WĄSIK, Joanna, KURKIEWICZ, Wojciech, WOŹNIAK, Oskar, MIKULSKA, Jowita, GRYBOŚ, Kacper, LEWANDOWSKI, Mikołaj, SEPIOŁO, Weronika, KIEŁBRATOWSKA, Julia and KWIATKOWSKA, Zofia. The role of diet and nutritional deficiences in neurodegenerative diseases. Quality in Sport. 2025;41:60334. eISSN 2450-3118.
https://doi.org/10.12775/QS.2025.41.60334
https://apcz.umk.pl/QS/article/view/60334

The journal has been awarded 20 points in the parametric evaluation by the Ministry of Higher Education and Science of Poland. This is according to the Annex to the announcement of the Minister of Higher Education and Science dated 05.01.2024, No. 32553. The journal has a Unique Identifier: 201398. Scientific disciplines assigned: Economics and Finance (Field of Social Sciences); Management and Quality Sciences (Field of Social Sciences).
Punkty Ministerialne z 2019 - aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).
The authors declare that there is no conflict of interest regarding the publication of this paper.
Received: 15.04.2025. Revised: 30.04.2025. Accepted: 12.05.2025. Published: 14.05.2025.

The role of diet and nutritional deficiencies in neurodegenerative diseases

Joanna Wąsik

1 Military Clinical Hospital with Polyclinic SPZOZ in Lublin

Aleje Racławickie 23, 20-049 Lublin, Poland

lek.wasik.joanna@wp.pl

https://orcid.org/0009-0004-9415-0508

Wojciech Kurkiewicz

Doctor of Medicine, Hospital of the Ministry of the Interior and Administration in Cracow,

Kronikarza Galla Street 25, 30-053 Cracow, Poland

https://orcid.org/0009-0000-0137-1311

kurkiewiczw@gmail.com

Oskar Woźniak

Medical University of Silesia, Katowice, Poland

Faculty of Medical Sciences in Katowice (FMS in Katowice)

Poniatowskiego 15, 40-055 Katowice

s81490@365.sum.edu.pl

https://orcid.org/0009-0006-1016-0720

Jowita Mikulska
Pabianice Medical Center Sp. o.o.,
ul. Jana Pawła II 68, 95-200 Pabianice, Poland
jowitamikulska@onet.pl
https://orcid.org/0009-0004-4176-8195

Kacper Gryboś
SPZOZ Myślenice, Szpitalna 2, 32-400 Myślenice
kacper.grybos@gmail.com
https://orcid.org/0009-0007-9598-8494

Mikołaj Lewandowski
Poznan University of Medical Sciences
ul. Fredry 10, 61-701 Poznań
lewymikus99@gmail.com
https://orcid.org/0009-0001-8533-0660

Weronika Sepioło
Provincial Specialist Hospital in Wrocław,
Kamieńskiego 73A, 51-124 Wrocław, Poland
weronika.sepiolo@gmail.com
https://orcid.org/0009-0007-3060-5966

Julia Kiełbratowska
Polish Red Cross Maritime Hospital in Gdynia,
1 Powstania Styczniowego Street, 81-519 Gdynia,Poland
https://orcid.org/0009-0005-3328-5502
juliakielbratowska@gmail.com
Zofia Kwiatkowska
Katowickie Centrum Onkologii
Raciborska 26 40-074 Katowice
kwiatkowskaa@icloud.com
https://orcid.org/0009-0002-4895-9002

Abstract

Purpose of reaserch: This is a review of the literature on the most common neurodegenerative diseases, their causes, impact on physical and mental health, diets and nutritional deficiencies influencing the development or course of the disease.
Materials and methods: The literature review was based on materials retrieved from the PubMed and Google Scholar databases, using keywords such as „neurodegenerative disease'', „Diet In Alzhaimer disease'', „Diet In Parkinson disease''. The scope of the studies analyzed covered the years 1999 to 2024, with a focus on recent research.
Results: Neurodegenerative diseases are incurable, and a factor relatively as easily modifiable as an adequate diet influences both the onset and course of the disease. An appropriate diet can delay the onset of some symptoms as well as alleviate them, but much research is still needed to determine which diets have a protective effect and which diets or products will increase symptoms.
Conclusions: Neurodegenerative diseases are increasingly common in ageing societies, more and more patients will face these diseases. This is why it is so important to protect the patient in every possible way, to prevent or reduce their ailments, and proper nutrition is a relatively easily modifiable factor that can aid this process. We compared the work of dozens of authors from different corners of the world who studied the effects of different products and diets on the development of neurodegenerative diseases.

Keywords: Alzhaimer disease, Parkinson disease, amyotrophic lateral sclerosis, nutrient, neurodegenerative disease, Dash, MIND

1.Introduction

Brain diseases are still one of the least understood diseases of the human body. Despite the increasingly better progress in technology and tools for understanding the human brain, many diseases and their pathomechanisms are still unknown. Most brain diseases are still incurable, and treatment only slows down the progression of the disease, which is a huge progress in science and a success for humanity. The brain and neurological and mental diseases have always aroused fear among society and admiration among researchers due to the complexity of the brain. The brain dies the fastest of all organs when it is hypoxic; after four minutes of hypoxia, the brain partially dies, and after 10 minutes of oxygen deprivation, permanent brain damage occurs. Research is still ongoing on the risk factors of brain diseases, the influence of heredity and what factors can cause symptoms to develop faster or slow down. It is common knowledge that not only pharmacological treatment can limit the worsening of the disease, but also appropriate diet, exercise and adequate sleep can influence the development of the disease. Due to the aging of societies, better diagnostics and due to more effective treatment of other diseases, there is a noticeable increase in the incidence of neurodegenerative diseases. That is why it is so important to constantly investigate these diseases and seek treatment that will improve the quality of life of both the patient and his family as well as the factors that exacerbate and alleviate the course of the disease. Neurodegenerative diseases significantly reduce the quality of life of patients because they are associated with progressive damage to

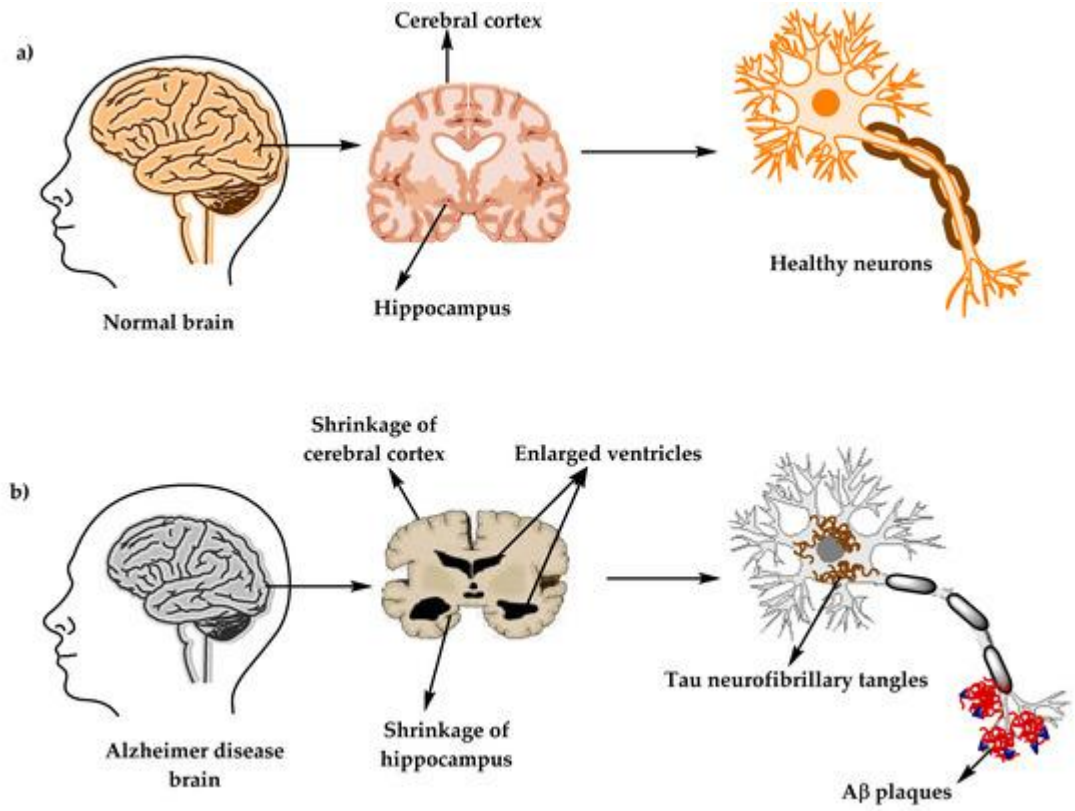
neurons and the deepening of symptoms and the appearance of new ones. In neurological diseases, it is very common for patients to be malnourished as well as having difficulty swallowing food. The most common neurodegenerative diseases in society include: Alzheimer's disease, Parkinson's disease and amyotrophic lateral sclerosis.

2. Alzhaimer disease

2.1 Pathophysiology

Alzheimer's disease is the most common dementia disease. It is estimated that approximately 44 million people around the world currently live with this disease, and a new diagnosis is made every 4 seconds. The World Health Organization (WHO) estimates that by 2030 there will be 65 million sick people, and by 2050 as many as 115 million sick people. More women suffer from Alzheimer's than men. Alzheimer's disease was first described by neuropathologist Aloysius (Alois) Alzheimer's in 1901. (1) He described a 50-year-old woman who suffered from a progressive disease manifesting itself in aggression, disorientation and progressive memory disorders. (1) The patient died 5 years later, and the autopsy showed a reduction in the size of the brain compared to the normal size and abnormal deposits, which he called plaques and neurofibrillary tangles. (1) However, the name Alzheimer's disease was first used in 1908. The so-called amyloid cascade theory has been valid for over a hundred years and these changes are believed to be the causes of the disease. It is believed that the accumulation of amyloid causes the formation of amyloid plaques, which interfere with the conduction of synaptic signal and as a result of formation free radicals (ROS), contributes to neuronal apoptosis. (11) Symptoms begin gradually and are non-specific, making early diagnosis difficult. Research has shown that on average 3 years before diagnosis, the family notices the first symptoms of dementia. The most common form of the disease is the amnesic form, which is characterized by impaired episodic memory, the centers of which are located in the temporal lobe. In moderate and severe forms of the disease, the changes spread to larger areas of the brain. The disease progresses and the patient becomes more and more dependent on other people and needs help with basic life activities. Most patients die due to increased susceptibility to falls and infections such as aspiration pneumonia.
Current research shows that the average survival time from diagnosis is on average 8-10 years, but this period will certainly be longer due to the increasing progress of medicine. Another hypothesis for the development of Alzheimer's disease is considered to be the cholinergic hypothesis for the development of the disease. This hypothesis states that there is a progressive loss of cholinergic innervation in the neocortex and the limbic system. Neurofibrillary degeneration of presynaptic cholinergic denervation is considered to be the factor responsible for cholinergic neuronal dysfunction and death. Cholinesterase inhibitors are among the few drugs that have been proven to be effective in this disease. Through their action they increase the amount of acetylcholine available. (2)
It is believed that the disease is polygenic, complex, and whose essence lies in abnormal interactions between several molecular pathways, but age is the strongest risk factor. (1) Neuropathologically, it is believed that there is an accumulation of abnormal proteins - amyloid-β in plaques and hyperphosphorylated tau protein in neurofibrillary tangles, which leads to the loss of dendrites, synapses and, consequently, neurons. With research into the cholinergic hypothesis of disease development, it was discovered that the nucleus basalis meynert in the basal forebrain is the source of cortical cholinergic innervation that undergoes neurodegeneration in Alzheimer's disease. Still, treatments that improve cholinergic function are the most effective.

Other pathomechanisms of the disease development include inflammation of the nervous system, insulin resistance, oxidative stress and abnormalities in the blood vessels of the brain. One study has shown that the loss of cholinergic innervation is presumably associated with neurofibrillary tangles. Despite enormous progress in neurobiological sciences and testing of new drugs, there is still no medicine that would cure this disease, which is why prevention is so important. (13)



a) Normal brain b) Brain in Alzhaimer disease, with characteristic Tau neurofibrillary tangles and plaques [30]

2.2 Diet In Alzhaimer disease

Lifestyle also plays a role in the pathophysiology of the disease, i.e. smoking, alcohol consumption, sleep, level education and physical activity. The occurrence of hypertension and stroke throughout life is closely related to dementia in old age. Unfortunately, there is still too little research on diets and products consumed that can both prevent and contribute to the disease. There are already several studies that have proven that following the DASH [12], MIND (Mediterranean-DASH Intervention for Neurodegenerative Delay) and Mediterranean diets can help protect against disease and have a positive impact on patients' cognitive functions. (3, 22, 24) Dietary Approach to Stop Hypertension (DASH), is a diet strongly recommended especially for patients with hipertension. (13) Following the DASH diet is associated with slower cognitive decline. DASH may also help halt the progression of mild

cognitive impairment and reduce the overall mortality rate of Alzheimer's patients. (13) It has been shown that the consumption of extra virgin olive oil may delay the progression of cognitive functions. (13,14) Consuming unsaturated fatty acids also maintains the structural integrity of neuronal membranes, thereby regulating neuronal transmission. (14)
Dietary deficiency of antioxidant vitamins C and E and B vitamins - vitamin B6 and vitamin B 12, as well as folate deficiency may be a risk factor for the development of the disease. (13) It has been shown that antioxidant vitamins reduce lipid peroxidation and oxidative stress by inducing β-amyloid. In addition, they inhibit the inflammatory cascade. B vitamins and folic acid are essential cofactors of homocysteine metabolism because they are involved in DNA methylation, and increasing homocysteine levels may contribute to the development of the disease and subsequent oxidation. A diet high in fat and an excess of saturated fatty acids may also influence the development of the disease. Higher fat consumption is associated with hyperinsulinemia, the impact of which on the development of the disease has been described above. More fat means higher cholesterol levels, which contribute to the accumulation of oxyterols in the brain, which have been proven in patients. Additionally, a relationship has been demonstrated between high cholesterol levels and the severity of dementia in patients. It has been shown that diet high in fats can cause hyperphosphorylation of the Tau protein and negatively affect memory, but there are also studies refuting this thesis. (4) Research has also been carried out on the relationship between cadmium and lead and Alzheimer's disease, but the research is inconclusive. Malnutrition in patients is very common and has an unfavorable prognosis and contributes to the acceleration of the disease progression. Studies have shown that changes in the central nervous system also affect the centers responsible for appetite control and regulation of food intake. It has also been proven that patients experience a decreased sense of taste, (5,10) which also leads to a reduction in the amount of products consumed and, consequently, to malnutrition. (5,10) Smell and taste disturbances may not be reported by patients because patients are often unaware of their loss.

Patients have problems with preparing meals, and often do not remember whether they have already eaten a meal, and also forget to drink fluids which causes some people to eat more food than they need, which can lead to excess weight. Studies have shown that being overweight can have the same negative effects on cognitive function as malnutrition does in patients. It is recommended that a person suffering from Alzheimer's eat a diet recommended for older people in order to optimize the patient's energy needs as much as possible. That is, people over 65 years of age should consume approximately 1,500-2,000 kcal each Day. (6) There are studies that show that an adequate supply of protein throughout life can prevent the occurrence of Alzheimer's disease. (7) A study conducted by Yeh T.S. et al. showed that the consumption of legumes, fish and lean meat has a more beneficial effect on cognitive functions than the consumption of fast food animal products, therefore the quality of the consumed protein is important. [8] Polish scientists have developed a diet calculator against Alzheimer's disease. They calculated the potential preventive amounts of macronutrients - fats, carbohydrates and protein, depending on age. (7) Scientists also calculated what the proportions should be between the consumption of particular types of fats. Studies have been conducted on the impact of the Mediterranean diet and a slower progression of cognitive function deterioration and a reduced risk of disease have been observed, as well as a longer survival period in patients. (5) A study by other researchers found that a higher BMI was associated with a lower risk of dementia than a normal BMI and being underweight. Being underweight increases the risk of all dementias. (9) But current research shows that low BMI in the elderly may predict the development of Alzheimer's disease. (9)

3.1 Pathophysiology

The second most prevalent neurodegenerative disease is Parkinson's disease, and it is predicted that the number of patients will increase significantly in the coming years. (16) The symptoms of the disease are due to degenerative changes in the black matter, resulting in a deficiency of dopamine in the black matter and striatum, and a predominance of glutamatergic neuronal activity. (16) Lewy bodies are also found, which are abnormal clusters of proteins, aggregation of α-synuclein that can also occur in other diseases. (16) The risk of the disease increases with age, and statistics say that more men get the disease. Genetic load is also an important factor in the disease, and factors such as smoking and a history of head trauma have also been shown to influence the incidence. (15) The main symptoms of the disease are bradykinesias, gait and postural changes, rigidity and resting tremor, and dysarthria or dysphagia. Up to 90% of patients complain of a reduced ability to smell, which can have a significant impact on the patients' food intake and diet. Tests are used to assess the sense of smell: UPSIT and the Sniffin'sticks test. Patients also often indicate significant sleep problems, a tendency to panic attacks, parasthesias or problems related to the digestive system such as constipation or gastroparesis. (15)

3.2 Diet In Parkinson disease

A change in diet and dietary intake, is one of the few modifiable factors and can be changed by both the patient already suffering from Parkinson's disease and by healthy individuals in order to prevent the development of the disease. Despite ongoing medical developments, there are still no methods to prevent the development of the disease, and various factors that may influence the development of the disease including diet have been studied, but research is inconsistent. Up to 60% of PD patients are at risk of malnutrition, especially In the Middle stage of disease. (17) Studies carried out have shown a possible cause for the constipation suffered by patients - through the loss of body fat. (16, 17) Furthermore, the researchers also observed the effect of malnutrition among patients on the progressive loss of motor function of patients. (16,18) Despite ongoing research into the disease, its pathophysiology, possible risk factors and drugs that can help alleviate symptoms, there are still not many cures, with Levodopa being the main one used, which is why it is so important to prevent the disease and prevent malnutrition. The many studies that have been carried out investigating the effects of specific ingredients on the development and prevention of the disease vary depending on where the study was carried out and the research sample, so it is currently believed that a holistic approach to diet can have positive effects. One study found that patients who consumed more coconut oil, fruit, vegetables, fish and oils reported less discomfort. (16, 19) Weight loss in patients is associated with a poorer quality of life on a daily basis and with a faster progression of parkinsonism, in addition, osteoporosis develops more often in these patients, bone fractures are more frequent and pressure ulcers are more noticeable in patients. It is believed that people with PD have a higher energy demand than healthy patients, because muscle stiffness and involuntary movements are activities that expend a lot of energy. Patients also have difficulty preparing meals themselves, and progressive hyposmia causes a smaller appetite and therefore a smaller amount of meals. The large number of diagnosed mood disorders, including depression, in patients is also not without influence, which also affects the reduction in the amount of food consumed by patients. (17)

A contentious issue is the consumption of dairy products, as some studies have reported a reduced risk of disease and others have reported an increased risk, studies done to resolve this contentious issue show that high-fat dairy products reduce the risk of disease, while low-fat dairy products increase the risk. (19)

A study of the effect of the DASH diet on Parkinson's disease was conducted. It showed no association between adherence to the diet and the disease (20), but another study showed that The MIND diet may be a associated with decreased risk and slower progression of parkinsonism in older adults. (21) A study by researchers found that iron supplementation, a diet high in beef, yoghurt, diet and non-diet drinks can accelerate disease progression. (18) Foods that are also associated with a reduced likelihood of PD are green tea, coffees and berries. (18,21,22) A Mediterranean diet has been shown to be associated with both a later age of diagnosis and a reduced incidence of disease. (23) This means that this diet has a positive effect on both of the most common neurodegenerative diseases. (12,14, 23)

4. Amyotrophic lateral sclerosis

4.1 Pathophysiology

Otherwise known as Lou Gehrig's disease (30) it progresses, leads to death, and despite ongoing research, many aspects of the disease are unexplored, or the research conducted has not yielded the desired results, the disease is quite rare, as there are between 2 and 4 people per 100,000 affected, and mainly older people in their 6th/7th decade are affected. (25,26,28) The disease is difficult to diagnose. It leads to the destruction of the anterior horns of the spinal cord of the cranial nerve nuclei of the medulla oblongata and the neurons of the pyramidal pathway. The aetiology of the disease is not precisely known, but there are mainly two types, one genetic, which accounts for up to 10 per cent of cases and is caused by a mutation in the superoxide dismutase gene on chromosome 21, which usually occurs at a younger age, and the other type which occurs in approximately 90% of patients, which is sporadic, meaning that there is no single gene that is responsible for the appearance of the disease in the patient. (29) The exact pathophysiology of the disease is not known, (28) but there are several hypotheses, the first hypothesis being that superoxide dismutase gains new catalytic functions as a result of genetic changes in its structure. (26) The second hypothesis called „gain of interaction'' assumes that the mutant SOD1 protein is catalytically inactive and degrades. (28) However, these are only hypotheses, possibly simultaneous and complementary, but research is still needed to prove them. It is currently thought that the main role in the development of the disease is a conformational change in the SOD1 protein, which results in a loss of control of inter-protein interactions with the formation of abnormal insoluble aggregates containing Beta-amyloid. Beta amyloid is probably responsible for the formation of free radicals. In addition, neuronal metabolism has been shown to be impaired and mitochondria are abnormal in structure, swollen and vacuolised. In the disease, there is a progressive degeneration of motoneurons which results, in simple terms, in the brain being unable to control muscle function, leading in the long term to the death of the patient. The main symptoms of the disease are muscle atrophy, spastic paresis of the lower limbs and even features of palsy syndrome. As the disease progresses, more muscles atrophy occurs, accompanied by bulbar tremor. With the duration of the disease there is progressive paralysis and eventually death due to respiratory muscle arrest. The average survival time from detection is approximately three to five years. The lesions affect various parts of the brain as well as the nerves - the sublingual nerve is often involved. The pathognomonic symptom for ALS is cytoplasmic inclusions. Patients receive palliative targeted nutritional therapy to meet the nutritional requirements of the patient as well as to facilitate mechanical ventilation, and

low respiratory factor diets are used. Depending on the organs affected, the method of treatment is adapted: if swallowing is impaired, patients are fed by gastrostomy or gastrojejunostomy; if masseter muscle paralysis occurs and solid food cannot be accepted, patients are fed liquid food.

4.2 Diet In AML disease

Some of the papers describe products whose increased consumption has been observed in ALS patients due to increased ROS release, but there are also products that have been studied and found to have potential disease-preventing and disease-slowing effects. (28) One such product for its anti-inflammatory and antioxidant properties may be curcumin, which is extracted from the rhizomes of Curcuma longa. It has beneficial effects on neurodegeneration. (28) Research is ongoing and several cases of patients on a ketogenic diet have already been described. The results of these studies are promising, one of the subjects was a patient suffering from a bulbar form of the disease, which has a very poor prognosis. (26) From detection, the estimated survival time is a maximum of 24 months. The patient's use of this diet significantly improved function by, among other things, increasing vital lung capacity, increasing forced expiratory volume, the patient feeling less tired and fatigued, but not swallowing disorders, but importantly the patient himself admitted that his quality of life had improved. (26) Following a ketogenic diet alters cellular metabolism and mitochondrial function, which has been used in this disease. When ketones are metabolised, fewer reactive oxygen species are also produced, and the production of oxidative stress resistance factors is increased. By following a ketogenic diet, the mitochondrial pool is renewed. (26) There is still too little research, but the effects of a ketogenic diet on mice have been studied and shown to be associated with longer retained motor performance. (27) The study patient had two meals a day and the diet consisted of approximately 60% fat, 30% protein, 5% fibre and 5% carbohydrates, whole foods - meat, eggs, vegetables, nuts, seeds, natural oils. No calories were counted in the diet and the patient ate to his heart's content. The patient reported no adverse effects. The patient had lost 10 kilograms over a period of almost two years before the ketogenic diet was introduced, and weight loss in ALS is an adverse prognostic factor. During his time on the diet, his weight decreased by approximately 3 kilograms over a period of 1.5 years. (26) Fasting and the ketogenic diet have been shown to influence mitochondrial renewal, through mitochondrial synthesis, in addition, the use of these diets has a positive effect on mitochondrial function. The ketogenic diet influences the formation of fewer reactive oxygen species and the production of oxidative stress resistance factors is increased.

Summary

Neurological diseases are still not thoroughly researched, despite the knowledge of their probable pathophysiology, their exact cause and the influence of factors both on the onset of the disease, on the occurrence of symptoms and on prevention are still unknown. That is why prevention through modifiable factors, i.e. adequate diet and exercise, is so important. As shown in the study, an adequate, balanced diet can both delay the onset of the disease and reduce the severity of complaints, and can prevent weight loss, which is significant in neurological diseases and can lead to problems with patient mobility. Adequate nutrition in these diseases also improves wellbeing, which has an impact on the patient's overall functioning. The diseases discussed above are severe conditions due to their incurable nature, and symptoms often progress over time despite treatment, which is why it is important to improve the patient's daily wellbeing through available methods other than drug treatment.

Disclosure:
Author’s contribution
Conceptualization: Joanna Wąsik
Methodology: Mikołaj Lewandowski, Weronika Sepioło
Software: Wojciech Kurkiewicz, Julia Kiełbratowska
Check: Jowita Mikulska, Wojciech Kurkiewicz
Formal analysis: Zofia Kwiatkowska
Investigation: Filip Nadolny, Kacper Gryboś
Resources: Julia Kiełbratowska, Oskar Woźniak
Data curation: Mikołaj Lewandowski, Kacper Gryboś
Writing- rough preparation: Joanna Wąsik
Writing – review and editing: Jowita Mikulska, Oskar Woźniak
Supervision: Weronika Sepioło, Filip Nadolny
Project administration: Zofia Kwiatkowska

All authors have read and agreed with the published version of the manuscript. Founding Statement: The study did not receive funding.
During the preparation of this paper, the authors utilized artificial intelligence (AI) tools, specifically ChatGPT, for writing and supporting the scientific work. AI was employed to assist in tasks such as content generation, data analysis, and the structuring of the paper. All AI-generated content has been properly reviewed, verified, and integrated into the research in accordance with academic integrity standards and the authors take full responsibility for substantive content of the scientific work. 11 Intuitional Review Board Statement: Not applicable. Informed Consent Statement: Not applicable. Data Availability Statement: Not applicable. Conflict of interest Statement: The authors declare no conflicts of interest. Acknowledgments: Not applicable.

References:

1. Eratne D, Loi SM, Farrand S, Kelso W, Velakoulis D, Looi JC. Alzheimer's disease: clinical update on epidemiology, pathophysiology and diagnosis. *Australas Psychiatry*. 2018;26(4):347-357. doi:10.1177/1039856218762308
2. Hampel H, Mesulam MM, Cuello AC, et al. The cholinergic system in the pathophysiology and treatment of Alzheimer's disease. *Brain*. 2018;141(7):1917-1933. doi:10.1093/brain/awy132
3. Stefaniak O, Dobrzyńska M, Drzymała-Czyż S, Przysławski J. Diet in the Prevention of Alzheimer's Disease: Current Knowledge and Future Research Requirements. *Nutrients*. 2022;14(21):4564. Published 2022 Oct 30. doi:10.3390/nu14214564

4. Ylilauri MP, Voutilainen S, Lönnroos E, et al. Association of dietary cholesterol and egg intakes with the risk of incident dementia or Alzheimer disease: the Kuopio Ischaemic Heart Disease Risk Factor Study. *Am J Clin Nutr*. 2017;105(2):476-484. doi:10.3945/ajcn.116.146753
5. Martín ISM, Barato VP, Oliva SL, et al. Body Composition, Dietary, and Gustatory Function Assessment in People With Alzheimer's Disease. *Am J Alzheimers Dis Other Demen*. 2018;33(8):508-515. doi:10.1177/1533317518782173
6. de Groot CP, van den Broek T, van Staveren W. Energy intake and micronutrient intake in elderly Europeans: seeking the minimum requirement in the SENECA study. *Age Ageing*. 1999;28(5):469-474. doi:10.1093/ageing/28.5.469
7. Studnicki M, Dębski KJ, Stępkowski D. Proportions of macronutrients, including specific dietary fats, in prospective anti-Alzheimer's diet. *Sci Rep*. 2019;9(1):20143. Published 2019 Dec 27. doi:10.1038/s41598-019-56687-2
8. Yeh TS, Yuan C, Ascherio A, Rosner BA, Blacker D, Willett WC. Long-term dietary protein intake and subjective cognitive decline in US men and women. *Am J Clin Nutr*. 2022;115(1):199-210. doi:10.1093/ajcn/nqab236
9. Cova I, Clerici F, Maggiore L, et al. Body Mass Index Predicts Progression of Mild Cognitive Impairment to Dementia. *Dement Geriatr Cogn Disord*. 2016;41(3-4):172-180. doi:10.1159/000444216
10. Steinbach S, Hundt W, Vaitl A, et al. Taste in mild cognitive impairment and Alzheimer's disease. *J Neurol*. 2010;257(2):238-246. doi:10.1007/s00415-009-5300-6
11. Varadarajan S, Yatin S, Aksenova M, Butterfield DA. Review: Alzheimer's amyloid beta-peptide-associated free radical oxidative stress and neurotoxicity. *J Struct Biol*. 2000;130(2-3):184-208. doi:10.1006/jsbi.2000.4274
12. Tangney CC. DASH and Mediterranean-type Dietary Patterns to Maintain Cognitive Health. *Curr Nutr Rep*. 2014;3(1):51-61. doi:10.1007/s13668-013-0070-2
13. Solfrizzi V, Panza F, Frisardi V, et al. Diet and Alzheimer's disease risk factors or prevention: the current evidence. *Expert Rev Neurother*. 2011;11(5):677-708. doi:10.1586/ern.11.56
14. Singh B, Parsaik AK, Mielke MM, et al. Association of mediterranean diet with mild cognitive impairment and Alzheimer's disease: a systematic review and meta-analysis. *J Alzheimers Dis*. 2014;39(2):271-282. doi:10.3233/JAD-130830
15. Tolosa E, Garrido A, Scholz SW, Poewe W. Challenges in the diagnosis of Parkinson's disease. *Lancet Neurol*. 2021;20(5):385-397. doi:10.1016/S1474-4422(21)00030-2

16. Knight E, Geetha T, Burnett D, Babu JR. The Role of Diet and Dietary Patterns in Parkinson's Disease. *Nutrients*. 2022;14(21):4472. Published 2022 Oct 25. doi:10.3390/nu14214472
17. Yong VW, Tan YJ, Ng YD, et al. Progressive and accelerated weight and body fat loss in Parkinson's disease: A three-year prospective longitudinal study. *Parkinsonism Relat Disord*. 2020;77:28-35. doi:10.1016/j.parkreldis.2020.06.015
18. Yang T, Zhan Z, Zhang L, et al. Prevalence and Risk Factors for Malnutrition in Patients With Parkinson's Disease. *Front Neurol*. 2020;11:533731. Published 2020 Dec 10. doi:10.3389/fneur.2020.533731
19. Mischley LK, Lau RC, Bennett RD. Role of Diet and Nutritional Supplements in Parkinson's Disease Progression. *Oxid Med Cell Longev*. 2017;2017:6405278. doi:10.1155/2017/6405278
20. Hughes KC, Gao X, Kim IY, et al. Intake of dairy foods and risk of Parkinson disease. *Neurology*. 2017;89(1):46-52. doi:10.1212/WNL.0000000000004057
21. Agarwal P, Wang Y, Buchman AS, Holland TM, Bennett DA, Morris MC. MIND Diet Associated with Reduced Incidence and Delayed Progression of ParkinsonismA in Old Age. *J Nutr Health Aging*. 2018;22(10):1211-1215. doi:10.1007/s12603-018-1094-5
22. Gao X, Cassidy A, Schwarzschild MA, Rimm EB, Ascherio A. Habitual intake of dietary flavonoids and risk of Parkinson disease. *Neurology*. 2012;78(15):1138-1145. doi:10.1212/WNL.0b013e31824f7fc4
23. Gao X, Chen H, Fung TT, et al. Prospective study of dietary pattern and risk of Parkinson disease. *Am J Clin Nutr*. 2007;86(5):1486-1494. doi:10.1093/ajcn/86.5.1486
24. Alcalay RN, Gu Y, Mejia-Santana H, Cote L, Marder KS, Scarmeas N. The association between Mediterranean diet adherence and Parkinson's disease. *Mov Disord*. 2012;27(6):771-774. doi:10.1002/mds.24918
25. Feldman EL, Goutman SA, Petri S, et al. Amyotrophic lateral sclerosis. *Lancet*. 2022;400(10360):1363-1380. doi:10.1016/S0140-6736(22)01272-7
26. Phillips MCL, Johnston SE, Simpson P, Chang DK, Mather D, Dick RJ. Time-restricted ketogenic diet in amyotrophic lateral sclerosis: a case study. *Front Neurol*. 2024;14:1329541. Published 2024 Jan 18. doi:10.3389/fneur.2023.1329541
27. Zhao Z, Lange DJ, Voustianiouk A, et al. A ketogenic diet as a potential novel therapeutic intervention in amyotrophic lateral sclerosis. *BMC Neurosci*. 2006;7:29. Published 2006 Apr 3. doi:10.1186/1471-2202-7-29

28. D'Antona S, Caramenti M, Porro D, Castiglioni I, Cava C. Amyotrophic Lateral Sclerosis: A Diet Review. *Foods*. 2021;10(12):3128. Published 2021 Dec 17. doi:10.3390/foods10123128
29. Pape, J A, and J H Grose. “The effects of diet and sex in amyotrophic lateral sclerosis.” *Revue neurologique* vol. 176,5 (2020): 301-315. doi:10.1016/j.neurol.2019.09.008
30. Breijyeh, Zeinab, and Rafik Karaman. “Comprehensive Review on Alzheimer's Disease: Causes and Treatment.” *Molecules (Basel, Switzerland)* vol. 25,24 5789. 8 Dec. 2020, doi:10.3390/molecules25245789