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Holistic perspectives on Parkinson's disease: a comparative observational study of risk factors

Choroba Parkinsona w ujęciu holistycznym: obserwacyjne badanie porównawcze czynników ryzyka

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Abstract

Introduction and objective: Although recent findings reveal potential contributing factors to Parkinson's disease, ongoing research remains essential to further understand and substantiate these connections, identifying lifestyle patterns and biopsychological factors that may contribute to either the onset or prevention of Parkinson's disease. **Materials and methods:** This study analyses the interactions among genetics, neuroplasticity, interoception, mindfulness, lifestyle, environment, and Parkinson's disease, facilitating exploration of the underlying choices that influence disease development. While mediated factors do not directly cause Parkinson's disease, they affect symptom severity, disease progression, and overall quality of life. With these insights, based on comparative observation and the application of a thought experiment, a Collaborative Impact Model was developed to enhance knowledge of contributing factors to Parkinson's disease. The study had no human subjects. **Results:** Observations suggest that genetics and neuroplasticity, significantly shaped by lifestyle, directly influence the onset and progression of Parkinson's disease. Genetic predispositions and adaptive neuroplasticity determine neural resilience or vulnerability, while environmental factors, interoception, mindfulness, and general lifestyle indirectly influence Parkinson's disease by modulating stress, inflammation, cognitive health, and emotional regulation, thus affecting overall disease trajectory and symptom severity. **Conclusions:** The outlined approach captures the complex interplay between genetic factors, neuroplastic adaptation, and external influences, emphasising how lifestyle choices can have both immediate and long-term effects. It facilitates a multidimensional strategy for prevention, management, patient support, and education in Parkinson's disease.

Keywords: neuroplasticity, interoceptive awareness, mindfulness, behavioural changes, Parkinson's disease

Streszczenie

Wprowadzenie i cel: Najnowsze badania wskazują na potencjalne czynniki sprzyjające wystąpieniu choroby Parkinsona, jednakże konieczne są dalsze analizy w celu lepszego zrozumienia i potwierdzenia tych zależności, a także identyfikacji wzorców stylu życia oraz czynników biopsychologicznych, które mogą wpływać na rozwój lub zapobieganie tej chorobie. **Materiał i metody:** Badanie analizuje złożone interakcje między czynnikami genetycznymi, neuroplastycznością, interocepcją, uważnością, stylem życia oraz środowiskiem w kontekście choroby Parkinsona. Pozwala lepiej zrozumieć, jak codzienne wybory i zachowania mogą wpływać na rozwój tej choroby. Chociaż czynniki pośrednie same w sobie nie wywołują choroby Parkinsona, mają istotny wpływ na stopień i tempo nasilenia objawów oraz ogólną jakość życia pacjentów. Na podstawie obserwacji porównawczej oraz eksperymentu myślowego zaproponowano autorski Model Wpływu Współdziałania, który poszerza wiedzę na temat czynników sprzyjających

występowaniu choroby Parkinsona. Badanie miało charakter wyłącznie teoretyczny. **Wyniki:** Obserwacje sugerują, że genetyka i neuroplastyczność, istotnie kształtowane przez styl życia, odgrywają ważną rolę w pojawieniu się i rozwoju choroby Parkinsona. Predyspozycje genetyczne oraz adaptacyjna neuroplastyczność mają bezpośredni wpływ na odporność lub podatność układu nerwowego. Natomiast czynniki środowiskowe, interocepcja, uważność oraz ogólnie pojęty styl życia oddziałują na chorobę Parkinsona pośrednio, modulując stres, procesy zapalne, zdrowie poznawcze i regulację emocji, przez co wpływają na przebieg choroby oraz nasilenie jej objawów. **Wnioski:** Przedstawione podejście ujmuje złożone zależności między genetyką, adaptacją neuroplastyczną i czynnikami zewnętrznymi, podkreślając, w jaki sposób wybory dotyczące stylu życia mogą mieć zarówno bezpośrednie, jak i pośrednie skutki, ułatwiając wielowymiarową strategię zapobiegania, zarządzania, wsparcia pacjenta i edukacji w zakresie choroby Parkinsona.

Słowa kluczowe: neuroplastyczność, świadomość interoceptywna, uważność, zmiany behawioralne, choroba Parkinsona

INTRODUCTION

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterised by motor and non-motor symptoms, profoundly affecting an individual's quality of life. The global prevalence of PD has increased, placing a significant burden on healthcare systems, particularly in aging populations. Despite extensive research, the exact causes of PD remain uncertain, as the disorder likely results from a complex interplay of genetic, environmental, and lifestyle factors (Kalia and Lang, 2015). Analysing these factors through an integrated model provides a valuable framework for better understanding of the development and progression of PD, enabling more effective prevention and intervention strategies. To address the complexity of PD development, this comparative observation incorporated a thought experiment to hypothesise the collaborative effects of multiple contributing factors. The objective of this study was to analyse and conceptualise lifestyle factors influencing PD, and to examine how overlapping direct and indirect mechanisms affect its onset and progression through a comparative analysis of related pathways.

MULTI-FACTORIAL MODEL OF DISEASE

The multi-factorial disease model posits that most diseases result from a combination of multiple interacting factors rather than a single biological change, such as declining immune function and impaired cellular repair processes that make the body more susceptible to illness.

Stress, social isolation, and mental health are increasingly recognised as significant contributors to disease (Lee et al., 2022). Additionally, exposure to pathogens is necessary for infectious conditions to develop. However, the severity and likelihood of illness depend on multiple factors like immune system strength and pathogen virulence, not any single factor alone. This concept is widely accepted in both chronic and acute conditions (Olan, 2023). Genetic susceptibility states that several diseases, including PD, involve a genetic predisposition (Zhou and Skolnick, 2016). However, genetic factors alone rarely cause disease. They increase vulnerability. For a disease to manifest, other factors typically need to be present (Fan et al., 2013). Environmental influences, such as exposure to toxins, pollution, and infectious agents, can trigger or accelerate disease development. In many instances, individuals with a genetic predisposition to a disease develop the condition only after specific environmental exposures. Lifestyle and behavioural choices, including diet, physical activity, sleep patterns, and stress levels, can significantly contribute to the onset and progression of disease (Vodovotz et al., 2020). Also important are age and characteristics of the infectious agent, exposure dose, health and lifestyle, and prior immunity (Sompayrac, 2022). These contributing elements can converge, creating a cumulative risk that triggers the onset or accelerates the progression of disease. Adopting an interrelated approach can facilitate preventive measures, such as modifying certain risk factors.

PRE-DIAGNOSTIC LIFESTYLE OF PD PATIENTS AND REGIONS WITH LOWER PD PREVALENCE

What lifestyle do typical PD patients have? Before being diagnosed with PD, a typical person may have led a lifestyle that did not account for the specific health concerns and adjustments that become necessary after diagnosis (Noyce et al., 2016). The standard lifestyle patterns observed in individuals before diagnosis often contrast with their habits afterward. An average individual diagnosed with PD may have led an average lifestyle in terms of physical activity. Some individuals may have engaged in regular exercise, such as jogging, cycling, or gym workouts, while others may have had more sedentary routines, with physical activity limited to daily tasks or occasional recreational pursuits (Cholewa et al., 2016; Fang et al., 2018; Gorzkowska et al., 2020; Uher et al., 2021). A typical diet may not have been specifically tailored to support brain health or symptom management. Diets varied widely and often lacked a focus on specific nutrients, hydration, or fibre intake. High-protein meals, processed foods, or inconsistent eating habits may have been typical, as there was no need to avoid certain foods to support the effectiveness of medication (Smalira et al., 2024). Regarding work and social engagement, many individuals led active social and professional lives before diagnosis, often maintaining full-time jobs, attending

social gatherings, and enjoying hobbies without restrictions. Individuals may have been deeply involved in their careers, family life, or community events, with no physical or cognitive symptoms that limited their participation. Before diagnosis, mental health practices and stress management strategies were generally less structured or intentional. Stress was handled in ways familiar to the general population, relying on exercise, social support, or hobbies rather than systematic practices like mindfulness or yoga, which later become helpful for symptom management (Postuma et al., 2015).

Sleep and rest patterns may have been less of a priority, as many individuals experienced occasional sleep disturbances due to work schedules, family obligations, or lifestyle choices. Sleep disorders specific to PD, such as REM sleep behaviour disorder, would not have been a concern or might have gone unnoticed until later (Postuma et al., 2012). While many engaged in mentally stimulating activities like reading, hobbies, or problem-solving, there was typically no specific focus on cognitive activities to delay cognitive decline. Cognitive health exercises would likely not have been a structured part of daily life for individuals before being diagnosed with PD. Some individuals may have been exposed to potential risk factors through their work or lifestyle, such as pesticides, herbicides, or industrial chemicals, particularly in rural or industrial areas (Goldman, 2014). Head injuries or participation in frequent contact sports might also have been part of some individuals' lifestyles, increasing risk factors unknowingly (Perry et al., 2016). As outlined above, it can be hypothesised that individuals who develop PD may have been less engaged in cognitive health-oriented behaviours compared to the general population. Studies have found associations between reduced engagement in cognitive, physical, and social activities and an increased risk of neurodegenerative diseases, including PD (Bakeberg, 2021). These activities are thought to promote brain health, potentially delaying or reducing the risk of PD. Concurrently, it is acknowledged that PD has a complex aetiology influenced by genetic, environmental, and lifestyle factors. Therefore, establishing a direct causal relationship between cognitive health practices and PD risk is challenging. For a more thorough comparison, controlled studies would be necessary to confirm whether individuals later diagnosed with PD differ significantly from the general population in their pre-diagnosis cognitive health behaviours.

Studies have highlighted that the Mediterranean, East Asia, and some Sub-Saharan African regions have lower PD rates, with factors like genetics, diet, environmental toxins, lifestyle habits, and even cultural practices potentially playing a role (Abbas et al., 2018). Similarly, studies of the Blue Zones show a lower prevalence of PD. Residents in these regions experience healthier aging with a lower incidence of health-related issues. Several lifestyle factors likely contribute to their lower prevalence of PD. More specifically, their diet is rich in antioxidants, which may protect brain cells from oxidative stress, a factor linked to PD, and low in processed foods while rich in whole foods, which may reduce inflammation and support brain health. Daily, low-intensity physical activity, such as walking, gardening, and manual labour, is integral to life in the Blue Zones. Such exercise is known to support neuroplasticity and may help protect dopamine-producing neurons, potentially reducing the risk of PD (Santacroce et al., 2024). Additionally, the low-stress lifestyle in those communities prioritises social connectedness and stress reduction, both associated with better mental and neurological health. Chronic stress is linked to higher PD risk, so these practices may indirectly protect against neurodegeneration (Dallé and Mabandla, 2018). In addition, low exposure to environmental toxins, such as pesticides and industrial pollutants, may contribute to the lower PD risk observed in the Blue Zones every day, particularly in regions where agriculture remains more organic or traditional. This suggests that lifestyle factors typical of the Blue Zones contribute to a lower PD risk and may be beneficial in promoting healthy aging more broadly. While no conclusive evidence directly links the Blue Zones to lower PD rates, their lifestyle patterns align with practices generally considered protective against neurodegenerative diseases. It is worth noting that, in addition to lower PD prevalence, populations in the Mediterranean region, Japan, East Asia, and parts of Sub-Saharan Africa tend to have higher life expectancies. These locations share common factors, including diets rich in natural foods, strong social networks, regular physical activity, and lower stress, all of which contribute to increased longevity compared to the global average. Thus, the Mediterranean, East Asian, and some Sub-Saharan African regions, as well as the Blue Zones, exemplify how lifestyle choices such as a nutrient-dense diets, regular physical activity, community engagement, and stress resilience can support longevity and overall well-being, potentially delaying neurodegenerative processes. A better understanding of the mediating and modulating factors underlying these processes may provide valuable insights into preventive strategies that could reduce the risk or delay the onset of PD.

HOLISTIC ACTION FRAMEWORK FOR PD INFLUENCES

This analysis attempts to conceptualise the factors influencing PD as driven by overlapping influences from direct and indirect sources (Fig. 1). This inquiry primarily categorises lifestyle, interoception, mindfulness, and environment as indirect influences and genetics and neuroplasticity as more direct influences. It can be argued that lifestyle, genetics, and environmental factors are viewed as both direct and indirect contributors to the disease, affecting various aspects of PD development and progression. Genetics is often viewed as a direct contributor to PD because specific gene mutations, such as SNCA (alpha-synuclein), LRRK2 (leucine-rich repeat kinase 2), PARK2 (parkin), and PINK1 (PTEN-induced kinase), can directly impact neurons, particularly dopamine-producing

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cells (De Rosa et al., 2015). These genetic mutations may initiate or accelerate the neurogenerative processes central to PD. Epigenetics can also indirectly affect PD, although it has direct consequences for gene expression and neurodegenerative processes through gene regulation and interaction with environmental factors. DNA methylation and histone modifications, for example, control gene upregulation or downregulation, allowing environmental factors and other influences to impact gene activity without altering the underlying DNA sequence. In PD, epigenetic mechanisms may influence cellular pathways and gene expression related to dopaminergic neuron loss, inflammation, and dopamine production, thereby affecting disease onset and progression (Ammal Kaidery et al., 2013; Labbé et al., 2016). Neuroplasticity directly influences how the brain responds to changes associated with PD. Adaptive neuroplasticity can help maintain function despite neuron loss, while maladaptive plasticity may exacerbate specific symptoms, directly influencing brain resilience to PD progression. Moreover, the perception of reality, mindset, engagement, and practices such as mindfulness, can directly influence neuroplasticity, thereby enhancing or limiting the brain's ability to adapt to PD-related changes. Promoting neuroplasticity through lifestyle habits, such as physical activity, social engagement, mental stimulation, and stress management, may help build a more resilient brain, potentially reducing vulnerability to PD or delaying its onset. Nevertheless, the neuroplasticity paradox means these mechanisms can manifest in both positive and negative ways. Furthermore, environmental factors can directly and indirectly impact PD, depending on the type and extent of exposure. Direct contact with toxins like pesticides, heavy metals, or pollutants has a direct neurotoxic effect on brain cells, contributing to PD risk by damaging dopamine neurons (Ball et al., 2019). Also, indirect aspects like chronic stress, diet, and access to healthcare, play a role. Pollution or long-term chemical exposure may increase oxidative stress or inflammation in the body, thereby indirectly influencing PD risk by affecting overall health, lifestyle choices, and exposure levels. Lifestyle behaviours, such as diet, physical activity, and stress management, further impacts PD risk and progression by shaping brain health and supporting or hindering neuroplasticity and resilience to neurodegeneration (Paola Caminiti et al., 2024). A balanced lifestyle supports both cognitive and motor functions, potentially delaying the onset or slowing progression of symptoms. However, lifestyle can also exert a more subtle direct influence in instances where chronic stress or poor diet triggers neuroinflammation or oxidative stress, which can impact neurons directly (Kip and Parr-Brownlie, 2023, 2022). Moreover, interoception, or the awareness of internal bodily states, indirectly affects PD by influencing stress regulation, emotional well-being, and self-awareness (Ricciardi et al., 2016). Although disrupted interoceptive awareness in PD is often seen as a consequence rather than a cause, healthy interoception may enhance both mental and physical resilience (Hazelton et al., 2023). Finally, mindfulness indirectly promotes emotional regulation, reduces stress, and improves interoception, collectively benefiting mental health and brain resilience. Hence, mindfulness practices may positively influence neuroplasticity, indirectly supporting PD management. The presented model emphasises that indirect influences, including lifestyle, environment, interoception, and mindfulness, promote resilience, while direct influences, such as genetics and neuroplasticity, directly impact PD pathophysiology. However, both types of influences are intrinsically linked and should be approached as components of an integrated system. It must be stressed that the current comparative observational study and thought experiment were designed to analyse naturally occurring differences in pre-diagnostic lifestyle patterns between PD patients and selected regions with lower PD occurrence. The aim was to establish associations and overlapping effects of biopsychological and lifestyle factors, creating a collaborative model for addressing PD risk and resilience factors. Unlike a meta-analysis, which aggregates and synthesises data from multiple studies, the present research focuses on direct comparisons with a defined sample. Data were extracted from PubMed, Web of Science, Google Scholar, and ResearchGate, and supplemented by relevant peer-reviewed research selected to reflect the study's focus on lifestyle patterns relevant to PD development. While further details on the dataset could enhance replicability, this study's objectives are rooted in contextual comparison rather than exhaustive statistical aggregation, providing unique insights into the relationship between lifestyle and PD. Moreover, this research attempts to frame the known factors driving PD as a result of a dynamic interplay among overlapping direct and modifiable pathways.

CONCLUSIONS

This research highlights the value of a comparative observational approach and thought experiment in developing the collaborative impact model, providing a theoretical foundation for future studies exploring the interplay of PD risk factors. By examining regions with low PD rates, such as the

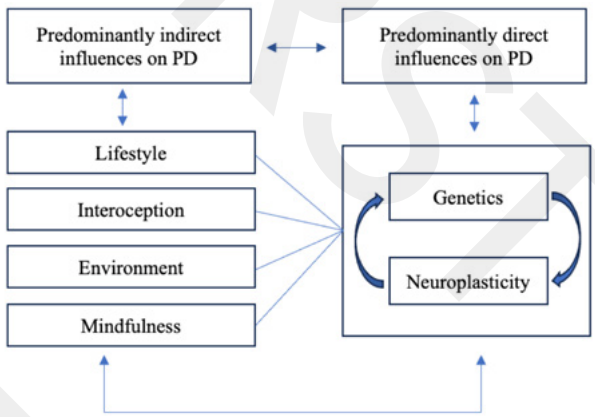

Fig. 1. Collaborative model for addressing PD risk and resilience factors. Source: the authors

Mediterranean, East Asia, Sub-Saharan Africa, and the Blue Zones, the study identifies lifestyle factors that may offer protective benefits against PD. Within this framework, neuroplasticity and genetics provide a flexible foundation, where repeated behaviours strengthen specific neural pathways. The direct impact model emphasises the roles of genes and neuroplasticity components influencing PD risk, while the modifiable impact model addresses interoception, mindfulness, lifestyle, and environmental factors. This framework implies that individuals are not bound by stereotypical behaviours. Through intentional changes and a supportive environment, neuroplasticity enables the reshaping of habits, potentially offsetting genetic predispositions. Overall, this research supports the holistic view that PD risk and occurrence are not solely determined by genetics or aging but are significantly shaped by lifestyle. Public health interventions could reduce PD incidence by promoting standards for healthy lifestyle practices in regions with low PD incidence. It is essential to highlight that the presented comparative observational study and conceptual thought experiment were aimed at examining the pre-diagnostic lifestyle of PD patients and areas with lower PD prevalence based on naturally occurring outcomes without manipulating the study environment. Additionally, the research aimed to establish associations and overlapping influences in order to create a collaborative model for addressing PD risk and resilience factors, and identify modifiable risk factors or lifestyle interventions that can be applied to prevent or manage PD. This study contributes to the growing understanding of PD by determining lifestyle factors that influence its onset and progression. Identifying these factors can help patients and at-risk individuals make informed decisions about their lifestyle, guide healthcare providers in developing targeted interventions, and foster a more holistic approach to disease management, ultimately improving quality of life. Future research should focus on refining direct and indirect impact modalities and evaluating preventive interventions across diverse populations. Longitudinal studies could further validate the protective effects of lifestyle modifications observed in low-PD regions while exploring the underlying biological mechanisms. This approach will deepen understanding of PD risk factors and support the development of targeted lifestyle-based prevention strategies.

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.

Data

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

Author contribution

Original concept of study: IU. Collection, recording and/or compilation of data: DK. Analysis and interpretation of data: IU, JJC, JB, WB. Writing of manuscript: IU, JC. Critical review of manuscript: JB, WB, JC. Final approval of manuscript: IU, JJC, DK.

References

Abbas MM, Xu Z, Tan LCS: Epidemiology of Parkinson's disease – East versus West. Mov Disord Clin Pract 2017; 5: 14–28.

Ammal Kaidery N, Tarannum S, Thomas B: Epigenetic landscape of Parkinson's disease: emerging role in disease mechanisms and therapeutic modalities. Neurotherapeutics 2013; 10: 698–708.

Bakeberg MC: Assessment of the determinants of cognitive decline and related symptoms in Parkinson's disease [Doctoral dissertation]. The University of Western Australia, 2021.

Ball N, Teo WP, Chandra S et al.: Parkinson's disease and the environment. Front Neurol 2019; 10: 218.

Cholewa J, Gorzkowska A, Kunicki M et al.: Continuation of full time employment as an inhibiting factor in Parkinson's disease symptoms. Work 2016; 54: 569–575.

Dallé E, Mabandla MV: Early life stress, depression and Parkinson's disease: a new approach. Mol Brain 2018; 11: 18.

De Rosa P, Marini ES, Gelmetti V et al.: Candidate genes for Parkinson disease: lessons from pathogenesis. Clin Chim Acta 2015; 449: 68–76.

Fan CK, Liao CW, Cheng YC: Factors affecting disease manifestation of toxocarosis in humans: genetics and environment. Vet Parasitol 2013; 193: 342–352.

Fang X, Han D, Cheng Q et al.: Association of levels of physical activity with risk of Parkinson disease: a systematic review and meta-analysis. JAMA Netw Open 2018; 1: e182421.

Goldman SM: Environmental toxins and Parkinson's disease. Annu Rev Pharmacol Toxicol 2014; 54: 141–164.

Gorzkowska A, Cholewa J, Małecki A et al.: What determines spontaneous physical activity in patients with Parkinson's disease? J Clin Med 2020; 9: 1296.

Hazelton JL, Fittipaldi S, Fraile-Vazquez M et al.: Thinking versus feeling: how interoception and cognition influence emotion recognition in behavioural-variant frontotemporal dementia, Alzheimer's disease, and Parkinson's disease. Cortex 2023; 163: 66–79.

Kalia LV, Lang AE: Parkinson's disease. Lancet 2015; 386: 896–912.

Kip E, Parr-Brownlie LC: Healthy lifestyles and wellbeing reduce neuroinflammation and prevent neurodegenerative and psychiatric disorders. Front Neurosci 2023; 17: 1092537.

Kip E, Parr-Brownlie LC: Reducing neuroinflammation via therapeutic compounds and lifestyle to prevent or delay progression of Parkinson's disease. Ageing Res Rev 2022; 78: 101618.

Labbé C, Lorenzo-Betancor O, Ross OA: Epigenetic regulation in Parkinson's disease. Acta Neuropathol 2016; 132: 515–530.

Lee JS, Jaini PA, Papa F: An epigenetic perspective on lifestyle medicine for depression: implications for primary care practice. Am J Lifestyle Med 2020; 16: 76–88.

Noyce AJ, Lees AJ, Schrag AE: The prediagnostic phase of Parkinson's disease. J Neurol Neurosurg Psychiatry 2016; 87: 871–878.

Olan A: A connection between factors causing diseases and diseases frequencies. It's application in finding disease causes. medRxiv 2023. DOI: 10.1101/2023.04.30.23289320. Available from: https://www.medrxiv.org/content/10.1101/2023.04.30.23289320v1.full-text.

Paola Caminiti S, Gallo S, Menegon F et al.: Lifestyle modulators of neuroplasticity in Parkinson's disease: evidence in human neuroimaging studies. CNS Neurol Disord Drug Targets 2024; 23: 602–613.

Perry DC, Sturm VE, Peterson MJ et al.: Association of traumatic brain injury with subsequent neurological and psychiatric disease: a meta-analysis. J Neurosurg 2016; 124: 511–526.

Postuma RB, Berg D, Stern M et al.: MDS clinical diagnostic criteria for Parkinson's disease. Mov Disord 2015; 30: 1591–1601.

Postuma RB, Lang AE, Gagnon JF et al.: How does parkinsonism start? Prodromal parkinsonism motor changes in idiopathic REM sleep behaviour disorder. Brain 2012; 135: 1860–1870.

Ricciardi L, Ferrazzano G, Demartini B et al.: Know thyself: exploring interoceptive sensitivity in Parkinson's disease. J Neurol Sci 2016; 364: 110–115.

Santacroce L, Bottalico L, Charitos IA et al.: Healthy diets and lifestyles in the world: Mediterranean and Blue Zone people live longer. Special focus on gut microbiota and some food components. Endocr Metab Immune Disord Drug Targets 2024; 24: 1774–1784.

Smalira J, Lęgas A, Przybysz B et al.: Role of selected nutritional factors in the prevention and treatment of Parkinson's disease – a review. Aktualn Neurol 2024; 24: 51–58.
Sompayrac L: How the Immune System Works. 7th ed., Wiley-Blackwell, 2022.
Uher I, Buková A, Švedová M et al.: Physical activity, natural environment, present moment awareness, and its consideration: a qualitative review. Sustainability 2021; 13: 8436.
Vodovotz Y, Barnard N, Hu FB et al.: Prioritized research for the prevention, treatment, and reversal of chronic disease: recommendations from the lifestyle medicine research summit. Front Med (Lausanne) 2020; 7: 585744.
Zhou H, Skolnick J: A knowledge-based approach for predicting gene-disease associations. Bioinformatics 2016; 32: 2831–2838.