDs) have been shown to reduce tumor weight, insulin-like growth factor, and glucose in rodent glioma models. They have also been shown to relieve cognitive and motor symptoms in Parkinson's disease, improve depression symptoms, and enhance mitochondrial function, reducing oxidative stress and neuroinflammation. KDs have shown neuroprotective qualities in animal models. The diet's indirect beneficial effect on intestinal microbiota remodeling is also being studied. The ketogenic diet significantly reduced migraine attacks, severity, and medication intake in 22 patients, compared to no significant changes in a low-carbs group. This suggests ketones' impact on headaches is related to their synthesis Researchers looked at neuroprotection, neuroplasticity, neuroinflammation, neurotransmitter function, epigenetics, nociception, and alterations in cell energetics and metabolism in 170 studies on the impact of the ketogenic diet on neurological disorders. Research indicates that KD has little effect on behaviours linked to depression but has a positive effect on cognition in models of epilepsy, traumatic brain injury, ageing, and naive animals. Research indicates that KD improves cognition in a variety of disease models, particularly in ageing, traumatic brain injury, and epilepsy, as well as in naive animals. It has little to no effect on behaviours related to anxiety or depression.

Author Contributions

Aqib Ishaq, Muhammad Abubakar, Muhammad Soban Mumtaz, and Zoobia Nadeem contributed to the conceptualization, literature search, review and synthesis of evidence concerning ketogenic diets, neurological function, cognitive performance, behavioural outcomes, mood, inflammation, and metabolic effects, organization of the scientific content, interpretation of the published literature, and preparation of the manuscript. All authors contributed to writing—original draft and writing—review and editing. All authors read and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

Conflict of Interest

The authors declare that they have no financial, professional, commercial, institutional, or personal conflicts of interest that could have influenced the content or conclusions of this article.

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Data Availability

No new experimental or clinical dataset was generated or analysed in this study. All information supporting the discussion and conclusions was obtained from the published scientific literature cited in the manuscript.

Informed Consent

Not applicable. The study did not involve the recruitment of human participants or the collection of primary personal or clinical data.

Use of Generative AI

The authors declare that no generative artificial intelligence tools were used to conduct the literature analysis, generate the scientific content, interpret the evidence, or formulate the

conclusions presented in this article.

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