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From plate to brain: Diet as a modifiable risk factor in brain diseases

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Abstract

Diet is more than mere sustenance; it plays a pivotal role in shaping brain health, influencing cognitive function, emotional well-being, and the risk of neurological disorders. Ongoing research reveals that dietary patterns exert protective and detrimental effects on the brain, positioning nutrition as a modifiable risk factor in neural health. Adherence to specific dietary regimens—such as the Mediterranean-DASH Intervention for Neurodegenerative Delay (MIND) diet, which integrates elements of the Mediterranean and Dietary Approaches to Stop Hypertension (DASH) diets—has been associated with reduced risk of cognitive decline and dementia. In contrast, certain cultural dietary practices have been linked to adverse neurological outcomes. A notable example is the Fore people of Papua New Guinea, whose ritualistic endocannibalism led to the transmission of kuru, a fatal prion disease. Additionally, protein malnutrition during the perinatal period disrupts brain development by reducing brain size, altering neuronal maturation, and impairing neurotransmitter systems, resulting in persistent cognitive and behavioural deficits.

This review critically examines current literature on the diet–brain health nexus, emphasizing the influence of macro- and micronutrients on neural function and disease progression. It advocates for integrating nutritional neuroscience into public health strategies to mitigate the global burden of neurodegenerative and neurodevelopmental disorders.

Keywords: Neuro-nutrition; Brain health; Diet; Neurodegeneration; Micronutrients

1. Introduction

Neural health is just as important as physical health, and the degree of neural healthiness an individual possesses would directly impact other domains of health [1,2]. According to the World Health Organization (WHO), health refers to complete physical, mental, and social well-being and not merely the absence of disease [3]. Hence, the concern and efforts to improve brain health, especially in pediatric and ageing populations, are highly justifiable [4]. The chemical composition of our brain can be influenced by the food we consume, which comes mainly from plants directly or indirectly by eating animals that feed on plants [5].

During fetal development, the mother's diet plays a crucial role in shaping the brain, and even after birth, the brain continues to depend on specific nutrients, such as fats and proteins, for proper maturation [6]. Poor maternal diets lacking essential nutrients like choline, folate, iodine, iron, protein, and vitamin D, which are critical for brain development and neuronal processes during the prenatal period, can lead to impaired neurodevelopment and a higher risk of cognitive and neurological disorders later in life [7,8]. A significant study by de Rooij et al. [9] demonstrated that prenatal undernutrition had a lasting impact on brain size.

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Food is a vital source of essential nutrients and a significant pathway for exposure to environmental contaminants such as heavy metals, organic pollutants, and pesticide residues found in harvested crops [10]. In addition, food processing, particularly heating, can also lead to the formation of beneficial and toxic Maillard reaction products [11]. Exposure to these chemicals during early childhood and postnatal development can lead to significant and irreversible damage to the developing brain [12].

Dietary pattern changes with age in individuals [13,14]. Recent studies suggest that for older adults, diets rich in antioxidants, anti-inflammatory components such as fruits, nuts, vegetables, and spices, and reduced caloric intake can help slow age-related cognitive decline and reduce the risk of developing neurodegenerative diseases [15]. Antioxidants play key roles in preventing brain diseases and slowing down existing neural and neurodegenerative diseases by reducing oxidative stress on neurons and mopping up free radicals in brain tissues [16,17,18].

Diet plays a vital role as a modifiable risk factor in brain disease prevention; nutritious eating enhances cognitive function and lowers neuroinflammation risk, while unhealthy eating habits can damage brain health and raise the chances of developing brain disorders [19,20,21]. This paper critically examines the growing body of literature on the relationship between diet and brain health, focusing on how macro- and micronutrients and food-transmitted infectious agents can influence neural function and the progression of neurodegenerative and neurodevelopmental diseases. It also advocates for integrating nutritional neuroscience into public health strategies to reduce the global burden of these disorders.

2. Nutrients and Brain Health

Nutrients are highly instrumental to ensuring the continual health of our bodies [22]. They are the key elements we derive from the food we consume, and they play significant roles in the nourishment of various tissues of our body [23]. Nutrients can be broadly classified into micronutrients and macronutrients as shown in Figure 1.

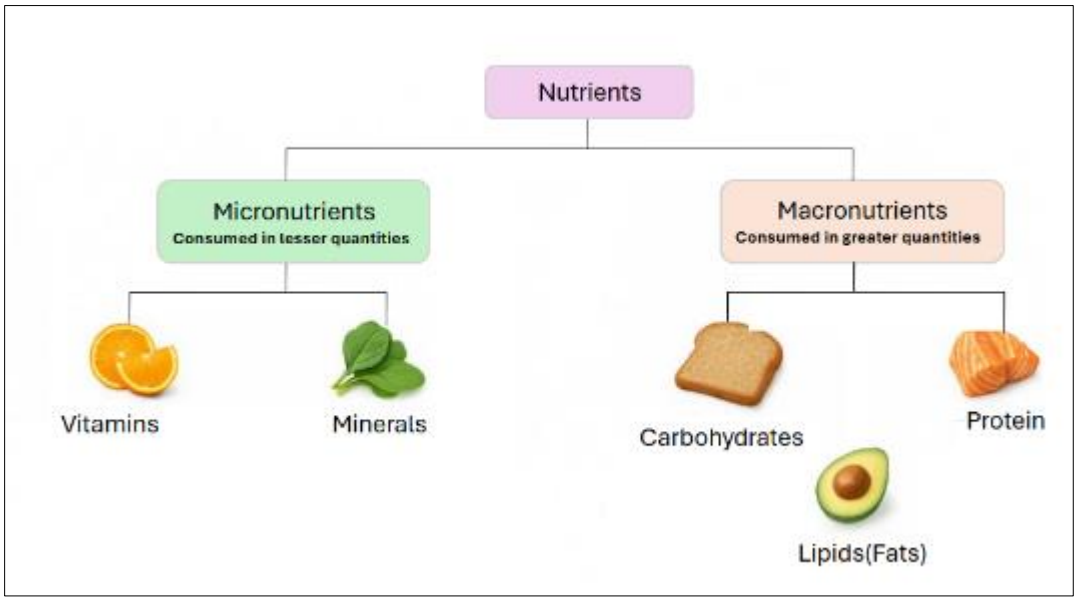

Figure 1 Classification of Nutrients into Macronutrients and Micronutrients. This infographic illustrates the two major nutrient categories (macronutrients and micronutrients) based on the relative quantities in which the human body requires them. Representative food sources are depicted for each category

2.1. Micronutrients

Micronutrients are vitamins and minerals that the body needs in small quantities for optimal functioning. They are typically divided into trace minerals, macrominerals, water-soluble vitamins, and fat-soluble vitamins [24].

2.1.1. Vitamins

Vitamins are organic substances essential in small amounts for normal cell function, growth, and development. They are typically composed of related compounds called vitamers [25,26]. While many of these vitamins are strictly obtained from diet, very few are produced by the body in sufficient quantities to maintain normal physiological and biochemical

processes [27]. Water-soluble and fat-soluble vitamins, with representative food examples shown in Figure 2, play a protective role in CNS diseases by preventing neuronal death [28].

B-Vitamins, including Vitamins B6, B12, and folate, play significant roles in homocysteine metabolism as co-factors [29]. Their functioning prevents the vascular and neurodegenerative effects of homocysteine build-up identified in B-vitamin-deficient patients [30]. Vitamin B6 is particularly involved in the transsulfuration pathway, where it catalyzes the conversion of homocysteine to cysteine via the provision of the enzyme cystathionine-β-synthase (CBS), which converts homocysteine to cystathionine [31]. A subsequent step, dependent on Vitamin B6, leads to the conversion of cystathionine to cysteine [30,31]. Also, vitamin B6 is involved in the remethylation of homocysteine to methionine. In the remethylation process, both Vitamin B12 and Methyl tetrahydrofolate (from folate) are needed as cofactors for Methionine synthase – the enzyme responsible for converting homocysteine to methionine [32]. Vitamin B12 has been shown to inhibit tau fibrillization and neurofibrillary tangle formation, thereby preventing tau aggregation and potentially slowing the progression and severity of Alzheimer's disease [33].

Vitamin D is a fat-soluble vitamin obtained from diet (vitamin D2 from plant sources and Vitamin D3 from animal sources) or produced in the body from 7-dehydrocholesterol [34]. Research has uncovered the neuroprotective properties of Vitamin D in addition to its classical role in calcium and phosphorus metabolism [35]. Vitamin D is vital in maintaining neurological homeostasis and protecting normal brain function. It has been implicated in preventing demyelination, neurodegeneration, and oxidative stress by suppressing the activation of reactive astrocytes and M1 microglia [36]. Also, $1,25(OH)_2D_3$ stimulates the expression of several neuroprotective factors such as antioxidant enzymes (for example, catalase, superoxide dismutase, and glutathione peroxidase) and neurotrophins, which enables it to decrease blood-brain barrier permeability, reduce leukocyte recruitment into the central nervous system, and enhance neuronal survival [37]. Significant impacts of vitamin D deficiency on biological processes include altered cell differentiation, signaling, protein expression, neurotransmitter release, and other biological processes within the nervous system [35]. All these impacts have been shown to play a role in the development of neurodegenerative diseases.

Vitamin E is a well-known potent antioxidant [38]. It can be obtained from the diet by consuming nuts, seeds, and green/leafy vegetables [39]. It protects the brain from oxidative stress by scavenging lipid peroxyl radicals and preventing a build-up of lipid peroxides [40]. Vitamin E isomers, α-tocotrienol (αTCT) and α-tocopherol (αTOC), support brain health by inhibiting oxidative lipid metabolism of arachidonic acid and blocking key enzymes like cytosolic phospholipase A2 and 12-lipoxygenase, thereby reducing brain injury and inflammation [41].

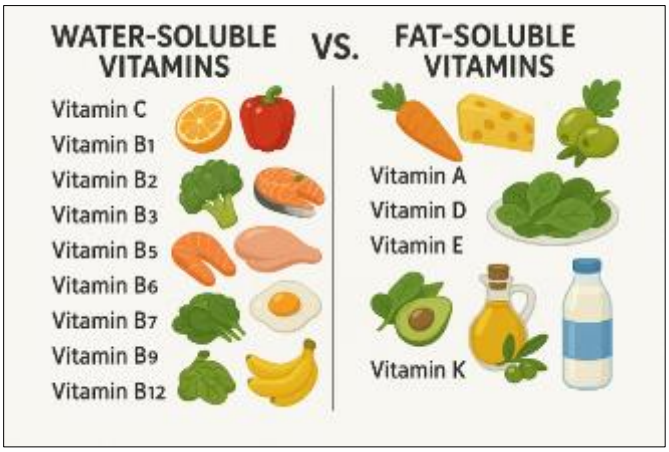

Figure 2 Vitamin type and associated food sources. The left panel illustrates water-soluble vitamins — Vitamin C and the B-complex group (B1 – Thiamine, B2 – Riboflavin, B3 – Niacin, B5 – Pantothenic acid, B6 – Pyridoxine, B7 – Biotin, B9 – Folate, B12 – Cobalamin)—with associated foods such as citrus fruits, leafy greens, eggs, and meat. The right panel displays fat-soluble vitamins (A, D, E, and K) in foods like carrots, cheese, dairy, nuts, and green leafy vegetables

2.1.2. Minerals

Apart from vitamins, minerals are essential inorganic substances that catalyze the various biochemical and physiological processes within the body and are also implicated in maintaining brain health [42]. Figure 2 shows different food sources for minerals that benefit brain health. Magnesium plays an essential role in maintaining brain

health by regulating neurotransmitter release and supporting nerve signal transmission by serving as a gatekeeper for NMDA receptors, where it prevents overstimulation and potential neuronal cell death [43].

Zinc is highly concentrated in several brain regions, such as the limbic system and auditory brainstem, where it serves as a cofactor for many enzymes and plays significant roles in neurotransmission [44]**.** Zinc is essential for producing acetylcholine, a neurotransmitter highly involved in memory and learning [45]. Calcium (Ca^{2+}) regulates key neuronal functions such as gene expression, membrane excitability, dendrite development, synaptogenesis, and other processes essential for information processing and memory storage [46].

Iron deficiency has been shown to reduce attention span, as highlighted in Kretsch's studies on healthy adults. The author's research revealed that declining iron levels were linked to decreased concentration, with one study showing a drop in attention span in men as iron levels decreased, and another connecting borderline anemia in women to reduced focus [47]. Increased extracellular potassium levels have been implicated in disrupting the resting membrane potential, altering neurotransmitter release, and impairing the function of voltage-gated ion channels, all of which can significantly affect neuronal excitability and information transfer [48].

Selenium is an antioxidant, promoting the synthesis of selenoproteins that mitigate oxidative stress, reduce inflammation, and decrease the risk of age-related cognitive decline in neurodegenerative diseases [49]. Sodium imbalance, characterized by elevated sodium content and enhanced blood-brain barrier (BBB) sodium transport, contributes to neuronal dysfunction and the development of brain edema, commonly observed during the early stages of focal cerebral ischemia [50].

A high-phosphate diet in a mouse model resulted in increased serum inorganic phosphate and upregulation of (Sodium-dependent phosphate transport protein 1) NPT1 in the cerebrum and cerebellum, accompanied by reductions in serum calcium, body weight, and the relative weight of the cerebellum [51]. Immunohistochemical studies have revealed that glial cells, especially astrocytes and microglia, accumulate higher levels of copper than neurons under normal and pathological conditions, emphasizing copper's role in modulating redox balance, neuroinflammation, and immune signaling [52].

Figure 3 Mineral-Rich Foods Essential for Neurological Wellbeing. Food sources rich in both trace minerals and macrominerals that support cognitive function and overall brain health

2.2. Polyphenols

Polyphenols are widely distributed in plant-based foods and have been found to contribute significantly to the reduction of neurodegenerative diseases and prevention of cognitive decline [53,54]. Polyphenol-rich foods include berries (they are an excellent source of anthocyanins), apples (an excellent source of quercetin), onions (containing quercetin and other polyphenols), dark chocolate (contains large amounts of flavonoids), and citrus fruits (including grapefruits,

oranges, and lemons) [55,56]. Polyphenols suppress inflammation, improve blood flow to the brain, and ensure the integrity of the blood-brain barrier [57]. They also promote neuroplasticity and inhibit the aggregation of specific proteins, such as amyloid proteins, which play a significant role in the pathology of Alzheimer's and Parkinson's disease [55,57].

2.3. Macronutrients

These are the nutrients the body needs in large quantities to function optimally. They are essential for growth, energy production, and nutrient absorption. Typical examples are Proteins, Fats, and Carbohydrates [58].

2.3.1. Fats (Saturated vs Unsaturated, Omega-3s)

Excessive consumption of saturated fats has been linked to detrimental effects on brain health, as they play a significant role in promoting neuroinflammation and contributing to cognitive decline [59]. However, consuming large amounts of unsaturated fats, namely Omega-3 fatty acids, Docosahexanoic Acid (DHA), and monounsaturated fats, is essential to maintaining the brain's structure and function [60]. In addition, various studies have reported that foods rich in Omega-3 fatty acids significantly improve cognitive and memory abilities in children and adults [61,62]. Healthy sources of these beneficial unsaturated fats are: Fatty fish (for example, Mackerel and salmon), flaxseeds, walnuts, and chia seeds [63].

2.3.2. Proteins

These macronutrients are essential for brain health as they provide the basic building blocks for optimal brain function [64]. Amino acids, the building blocks of proteins, are vital for maintaining brain cells and producing neurotransmitters and neuromodulators derived from tyrosine, tryptophan, histidine, and arginine [65]. Some studies have demonstrated that mild malnutrition or exposure to stress, combined with tryptophan deficiency, led to impaired cognitive performance in a cohort of military personnel [65,66]. Similarly, fortifying foods with tyrosine and tryptophan-rich substances significantly improves catecholaminergic neurotransmission, behavioral patterns, and increases stress resistance [67]. Tryptophan is the major precursor of serotonin; hence, high tryptophan levels induce increasing levels of serotonin, thereby suppressing depression and promoting calmness, while deficient states induce aggressive behaviors and alter pain sensitivity. [68,69].

2.3.3. Carbohydrates

They constitute a significant energy source for body tissues after metabolizing glucose. When under stress, the body can get energy from various sources simultaneously; however, the brain differs from other body tissues as its primary energy source is glucose, which is selectively passed across the blood-brain barrier [70]. Hence, the glucose passage rate across the blood-brain barrier determines the amount of glucose available to the brain. This affects attention, cognition, and memory [71,72].

3. Protective Dietary Patterns and Brain Health

A balanced diet forms the foundation for an individual's physical, mental, psychological, and social fitness [73]. Diet plays a crucial role in maintaining overall health, preventing disease, supporting cognitive development, and influencing character and behaviour, making it a key modifiable risk factor for promoting brain health through appropriate dietary patterns [74].

3.1. Mediterranean Diet

This is also popularly referred to as 'the Medi diet.' It is a healthy eating pattern derived from and fashioned after the traditional cuisines of countries around the Mediterranean Sea coast [75]. It incorporates and encourages the consumption of more plant-based foods than the typical Western diet [76]. The diet is rich in vegetables, legumes, whole grains, olive oil, fruits, and moderate amounts of fish, dairy, and poultry products. Also, it allows for small quantities of red meat and a minimal range of processed foods [75].

Several population-based and prospective epidemiological studies [77] have examined and validated the effectiveness and efficiency of this diet. They have shown that adherence to the Medi diet appears to have a protective effect against stroke, cardiovascular disease, obesity, diabetes, hypertension, cancer, and, most recently, neurodegenerative diseases such as Parkinson's disease and Alzheimer's disease [78]. More importantly, adherence to this diet has been associated with a reduction in the extent of cognitive decline [79].

3.2. Dash Diet

This diet, fully referred to as 'Dietary Approaches to Stop Hypertension', is also highly effective for improving neural health [80]. Although the link between the DASH diet and neural health reveals a crucial connection between blood pressure regulation and brain health, the primary aim of this diet is to manage blood pressure levels and prevent hypertension [80,81,82]. Its approach is based on consuming balanced proportions of vegetables, fruits, lean protein, low-fat dairy, and whole grains, while the intake of red meat, sugary drinks, processed foods, and sodium is limited [80,83]. The ripple health effect of this diet contributes to high vascular integrity and low incidence of neuronal cell death and inflammation.

Furthermore, the Mediterranean diet is commonly combined with the DASH diet to improve health outcomes. The combined diet is referred to as the Mediterranean-DASH Intervention for Neurodegenerative Delay (MIND) Diet [84,85]. This combination is targeted at reducing the risk of dementia, as the components (chiefly berries, leafy green vegetables, and nuts) are carefully selected to yield high health benefits to the brain [85].

4. Harmful Dietary Patterns and Mechanisms

4.1. Sugar and ultra-processed foods

Consuming high amounts of sugar and ultra-processed foods (UPFs) could result in insulin resistance and inflammation, as these foods trigger various inflammatory responses across several metabolic pathways and organs [86,87]. These physiological responses are clinically significant as the hippocampus, the brain's seat of memory and learning, becomes affected and impaired in functioning [88]. The consumption of ultra-processed foods could contribute to brain atrophy and reduced plasticity and dendritic spine density in the hippocampus, impairing cognitive function [89].

4.2. Food-Borne Toxins and Pathogens

Consuming contaminated food, commonly called food poisoning, can have widespread consequences; beyond impacting peripheral tissues, it may also pose serious risks to the central nervous system [90,91]. This is the case with known neurotoxins such as pesticides, acrylamide, and mercury, which result in behavioural changes, headaches, tremors, meningitis, and death in many cases [92]. Furthermore, exposure via food to parasites such as the tissue cysts (bradyzoites) of *Toxoplasma gondii*, the metacestodes of *Taenia solium*, rotavirus, prions, and Salmonella, among many others, could result in several fatal neurological complications [93].

Furthermore, certain brain diseases, such as Creutzfeldt-Jakob Disease and kuru, are linked to consuming contaminated food, where misfolded prion proteins in infected food cause brain cell dysfunction [94,95]. These prions, which spread to the brain, are often transmitted through infected meat, such as cattle consuming scrapie-infected bone meal, leading to conditions like mad cow disease (Bovine Spongiform Encephalopathy) and its human variant, vCJD (variant Creutzfeldt-Jakob Disease) [96].

Kuru, a fatal neurodegenerative disease first identified in the Fore tribe of Papua New Guinea, was found to be associated with the practice of cultural cannibalism. In this ritual, the relatives of the deceased were compelled to cook and consume the body, with women and children specifically instructed to eat the brain of the deceased. This practice contributed to the transmission of the prion disease [97]. These diseases highlight the critical role of diet in the transmission of prion infections [98,99].

5. Gut-Brain Communication

Communication between the gut (or digestive system) and the brain occurs via a two-way channel called the 'gut-brain axis' [100]. The communication channel usually involves neurotransmitters, biochemical signaling, and the vagus nerve. This highlights how substances and microorganisms present in the gut could gain access to the brain and influence its functioning, health, and behavior [101]. The gut microbiome could interact with glial cells (microglia, astrocytes, and oligodendrocytes) via this channel and influence the incidence of various brain diseases [102].

Furthermore, exposure of the gut to pathogens or disruption of the commensal-pathogen balance within the gastrointestinal tract could transmit harmful pathogens or toxins into the brain via the gut-brain axis [103]. The usual response to such exposure is neuroinflammation. At the same time, acute inflammation of the glial cells and other associated cells has been established as protective and beneficial to ensuring neural health; however, chronic

inflammation significantly damages brain cells, leading to neurodegeneration and impairment of memory and learning ability [104].

6. Diet-linked Neurodegenerative Conditions

6.1. Alzheimer's Disease (AD)

This is the most common type of dementia, which affects about 5.2 million Americans over the age of 65. It is the seventh leading cause of death worldwide and one of the significant causes of dependency and disability among the elderly [105]. It often starts unnoticed and progresses to a full-blown manifestation of impaired cognitive and behavioral functions [105]. Malnutrition is one of the secondary antecedents to dementia [106]. Multiple studies, including meta-analyses, have consistently shown that individuals with Alzheimer's disease or dementia have lower serum concentrations of vitamins A, B (particularly B6, B9, and B12), C, D, E, and K compared to cognitively healthy controls, and a cross-sectional study further demonstrated that higher levels of vitamin B12 and folate were associated with improved cognitive performance and potential therapeutic benefits in AD patients [107].

Concerning a sugar-rich diet, AD is highly related to diabetes, which is referred to as 'type 3 diabetes', as a decrease in brain glucose metabolism is observed in dementia patients [108]. While the brain constitutes just about 2% of total body weight, it consumes one of the most considerable proportions of energy, with about 25% of total body glucose consumed in the resting awake state. Glucose enters the brain through insulin-independent transporters (GLUT1 and GLUT3), while GLUT4 plays a role during high synaptic activity, suggesting that insulin resistance can still impact brain synaptic function [109,110]. Furthermore, impaired glucose metabolism starves the brain of essential glucose supply, stimulates neuroinflammation, and accelerates the accumulation of amyloid-beta and tau [111]. Also, advanced glycation end products (AGEs) interact with the receptor for advanced glycation end products (RAGE) located on various brain cells to modify proteins within the brain and contribute to the formation of amyloid plaques and neurofibrillary tangles [112].

6.2. Parkinson's Disease (PD)

It is the fastest growing neurodegenerative disease characterized by selective degeneration of dopaminergic neurons in the substantia nigra and the accumulation of α-synuclein [113]. More than 85% of cases are of unknown etiology, and among the risk factors of PD, exposure to neurotoxic pesticides stands out [113]. Pesticides that can accumulate in the food chain, contain paraquat, rotenone, dieldrin, and maneb, which have been shown to affect critical cellular pathways that lead to the disruption of dopamine metabolism [113,114].

However, some dietary factors have been found to increase or decrease the risk or progression of the disease. Elevated homocysteine levels, resulting from high dietary methionine intake and insufficient B-vitamin status (particularly folate, vitamin B6, and vitamin B12), have been implicated in the degeneration of nigrostriatal dopaminergic neurons in Parkinson's disease [107]. Consumption of high levels of processed foods, saturated fat, and red meat has been found to increase the risk. In contrast, consuming vegetables, fruits, olive oil, and diets rich in antioxidants appears to be protective [115].

6.3. Multiple sclerosis

This inflammatory condition is characterized by demyelination, inflammation, and neurodegeneration in the central nervous system [116]. Vitamin D, known for its potent anti-inflammatory effects, has been linked as a risk factor for multiple sclerosis when deficient, and supplementation has been shown to enhance white matter oxidation [117]. Some researchers have reported the anti-inflammatory role of the active form of Vitamin D, $1,25(OH)_2D_3$, and the significant contributions observed in the remyelination process via the promotion of stem cell proliferation and its ability to drive the differentiation of neural stem cells into oligodendrocytes [118].

7. Clinical and Epidemiological Implications

At-risk individuals and people living with diagnosed brain diseases can benefit from modified dietary lifestyles such as DASH or MIND diets to supplement their chemotherapeutic treatment [119,120]. Enhancing public awareness of the health implications associated with food additives is essential, particularly in promoting informed choices and regulatory compliance in processed food production [121]. In addition, strict hygiene in food handling and careful assessment of meat sources are crucial to reducing the risk of contamination with harmful biological or chemical agents [98].

Malnutrition has been flagged as a contributor to neural diseases, hence the need to fortify foods with essential amino acids such as Tryptophan, tyrosine, and omega-3 fatty acids [122]. Also, the Food and Drug Administration agencies should ensure all food and drug manufacturers comply with strict manufacturing and labeling standards.

The critical role of diet in brain health, from prenatal development to aging, cannot be overstated. Beyond lifestyle modifications and chemotherapy, nutrition is a fundamental factor in preventing brain diseases, representing a key modifiable risk factor that can shape the future of neurological health.

8. Conclusion

Dietary patterns and food processing methods have a profound impact on brain health across the lifespan. Expanding evidence-based research in nutritional neuroscience is crucial to revising dietary guidelines and integrating nutritional strategies into the prevention and management of brain diseases. Enforcing stringent regulations against the consumption of foods that may negatively impact brain health can serve as an additional public health measure to reduce the global burden of neurological disorders.

Compliance with ethical standards

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Disclosure of conflict of interest

The authors declare no conflict of interest.

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