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Editorial: Alzheimer's disease

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Alzheimer's disease is a neurodegenerative disease of old age that affects not only patients but also their caregivers, and is one of the leading factors of disability and mortality [1]. In such a situation, patients are ultimately bedridden and require 24-hour care. There are currently around 50 million Alzheimer's disease patients worldwide, and this number is expected to double every 5 years and increase to 152 million by 2050 [2]. Alzheimer's disease accounts for 70% of dementia cases worldwide [3]. Alzheimer's disease is among the ten most common causes of death in the world [4]. More effective symptomatic treatments or first-of-a-kind disease-modifying therapies for Alzheimer's disease remain a huge unmet medical need. These treatments have a significant impact on annual healthcare spending for Alzheimer's disease patients, which is estimated to reach $1.1 billion per year by 2050 [5]. Alzheimer's disease affects individuals, their families and the economy, with an estimated global cost of $1 trillion per year [2].

Alzheimer's disease is a neurodegenerative disease, which means it progresses over time. It is assumed that Alzheimer's disease begins 20 or more years before the first symptoms appear, and that changes in the brain are initially imperceptible to the affected person [3]. It is only after years of brain changes that patients notice symptoms such as memory loss. Symptoms appear because neurons die in the brain structures responsible for thinking, learning and memory. As the disease progresses, neurons in other brain structures also die. Amyloid plaques and neurofibrillary tangles are characteristic of the brain affected by Alzheimer's disease. To date, various theories have been put forward to explain the etiology of Alzheimer's disease, including the amyloid theory, abnormal tau protein phosphorylation, cholinergic and ischemic [1, 2, 6]. However, despite over a hundred years of research, the cause of Alzheimer's disease remains unresolved and no effective causal treatment has been found. Despite many years of research, the role of amyloid and tau protein as the cause of the disease is questioned [7]. Currently, other causes of Alzheimer's disease are considered, including cerebral ischemia [6]. The fact that Alzheimer's disease can manifest itself 20 or more years before the first symptoms appear creates an opportunity for early diagnosis if appropriate biomarkers are developed and provides a wide therapeutic window. The complexity of the disease means

that there is no effective causal treatment, so therapeutic activities focus mainly on alleviating clinical symptoms. Alzheimer's disease is one of the most debilitating and difficult to treat diseases in the world. The incidence of Alzheimer's disease in humans is steadily increasing due to the ageing population and this is one of the major challenges facing healthcare services. Over the years of research, significant progress has been made in the diagnosis of Alzheimer's disease. The etiology of the disease is still unknown, which makes causal treatment difficult. Despite this, many studies are being conducted in search of treatment using immunotherapy or various substances affecting different processes occurring during the development of Alzheimer's disease.

Although the neuropathological processes occurring during the development of this disease are known and fairly well described, we know little about what triggers them. The primary goal of this Special Issue was to create a repository of information and resources and to provide a platform for publishing cutting-edge research that advances our knowledge of Alzheimer's disease and its treatment. The underlying assumption was that advances in molecular, cellular, and systems neuroscience would open up new possibilities for the development of treatments that the world was eagerly awaiting. The research topic presented in the Special Issue entitled Alzheimer's disease aimed to shed light on the current state of knowledge and progress in research on this disease, as well as to consider future challenges. Of the 9 articles published in our Special Issue, 2 are original research and 7 are review articles on Alzheimer's disease. The content of these works is presented below.

Błaszczyk's [8] review provides a critical analysis showing how sleep abnormalities may influence Alzheimer's disease. Disturbed sleep patterns are also a major risk factor for developing mild cognitive impairment, which can convert into Alzheimer's disease. The sleep of patients with Alzheimer's disease is characterized by a gradual decline in slow wave activity and a significant shortening of the REM phase. Sleep disorders may accelerate the development of Alzheimer's disease neuropathology by promoting the accumulation of amyloid and tau protein. Changes in sleep quality resulting in amyloid aggregation and tau protein modification, which are thought to cause hippocampal degeneration and ultimately memory impairment. Sleep disorders are also associated with impaired neuronal activity in the cerebral cortex, which may accelerate and intensify neurodegenerative processes. Sleep disturbances due to cortical and environmental changes are often reported many years before the onset of clinical symptoms of Alzheimer's disease in older adults, making them a potential biomarker for Alzheimer's disease. After the onset and during the progression of mild cognitive impairment and Alzheimer's disease, sleep disturbances worsen even more rapidly. These suggest a complex bidirectional influence, which implies that sleep disturbances may both causally contribute to the development of Alzheimer's disease and be a consequence of its occurrence. The results demonstrate the unique potential of sleep-stage-specific changes as biomarkers for diagnosing, stratifying, and monitoring the progression of Alzheimer's disease. Therefore, interventions targeting sleep disorders in older adults and patients with mild cognitive impairment are proposed as a possible strategy to prevent or slow the conversion to Alzheimer's disease dementia. This review proposes a framework for generating new hypotheses and conducting additional research. This should include further understanding of the diagnostic and prognostic value of sleep as a biomarker of Alzheimer's disease and the therapeutic potential of modulating sleep functional networks in Alzheimer's disease.

In the original article, Holston [9] introduced an electrophysiological biomarker of Alzheimer's disease based on electrophysiological data from older adults who were functioning normally until the diagnosis of Alzheimer's disease. This indicator was observed as a progressive increase in theta waves in electroencephalographic studies. This rising theta wave began in the frontal region of brain and spread to the back of the head. This electrophysiological biomarker has the potential to help us better understand the course of Alzheimer's disease, but further research is needed to clarify its clinical utility. Further research may help in diagnosing Alzheimer's disease in its early stages and not relying solely on subjective assessment of the clinical symptoms presented. An electrophysiological biomarker may improve the treatment of people with Alzheimer's disease and enable preventive measures to be taken in the long, asymptomatic period of the disease, lasting up to 20 years [3]. Thus, the continuously increasing number of theta waves as an electrophysiological marker of Alzheimer's disease may help in treating the risk, onset

and progression of Alzheimer's disease. The results of this study may become the seed for further research that will contribute to the implementation of proactive care at all stages of Alzheimer's disease, changing the current prognosis for patients, leading from complete loss of independence to independent living.

The apolipoprotein E (ApoE) ε4 allele is a genetic risk factor for sporadic Alzheimer's disease. The presence of one or two copies of the ApoEε4 allele accelerates the onset of Alzheimer's disease by about 10–20 years [3]. Yuri et al. [10] developed a fully automated chemiluminescent enzyme immunoassay kit for ApoEε4 and Pan-ApoE on the LUMIPULSE® platform using monoclonal antibodies recognizing ApoE proteins, including ApoEε4-specific antibodies, and assessed their diagnostic agreement with ApoE genotyping. One hundred and seventy-eight serum samples were analyzed using the above method. Authors assessed its diagnostic compatibility with ApoE genotyping and demonstrated that chemiluminescent enzyme immunoassay for ApoEε4 and Pan-ApoE discriminates between ApoEε4 carriers in plasma. In conclusion, the authors stated that the combination of the ApoEε4 and the Pan-ApoE chemiluminescent enzyme immunoassay is useful in determining the ApoEε4 allele.

Mulumba et al. [11] point out that Alzheimer's disease continues to pose a major challenge in terms of rapid and accurate diagnosis, monitoring its progression, and developing effective treatments. The article highlights the role of neuroimaging as a modern diagnostic tool for monitoring the course of the disease and its treatment. Various imaging techniques, such as magnetic resonance imaging, single-photon emission computed tomography, and positron emission tomography, have been evaluated for their ability to detect amyloid plaques and neurofibrillary tangles in the brain, the characteristic neuropathological hallmarks for Alzheimer's disease. Neuroimaging enables visualization of Alzheimer's disease-related phenomena such as amyloid plaques, neurofibrillary tangles, neuroinflammation, and synaptic dysfunction, providing valuable information on the pathogenesis and progression of the disease. Imaging techniques have been shown to enable early detection of Alzheimer's disease, distinguish it from other conditions, and assess the effectiveness of its treatment. The authors indicate that this could change the approach to treating Alzheimer's disease. The authors further emphasize that neuroimaging has enormous potential to accelerate drug development by allowing scientists to assess the effectiveness of new therapies in real time. Overall, combining neuroimaging with the treatment of Alzheimer's disease has the potential to revolutionize the way we approach diagnosis, personalized treatment, and, most importantly, research into the etiology of this disease. Mulumba et al. [11] indicate that continued advances in neuroimaging technology will, in the future, provide hope for solving the mystery of Alzheimer's disease and ultimately for finding causal treatments. The authors believe that further research in this area will likely lead to the development of new imaging agents that will target different aspects of Alzheimer's disease neuropathology and enable earlier and more accurate diagnosis. They further note that the incorporation of neuroimaging into clinical trials and the development of personalized medicine could revolutionize the treatment and management of Alzheimer's disease in the future. Overall, neuroimaging is an indispensable tool at this time that will continue to play a key role in the fight against Alzheimer's disease.

Xiao and Zhang's [12] review provides a historical overview of the progress in drug development for Alzheimer's disease and the clinical trials of these drugs that ended in failure or little success. The authors searched Clinicaltrials.org web and reviewed randomized controlled trials published within the last 10 years. Of the 3,388 clinical trials for Alzheimer's disease, only 211 interventional trials registered for Alzheimer's disease met the eligibility requirements. Their review covers the development of drugs targeting amyloid, tau protein, neuroinflammation, neurotransmitter receptors, multitarget studies, non-pharmacological interventions, and the development of treatments for neuropsychiatric symptoms in dementia. They stated that current clinical trials are ongoing, but there are no results yet. Given the diversity of drug targets being studied, their review suggests future directions for drug design and clinical trials and refinements of current efforts to find a cure for Alzheimer's disease.

After analyzing currently available drugs for the treatment of Alzheimer's disease, the authors summarized their observations in the following proposals. There is an urgent need to develop a potent, selective and effective drug that could treat Alzheimer's disease patients and those predisposed to developing the disease. Given that tau protein has a more dominant correlation with Alzheimer's disease

than amyloid and is involved in the spread of the disease, the ongoing intensive work on tau protein immunotherapies for safety and efficacy may prove to be a cheaper and more effective treatment in the future compared to the currently approved anti-amyloid drugs. The interplay between amyloid and tau protein pathology remains incompletely understood, and targeting both pathways simultaneously could hold promise for developing effective treatments. The authors believe that future efforts should be directed, first, at increasing the effectiveness of drug delivery across the blood-brain barrier to brain tissue, which should reduce drug dose and limit side effects, and, second, at discovering therapeutic agents that will slow down the decline of cognitive functions, including memory. The authors believe that an appropriate new model of Alzheimer's disease that goes beyond the amyloid model will allow for specific, causal "precision" treatment of Alzheimer's disease, targeting pathology other than amyloid and tau protein. Since Alzheimer's disease manifests itself after 10–20 years, the best approach and current priority should be to predict its occurrence as early as possible, which would certainly facilitate treatment [3]. Combination therapies that target multiple pathways or multiple aspects of the same pathway may hold the key to effectively treating Alzheimer's disease through a personalized approach.

Medeiros [13] notes in his review that natural products such as phenolic compounds exhibit a wide range of bioactivities and urgently need to be tested and evaluated for their effect on inhibiting specific clinical symptoms of Alzheimer's disease. Phenolic compounds may inhibit or prevent the above changes by inhibiting the production of amyloid, proinflammatory factors, acetylcholinesterase and butyrylcholinesterase and preventing the development of neurofibrillary tangles. The author states that if phenolic compounds affect more than one mechanism in Alzheimer's disease, they may become a leading method of treating this disease. The author analyzed the scaffolds of several phenolic compounds leading to the highest activity against different phenomena in Alzheimer's disease, with the aim of finding a phenolic compound active against the most important pathological processes in Alzheimer's disease. He showed that compounds presenting scaffolds, such as rugosin E or isocorilagin, may have significant potential in the fight against Alzheimer's disease.

Gutierrez-Merino's [14] review presents hydrophobic peptides as amyloid antagonists that bind to the COOH terminus of amyloid and their endogenous creation in brain tissue, emphasizing the role of the proteasome as the source of these peptides. This review focuses on hydrophobic peptides that have been shown to be antagonists of neurotoxic forms of amyloid in in vitro and in vivo studies of Alzheimer's disease models. It has been found that the level of endogenous hydrophobic neuropeptides in the brains of Alzheimer's disease patients is different from that in the brains of healthy individuals of the same age. The author notes that hydrophobic neuropeptides may be useful biomarkers in assessing the development of sporadic Alzheimer's disease. In addition, hydrophobic anti-amyloid peptides that bind to its COOH terminus appear to be interesting candidates for the development of new Alzheimer's disease therapies that could be used in combination with existing therapies. The author briefly discussed the prospects and limitations of using these peptides in the treatment of Alzheimer's disease. The presented studies indicate that measurements of the levels of hydrophobic endogenous neuropeptides in the brain that bind to the COOH-terminal domain of amyloid can be used as additional biomarkers to determine the onset and progression of Alzheimer's disease. Furthermore, these peptides, by binding to the COOH-terminal domain of amyloid, indicate a direction for the development of new drugs for the treatment of neurodegenerative diseases with the presence of amyloid. Although the presented evidence opens new perspectives for the treatment of Alzheimer's disease, further research, including clinical studies, is necessary to overcome the current limitations in their translational application. The author draws attention to the protection of proteasome activity in the brain affected by Alzheimer's disease, which proteasomes produce hydrophobic peptides, and this problem is still a difficult scientific issue. Pharmacological methods of treatment aimed at stimulating proteasome activity in the brain have not yet been developed. Studies conducted in the brains of patients with Alzheimer's disease show that endogenous hydrophobic neuropeptides co-localize in amyloid plaques. Therefore, to monitor regional changes in the brain related to endogenous neuropeptides during the development of Alzheimer's disease at early stages, specific radiochemical markers will be needed, which are currently lacking. Most importantly, in the case of synthetic peptides that are to be used

as therapeutic agents, experimental studies are necessary on Alzheimer's disease models before they are used in clinical trials. These should include the development of nanoparticles that enable their efficient transport across the blood-brain barrier, as well as pharmacological, pharmacokinetic and toxicological studies.

The review by Sengupta et al. [15] focuses on the impact of receptor tyrosine kinases-mediated signaling and its role in cytoskeletal degradation in the penultimate stage of cellular degeneration in Alzheimer's disease. At the same time, the review by Sengupta et al. [15] indicates the possibility of therapeutic strategies targeting receptor tyrosine kinases-mediated pathways that may be effective through multifactorial effects on neurodegenerative cascades. Although there is evidence indicating the importance of cytoskeletal stability in the development of neurodegenerative changes, research on the participation of receptor tyrosine kinases in generating signaling cascades that affect the fate of the cytoskeleton is still insufficient to develop effective therapies. Therefore, further studies focusing on this pathology may lead to the development of effective therapeutic strategies, for example, in Alzheimer's disease. The authors emphasize that addressing the above topic may open the way to the development of more effective therapies for neurodegenerative diseases in the future.

In their review, Perez et al. [16] discuss the mechanism of action and therapeutic indications of neurostimulation devices in Alzheimer's disease. Rapid progress in neurostimulation technologies allows non-pharmacological treatment of patients affected by Alzheimer's disease. Neurostimulation therapies include electrical stimulation, which targets neuronal network connections in important brain regions such as the hippocampus to induce therapeutic neuromodulation of dysfunctional neuronal networks, and electromagnetic field stimulation, which targets anti-amyloid molecular pathways to reduce brain amyloid levels. These devices target specific and distributed subcortical and cortical areas, modulating neuronal activity and providing therapeutic effects in Alzheimer's disease. The authors presented several electromagnetic field-generating devices that have shown beneficial or harmful effects in in vitro and in vivo Alzheimer's disease models. They explained that the conflicting data may be related to the stimulation parameters of the devices used, such as frequency, penetration depth, power deposition, exposure time, cell type, and tissue dielectric properties. On this basis, the optimal stimulation parameters of devices using electromagnetic fields in Alzheimer's disease should be determined. The authors indicate that repeated electromagnetic field stimulation is the most appropriate method in the treatment of Alzheimer's disease.

This review suggests that factors preventing routine use of neurostimulation include a lack of well-controlled studies, inconsistent experimental results, and poor methodological standardization. Some electromagnetic neurostimulation devices have been shown to have beneficial effects in preclinical studies. However, their application in humans is fraught with challenges, including differences in anatomy, geometry, tissue layers, and depth of penetration. In addition, some devices are invasive, which carries risks, and others do not reach multiple areas of the brain affected by Alzheimer's disease at the same time. The review concluded that repetitive electromagnetic field stimulation is the most appropriate treatment strategy for Alzheimer's disease because it provides parameters relevant to human treatment and influences multiple processes related to Alzheimer's disease. It may stop the progression of Alzheimer's disease by acting against amyloid without causing bleeding or swelling. Moreover, repetitive electromagnetic field stimulation is not blocked by the blood-brain barrier and reaches all areas of the brain, including those responsible for memory. The rationale for repetitive electromagnetic field stimulation is the phenomenon of proteostasis, which is considered a risk factor for the development of Alzheimer's disease. Furthermore, loss of proteostasis is considered a potential cause of amyloid accumulation in Alzheimer's disease. Additionally, since heat shock factor 1 plays an essential role in proteostasis, autophagy, and amyloid clearance, this justifies the use of repetitive electromagnetic field stimulation to activate it and reduce brain amyloid level. Another advantage of repetitive electromagnetic field stimulation technology is its scientific basis, which indicates the interaction of the electromagnetic field with biological systems. Available studies have shown that cognitive functions impaired in Alzheimer's disease can be effectively regulated by repeated electromagnetic field stimulation.

This will enable the acceleration of currently urgently needed therapies for Alzheimer's disease towards electromagnetic field treatment.

The rising incidence of Alzheimer's disease highlights the need for effective causal treatments and a deeper understanding of its etiology. Despite enormous research efforts, there are currently no effective treatments for Alzheimer's disease, which makes the treatment problem one of the most urgent to solve. Available medications can only temporarily alleviate some symptoms of the disease. The lack of an effective cure is mainly attributed to its complexity, still not fully understood, multifactorial nature, which is why the development of new, inexpensive and effective treatments that could stop or slow down the progression of Alzheimer's disease continues to be a major challenge in the 21st century. Accordingly, there is extensive ongoing research into new drugs that can target multiple pathological mechanisms associated with Alzheimer's disease, including the development of multi-target or pleiotropic drugs to enable simultaneous action on several key processes associated with Alzheimer's disease, in contrast to the current strategy of single-target drugs. The appearance of mild cognitive impairment before the development of full-blown Alzheimer's disease offers promising opportunities for intervention to prevent disease progression over a relatively long period of time, up to 20 years.

The Special Issue presents the current state of knowledge about Alzheimer's disease and available treatments for this disorder and the strategies used to develop effective drugs. The proposed approaches are largely based on combining one process with several pharmacological substances, which allows for multi-target activities, with the possibility of identifying the shortcomings of already approved single-target drugs. Future drug development for Alzheimer's disease will require multiple multidisciplinary efforts to meet all structural requirements, remove unwanted interactions and reactions, and achieve clinical validation, which will be a difficult task. The authors point to the enormous potential of combining the pharmacological effects of different substances for the future development of new disease-modifying drugs based on multifaceted actions. Unfortunately, looking at the current state of advancement of research on new generation drugs and effective clinical solutions, we will have to wait a few more decades.

In this context, it should be emphasized that in the current situation, non-pharmacological treatment methods aimed at improving residual cognitive functions, such as non-invasive neurostimulation, are becoming increasingly popular due to limited side effects and the possibility of adapting protocols to an individual patient. However, the optimal stimulation targets and parameters necessary to induce long-term cognitive benefits have not yet been determined.

Future efforts in the field of Alzheimer's disease should focus on: modeling (etiology) of the disease to track the development of neuropathological changes, defining early biomarkers to identify individuals at risk of the disease, and defining personalized therapies to prevent and counteract disease progression. I personally hope that the extensive and intensive research presented will inspire future researchers to discover more effective therapies for Alzheimer's disease, as well as new methods for early diagnosis of this disease at an initial stage. I thank the authors for their contributions and hope that each article presented here will stimulate further research interests that will deepen our neuropathological understanding of Alzheimer's disease and its treatment, benefiting affected patients.

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Author contributions

RP: Conceptualization, Investigation, Writing—review & editing.

Conflicts of interest

Ryszard Pluta who is the Associate Editor and Guest Editor of *Exploration of Neuroscience* had no involvement in the decision-making or the review process of this manuscript.

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