International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

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ARTICLE TITLE	EFFECTIVENESS OF THE KETOGENIC DIET IN TREATING SYMPTOMS OF AUTISM SPECTRUM DISORDER – A SYSTEMATIC REVIEW
ARTICLE INFO	Marta Danuta Cendrowska, Łukasz Brzost, Beata Choromańska, Julia Maszewska, Szymon Milnerowicz, Julia Procyk, Barbara Ponitka, Karolina Stępień, Justyna Berent, Paulina Rzepa, Aleksandra Klukowska, Wiktoria Szumlińska. (2025) Effectiveness of The Ketogenic Diet in Treating Symptoms of Autism Spectrum Disorder – A Systematic Review. *International Journal of Innovative Technologies in Social Science*. 2(46). doi: 10.31435/ijitss.2(46).2025.3401
DOI	https://doi.org/10.31435/ijitss.2(46).2025.3401
RECEIVED	23 May 2025
ACCEPTED	25 June 2025
PUBLISHED	27 June 2025
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EFFECTIVENESS OF THE KETOGENIC DIET IN TREATING SYMPTOMS OF AUTISM SPECTRUM DISORDER – A SYSTEMATIC REVIEW

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ABSTRACT

Introduction: Autism spectrum disorder is a multifactorial condition characterised by challenges in interpersonal engagement, communication deficits and distinct behavioural patterns that deviate from typical developmental norms. While this condition is estimated to affect about 1 in 100 children worldwide, its treatment options are still limited. The purpose of this systematic review is to determine the potential of ketogenic diet - high-fat, low-carbohydrate regimen valued primarily for anticonvulsant qualities - in alleviating autism spectrum disorder clinical signs.
Materials and methods: An extensive review of the literature was performed focusing on publications published since 2017.
Results: Evidence suggests that the ketogenic diet influences metabolic activity of cells, display anti-inflammatory benefits, alter gut microbiota composition, positively influence mitochondrial activity and modulate brain function and behavioural phenotypes via epigenetic pathways. Therefore, ketogenic diet has emerged as a potentially effective therapeutic option in ameliorating ASD symptoms. Additionally, combining ketogenic diet with other therapeutic diets and their beneficial effects seems reasonable and justifiable.
Conclusions: While these initial findings appear encouraging, extended research on larger and more diverse populations is required. Current evidence on the efficacy of ketogenic diet in treating symptoms of autism spectrum disorder is limited by variable study designs, small sample sizes, and frequently suboptimal adherence.

KEYWORDS

Autism Spectrum Disorder, ASD Symptoms, Ketogenic Diet, Dietary Intervention, Diet Therapy

CITATION

Marta Danuta Cendrowska, Łukasz Brzost, Beata Choromańska, Julia Maszewska, Szymon Milnerowicz, Julia Procyk, Barbara Ponitka, Karolina Stępień, Justyna Berent, Paulina Rzepa, Aleksandra Klukowska, Wiktoria Szumlińska. (2025) Effectiveness of The Ketogenic Diet in Treating Symptoms of Autism Spectrum Disorder – A Systematic Review. *International Journal of Innovative Technologies in Social Science*. 2(46). doi: 10.31435/ijitss.2(46).2025.3401

Introduction.

Autism spectrum disorder (ASD) encompasses a wide range of conditions. They are mainly distinguished by difficulties in establishing social relationships and communication. Typical symptoms also include atypical behavioural and activity patterns, such as trouble shifting between tasks, a strong focus on details and unusual responses to sensory stimuli (Patil & Kaple, 2023). ASD comprises a broad spectrum of co-morbidities and complex underlying mechanisms such as mitochondrial dysfunction and oxidative stress (Frye, 2020), gastrointestinal issues (Ristori et al., 2019), immunological disbalance with increased inflammation (Robinson-Agramonte et al., 2022) and rising prevalence of epilepsy (Zarakoviti et al., 2022) and psychological conditions including ADHD, anxiety and depression (Mosner et al., 2019). It is estimated that approximately 1 in 100 children worldwide have autism (Zeidan et al., 2022). At the same time, current therapeutic options for individuals with ASD are still limited and mainly focus on educational therapy and behavioural interventions. Available treatments for ASD are inadequate, and the side effects of antipsychotic drugs are well recognised (Jobski et al., 2016). The development of new therapeutic approaches should be prioritized in ongoing research efforts.

The ketogenic diet (KD) is a high-fat, low-carbohydrate dietary regimen that changes the body's metabolism from relying on glucose to using fat as the main source of energy. When carbohydrate intake is significantly reduced, liver converts fats into ketone bodies, which then serve as alternative fuel for the body and brain. The KD, originally developed in the 1920s as a form of treatment for drug-resistant epilepsy in children, has since been intensively studied for its potential health benefits (Masood et al., 2023). Among other things, its efficacy has been confirmed in reducing epileptic seizures in children, which is important given the high prevalence of epilepsy as a comorbid condition with ASD (Pizzo et al., 2022). Currently, KD is one of the most researched diets in alleviating ASD symptoms.

It is also worth mentioning that children with ASD and gastrointestinal symptoms have been reported to have reduced levels of disaccharidase transcripts and hexose transporters, as well as abnormal gut microflora.

The dysbiosis correlates with deficiencies in these enzymes and transporters. Such changes may affect the carbohydrate environment in the gut, promoting the proliferation of specific bacteria and leading to specific changes in the microbiota of children with ASD (Williams et al., 2011). Disturbed carbohydrate absorption may play a role in physio-pathological mechanisms in ASD patients. Consequently, diets low in carbohydrates - such as the KD - may be a beneficial nutritional option for this group of patients.

Another hypothesized mechanism mediating the therapeutic effects of the KD, which deserves attention in the context of treating ASD symptoms, is its effect on neurobiological processes and anti-inflammatory proprieties (Monda et al., 2024).

To further support these claims, it is relevant to point out that according to a national survey (Matthews & Adams, 2023) conducted in the USA the KD has proven to be highly promising in improving symptoms of ASD, especially those related to neurocognitive processes. The KD scored highest in terms of efficacy in improving nine significant symptoms, including attention, cognitive abilities, anxiety levels, language and communication competency, social interaction, seizures, depressive symptoms, lethargy and constipation. Unfortunately, due to the small respondent pool it is challenging to capture the diversity of the target population. The ability to generalize the findings is also restricted. It is also notable that a slightly higher rate of side effects has been reported compared to other diets, despite that KD maintains its potential as a promising therapeutic option.

Methodology

A systematic review of full-text scientific publications available in the PubMed database was conducted. The search was based on keywords such as: 'autism', 'autism spectrum disorders' 'ketogenic diet,' and 'diet therapy,' combining them with AND and OR logical operators, as applicable. The search was primarily focused to the last 8 years (2017- 2025). This paper emphasizes studies involving human subjects; however, several earlier animal studies will also be referenced as they served as a crucial precursor in shaping early hypotheses and guiding clinical trials on the KD for the individuals with ASD. The relevance and source of publications were manually assessed based on titles and abstracts. The collected information was analysed and synthesized.

Results and Discussion

Evidence from animal model studies laid the foundation for considering KD as a therapeutic strategy. Several studies conducted in the past have yielded promising results that a KD may help reduce core symptoms associated with ASD on murine models. Rodents subjected to a long-term KD demonstrated improvements particularly in social behaviours (Ruskin et al., 2013; Castro et al., 2016; Ruskin et al., 2017). Notably, the BTBR mouse strain is widely employed in ASD research. This is attributed to its manifestation of traits commonly associated with the disorder, including reduced social engagement, unusual patterns of play, and distinct vocal behaviours when compared to other inbred strains. BTBR mice that followed the KD exhibited greater sociability in the three-compartment test – a standard assay for measuring sociability in mice, as well as reduced repetitive and self-stimulatory behaviours, and improved social communication in the context of food preference (Ruskin et al., 2013).

Another study used valproic acid (VPA) in pregnant mice, resulting in autism-like symptoms in the offspring, such as impaired pain perception, repetitive behaviour and reduced sociability. KD effectively prevented social impairments and repetitive behaviours (Castro et al., 2016).

The effect of KD was also confirmed in another rodent ASD model, related to maternal immune activation (MIA) (Ruskin et al., 2017), which is considered one of the possible causes of ASD. This study analysed the effect of the KD on key symptoms of autism. Male mice exposed to MIA and fed a control diet showed social deficits and increased repetitive behaviour. Application of the KD to males partially or completely reduced these behavioural impairments. MIA female offsprings displayed less ASD-like traits than males and were visibly less affected by implemented dietary intervention. Findings remain consistent with the higher prevalence of ASD symptoms in human males. Autism is substantially more frequently diagnosed in men than in females, with a commonly cited male-to-female ratio of 4:1; however, some studies suggest the ratio may be closer to 3:1 (Loomes et al., 2017). One reason for this gap may be that females tend to use compensatory strategies – 'camouflaging' - to hide symptoms of the disorder to better fit in socially (Schuck et al., 2019; Wood-Downie et al., 2020). The observed difference implies that males and females with ASD may exhibit different behavioural profiles.

Although the exact mechanisms behind these beneficial effects of KD are not yet fully understood, many of them appear to be related to the communication between the gut microbiota and the brain, confirming the existence of a gut-brain axis. Significant changes in the composition of the gut microbiota have been observed

in both caecal and faecal samples of BTRB mice following KD (Newell et al., 2016). This may indicate that this animal models provide a valuable tool for studying gut-nervous system interactions in the context of ASD. The KD also exerted antimicrobial effects, as manifested by a significant decrease in the total number of bacteria present in caecal and faecal matter.

Studies investigating the impact of KD on individuals diagnosed with ASD is mainly focused on its effects on behaviour. To measure outcomes, instruments such as the Childhood Autism Rating Scale (CARS), Autism Treatment Evaluation Test (ATEC) and the Autism Diagnostic Observation Schedule, Second Edition (ADOS-2) are routinely employed.

In a randomized controlled trial (El-Rashidy et al., 2017) the authors compared the effectiveness of the KD and the gluten-free diet. 10 children with ASD aged 3-8 years, followed a modified Atkins diet (MAD) - a less restrictive form of KD - for 6 months. Significant improvements in autism-related symptoms were found, with the most notable changes seen in speech, sociability, and cognition, as evidenced by enhanced scores on the CARS and ATEC autism assessment scales. Comparative analysis of autism scores between the two diet plans – KD and gluten-free diet - showed no significant differences, indicating that both could be considered as potential treatment approaches for ASD patients. In MAD study group 5 children dropped out at the beginning of the clinical trial due to poor compliance. Following a certain diet might be difficult for patients with ASD as they experience notably more dysfunctional behaviours and are more food selective (Esposito et al., 2023).

A clinical trial conducted by Lee et al. (2018) investigated the effects of a modified ketogenic, gluten-free diet supplemented with medium-chain triglyceride oil (KD/GF/MCT). The study aimed to evaluate the therapeutic potential of this dietary approach in individuals with ASD. It's worth mentioning that again the participants had trouble with compliance. Out of 46 initially recruited individuals only 15 consistently followed the dietary protocol over a period of 3 months and 10 throughout 6 months. After 3 months of complying with the KD/GF/MCT regimen almost half of the participants demonstrated moderate to substantial improvement in ADOS-2 scores, especially in social interactions and emotional expression. One participant has completely stopped his restricted and repetitive behaviour. Although when it comes to the entire study group, no significant progress was observed in reducing restricted and repetitive behaviours. It is worth mentioning that caregivers additionally reported improvements in eye contact, social relationships, language comprehension, adaptation to change, concentration and hyperactivity. The total CARS-2 score also significantly decreased after 3-month treatment with the modified KD/GF/MCT diet. The greatest improvements were in imitation, body use, and anxiety levels. The group of 10 subjects that complied with the dietary protocol for a total of 6 months preserved clinical improvements in CARS and ADOS-2 scores.

Blood test results showed increase in HDL, LDL and total cholesterol levels. Also noteworthy was a significant decrease in the percentage of eosinophils and a trend toward a reduction in the number of leukocytes. Eosinophils are involved in the regulation of immune and inflammatory responses, and their increase correlates with allergic, rheumatologic, infectious, neoplastic and idiopathic diseases (Lombardi et al., 2022). Immune dysfunction and neuroinflammatory processes are recognized as ones of the potential mechanisms in the pathogenesis of ASD (Estes & McAllister, 2015; Siniscalco et al., 2018). A positive correlation was found between higher HDL and albumin levels and improvements in ASD symptoms and ADOS-2 scores. Moreover, better behavioural improvement was correlated with lower initial HDL and albumin levels. The review of existing research indicates that ASD may be associated with an increased occurrence of dyslipidemia (Dhanasekara et al., 2023).

An increasing number of studies is pointing to a significant link between lipid metabolism - particularly cholesterol and fatty acids - and ASD. Abnormalities in the lipid profile, such as reduced cholesterol levels or altered fatty acid ratios, are often observed in individuals with ASD, and may influence the severity of symptoms. Confirmation of this relationship includes cases of Smith-Lemlie-Opitz syndrome, in which a deficit in cholesterol synthesis is associated with autism-like behaviours. Reports suggest that dietary interventions may favourably modulate lipid metabolism and alleviate ASD manifestations (Esposito et al., 2021).

Mu et al. (2019) investigated in a pilot study the effects of dietary intervention like the one suggested by Lee et al. The protocol, lasting three months and involving 23 children, implemented a gluten-free, low-carbohydrate KD with additional medium-chain triglycerides (KD/GF/MCT). Overall, 17 children with ASD maintained the dietary regimen. Individuals with ASD displayed unique metabolic profiles compared to typically developed controls. The differences included increased values of intermediate compounds in galactose metabolic pathway such as galactonate, myoinositol, and glycerol, elevated level of the gut microbiota metabolite – TMAO and higher concentration of n-acetylserotonin.

Increased biosynthesis of intermediate compounds of galactose metabolism in children with ASD is worth investigating as one of the possible mechanisms of pathophysiology of ASD. Notably, galactose metabolism has been singled out in the past as one of the metabolic pathways identified as altered in the prefrontal cortex of individuals with ASD and was also noted in urine and blood metabolomic studies (Kurochkin et al., 2019). Galactosemia is a classic example of altered galactose metabolism. Galactosemia especially when insufficiently treated is linked to various neuropsychiatric syndromes such as deficits in cognitive functions, speech and language impairments, neurological deficits namely tremors, decreased information processing speed, lower IQ and higher prevalence of anxiety and depression (Welsink-Karssies et al., 2020). Furthermore, a study conducted by Korner et al. (2019) identified a specific deficit in facial emotion recognition that can significantly affect social functioning. Thus, while galactosemia and autism remain separate conditions, there are areas in which there is a possible link, and shared difficulties observed in individuals with galactosemia who also present some autistic features. Mu et al. (2019) observed that galactose levels among high responders that achieved the greatest improvements in ADOS-2 score after compiling to the dietary intervention were visibly lower than those within low-responders group. This inverse relationship between reduced galactose levels and improvement in ASD symptoms following KD/GF/MCT further proves that potential importance of galactose metabolism in ASD pathogenesis requires further investigation.

Participants that consistently implemented the KD/GF/MCT showed a significant increase in ketone bodies and acetylcarnitine involved in mitochondrial function. In addition, a noteworthy elevation of selenium levels – trace element responsible for antioxidant enzymes that protect mitochondrial membranes from oxidative stress – was observed. Oxidative stress plays an important role in the pathogenetic mechanisms of neurodegenerative disorders (Guo et al., 2013). The KD proved to be potentially effective in reducing oxidative stress and can play role in promoting cognitive improvements (Drabińska, 2024). There are indications suggesting that mitochondrial dysfunction and metabolic abnormalities may play a crucial role in the pathophysiology of ASD (Siddiqui et al., 2016). Mitochondrial dysfunction disrupts synaptogenesis and synaptic transmission, contributing to atypical brain development and behavioural symptoms. Considering their fundamental involvement in cellular processes and susceptibility to various impairments, mitochondrial dysfunction may explain common behavioural traits with ASD patients (Khaliulin I;Hamoudi W;Amal H, 2024).

In terms of elevated levels of trimethylamine N-oxide (TMAO) in participants with ASD, TMAO is emerging as a notable factor linking diet, gut microbiota, and health. Elevated levels are associated with inflammation, endothelial dysfunction, and atherosclerosis. The link between ASD and TMAO concentration is yet unclear. Although the modulation of TMAO values through diet presents promising therapeutic potential, it needs to be further studied (Caradonna et al., 2025). The recorded shifts emphasize the potential relevance of the gut-brain axis ins ASD with gut microbiome variations potentially influencing brain activity (Morton et al., 2023).

In the interventional pilot study (Allan et al., 2024) once more a combination of KD/GF/MCT was implemented by children with ASD for a period of 3 months. It is worth mentioning that adherence to diet by only 55% of participants shows again that feeding difficulties and selective eating patterns may be a significant problem among people with ASD (Alibrandi et al., 2023).

Research conducted by Allan et al. (2024) refer to the impact of the KD on gut flora. The KD shows potential in promoting better behavioural functioning of children with ASD by amplifying gut microbial diversity and rising the production of butyrate. Butyrate is acknowledged for its anti-inflammatory properties through the regulation of gut microbiota composition (Chen et al., 2024) and suppression of production of proinflammatory cytokines (Chen & Vitetta, 2020). The researchers urge further study of KD effect on social behaviour and its link to the microbiome changes

The study also analyses the effects of a KD on neuroinflammatory biomarkers and the gut-brain axis in the context of ASD. KD was shown to reduce plasma brain-derived neurotrophic factor (BDNF) levels, which may indicate a normalization of previously elevated values, possibly related to its greater accumulation in the brain. In addition, changes in the expression of microRNAs regulating neuronal development and inflammatory responses were observed, suggesting a potential effect of KD on brain activity through epigenetic mechanisms.

The study also investigated the effects of a KD on neuroinflammatory biomarkers in individuals with ASD. A decrease in circulating BDNF concentrations was documented, which could imply a normalization of previously elevated values. In addition, changes in the expression of four BDNF-related microRNAs provides supplementary evidence supporting the hypothesis that KD may affect the brain and behaviour through epigenetic mechanisms. Another recent study (Amin et al., 2024) also found significantly higher BDNF levels in children with ASD. A positive correlation was also marked between the BDNF concentrations and autism severity, Elevated BDNF levels may point to synaptopathies and brain development dysfunctions —both of

which are proposed etiologies of ASD (Won et al., 2013) - noting a potential association between changes in BDNF concentrations, core autism symptoms, and underlying foundational brain abnormalities. Nevertheless, the linkage between BDNF and autism remains unclear, with many with conflicting results.

Conclusions

These findings suggest that KD may support improvements in social affect in children with ASD. However, further research is needed to uncover the underlying mechanisms of action of this dietary plan and its effects on human behaviour.

Current evidence is limited by heterogeneous study designs, small sample sizes and poor adherence do the dietary interventions. The etiology of ASD is profoundly multifactorial and still not fully researched. Thus, pinpointing effective targeted treatment options remains challenging. Changes in mitochondrial function, metabolic activity of cells inflammation-related immune responses, modulation of the gut microbial diversity, epigenetic changes and neurostructural deviations may play a key role in the mechanisms responsible for improving behavioural symptoms in children with ASD following a KD.

Another promising area of investigation involves exploring simultaneous use of several dietary interventions. There are reports suggesting that the combination of a KD with a gluten-free diet could potentially increase the effectiveness of dietary intervention.

Acknowledgements

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All authors have read and agreed to the published version of the manuscript.

Funding statement: This research received no external funding.

Conflict of interest statement: The authors declare no conflict of interest.

Declaration of the use of generative AI and AI assisted technologies in the writing process: In preparing this work, the authors used ChatGPT for the purpose of improving language and readability. After using this tool, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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