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Cognitive Decline and Mental Distress: A Potential Mismatch between the Contemporary Diet and the Evolved Brain

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Review Article

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Abstract

Archeological studies describe that human cognition evolved with the increase in human brain size. Evidence also suggests that our ancestors' diet, which was a high-energy-nutrient-dense diet, contributed significantly to the complexity of the brain as dietary components act as structural and functional precursors. Therefore, the brain requires a repertoire of nutrient-dense food to grow, maintain and repair different brain structures. Additionally, ancestor males and females had different physical responsibilities that may have necessitated different energy requirements and may have led to a disparity in food consumption. Therefore, the gender-based differential food consumption of our ancestors and their energy intake may explain the differential brain connectivity between human males and females. Therefore, a potential mismatch may exist today between contemporary diet and the evolved brain which may be contributing to cognitive decline and mental distress in humans. Hence, the purpose of this review is to explore the evidence in the literature that potentially supports this hypothesis to better understand the role of diet on brain health.

Keywords: Diet; Brain Health; Cognitive Decline; Mental Health; Brain evolution

Abbreviations: WD: Western Diet; MD: Mediterranean Diet; GI: Gastrointestinal; LS: Limbic System; PC: Prefrontal Cortex; CLC: Cortical-Limbic Circuit; GM: Grey Matter; WM: White Matter; PC: Pre-Frontal Cortex; HPA: Hypothalamic-Pituitary-Adrenal; OCMP: One-Carbon Metabolism Pathway; SAM: S-Adenosyl Methionine; LDL: Low Density Lipoprotein; FRF: Farm-Raised Fish; PE: Physical Exercise; BDNF: Brain-Derived Neurotrophic Factor; OS: Oxidative Stress.

Introduction

The notion of "you are what you eat" has increasingly been proven accurate as the field of nutritional science and nutritional genomics decipher the role of nutrients at the cellular and molecular levels. However, "you are what your brain eats" is a novel concept. The emerging field of nutritional neuroscience is laying the ground for work on the effect of diet on brain structures and health.

The diet provides structural and functional precursors needed for optimal brain health [1]. Therefore, dietary patterns may influence different brain functions. Epidemiological studies link the Western diet (WD) to mental disease and cognitive decline while the Mediterranean diet (MD) typically associates with mental wellbeing and improved cognitive functions [2,3]. Neurocognitive deficits typically associate with many neuropsychiatric disorders; therefore, these conditions often need to be evaluated concomitantly. Fossil and archeological studies reveal that human cognition evolved with the increase in human brain size [4]. Evidence also suggests that our ancestors' diet, which was a high-energy-nutrient-dense diet, contributed significantly to the increase in brain size and cognitive evolution [5]. Additionally, males and females had different physical responsibilities that may have necessitated different energy requirements. Thus, gender-based differential food and energy intake may explain the differential brain connectivity between females and males [6]. Therefore, a mismatch between the quality of our ancestors' and contemporary diets may be at the heart of cognitive decline and mental health. Hence, the purpose of this review is to explore the evidence in the literature that potentially supports this hypothesis to better understand the role of diet on brain health.

The Evolution of the Human Brain

To gain insights into the evolution of brain size with the emergence of the genus Homo, anthropologists used an energetic approach to compare the energy demands associated with brain size in modern humans relative to hominin fossil records. There is substantial evidence supporting the notion that dietary components played a significant role in the evolution of the human brain [5,7,8]. Studies of hominin fossil and dental records suggest that feeding adaptations and food preferences were part of human evolution as well [9,10]. Additionally, archives were examined to assess changes in brain size, and dietary patterns associated with the evolution of early Homo. One evident fact is that the brain evolved to be a high metabolic organ. A sizable percentage of the human nutritional requirements are linked to the energy demands of human large brains, which are about 16 times that of the skeletal muscle [11]. Several studies support the link between brain size and dietary quality. A positive relationship between relative brain size and dietary quality exists for early humans [12]. In addition, there is a strong positive relationship between nutrient density and the amount of energy allocated to the brain [13]. Humans tend to be at the positive extremes for both criteria, having the largest relative brain size and the highest quality diet. Therefore, the evolution of larger hominin brains may have necessitated a consumption of a nutrient-dense diet to support the increased metabolic demands of encephalization. This notion is further supported by the different dimensions and anatomy of the human gastrointestinal (GI) tract that reflect an adaptation to digestion and absorption of a diverse high-quality diet [14]. Together, these data suggest that the consumption of a nutrient-dense diet stimulated the expansion of brain size over the course of human evolution. Therefore, a high-quality diet may be needed to maintain the integrity of the contemporary human brain.

The Limbic System and the Pre-Frontal Cortex

The limbic system (LS) has been described to be among the oldest brain structures based on a phyletical classification. LS controls basic functions such memory, emotions and behaviors (including motivation and arousal). Although LS is wired to stimulate strong emotions, a balance between neuro-activation and neuro-inhibition of the limbic system is in place to keep the extreme emotions at bay [15]. This rheostat is further underlined by the evolution of the prefrontal cortex (PC) which is critical to many cognitive abilities. PC evolution is believed to involve specific reorganization of neural circuitry in response to specific selection pressures. Consequently, the cortical-limbic circuit (CLC) connectivity supports further regulation of emotions by empirically processing environmental cues to keep negative emotions under control [16]. Based on phylogenetically characterization of frontal cortical circuitry, there is evidence that indicates that this part of the brain is among the most recent evolved parts [17]. Therefore, abnormal PC development induces abnormalities in the CLC connectivity leading to psychiatric disorders and neurological disorders.

Male Versus Female Brain

Although the sexual dimorphism of the brain is still debated in the literature, there is a strong evidence that functionality of female and male brains differ [18-20]. Many reports that describe the gender-based difference in the human brain attribute the disparity to selection pressure as part of evolution. Variability of social life, human behavior and parenting role necessitated differential cognitive requirements [21,22]. However, a very recent report describes the diet as being the major contributor to brain evolution [5]. Additionally, differential physical demands of human males and

females may have dictated preference for food consumption through evolution. Functionality of brain circuits may represent connectivity between regions, type of circuitry activation and brain region recruitment under certain tasks. With the emergence of the epigenetic field, better insights were gained on the mechanisms of dietary adaptations. Therefore, dietary patterns between males and females may have played and may continue to play a role in brain health. Consequently, failure to meet the high-quality nutrient demands of the contemporary brain may be the root cause of cognitive decline and mental disease in humans. In addition, it may also support the evidence in the literature that describes that females have a higher risk for anxiety and depression compared to males while males carry the higher risk for developing schizophrenia [23].

Grey Versus White Matter: Why do they Matter?

The central nervous system consists of two distinct types of tissues, grey matter (GM) and white matter (WM). GM is concentrated in information-processing areas such as the cerebral cortex and the limbic system while WM represents the network connectivity with the processing regions. Although there are conflicting reports in the literature on the actual GM and WM densities between female and male brain, the consensus is that women have thicker GM in the cortices, potentially more WM connecting with subcortical regions and smaller amygdala volume compared to men[24-27]. On the other hand, men tend to have more GM in the subcortical regions; however the hippocampus is not sexually-dimorphic [28]. Therefore, females may require a larger repertoire of nutrients compared to men to maintain the larger complex connectivity in their brain [29].

The Hippocampus

The hippocampus, part of the limbic system, mostly comprises of GM. It houses nerve fibers, hippocampal pyramidal cells, cell bodies of interneurons and an elaborate dendritic meshwork. Additionally, the hippocampus controls spatial learning, declarative memory, and emotions. Therefore, the hippocampus is an integral junction between different brain regions essential for cognitive function and mood regulation [30]. The hippocampus grows afferent neural circuit with emotion-related brain regions, such as the pre-frontal cortex (PC) and the amygdala. While the PC rationalizes emotions, the amygdala is responsible for the fear conditioning. Therefore, an optimal CLC circuitry is critical for regulation of emotions and mood. The hippocampus has extensive glucocorticoid and glutamate receptors that modulate the hypothalamic-pituitary-adrenal (HPA) axis activity. Chronic stress-induced hyperactivity of HPA axis leads to structural damages in key brain regions, including the hippocampus. Consequently, HPA dysregulation induces hippocampal atrophy secondary to alterations in neurochemistry and neuroplasticity [31]. Moreover, mental distress negatively impacts CLC which induces abnormal amygdala functional connectivity in areas that integrate affective processes [32]. Therefore, structural and functional disturbances in these brain regions increase risk of cognitive decline and mental distress [33,34].

Hippocampal Atrophy

Reduction in hippocampal volume may be an age-related phenomenon but may result from pathological conditions as well. In both instances, hippocampal atrophy relates to structural abnormalities and associates with cognitive decline and depressive symptoms [35,36]. Multiple theories have been proposed to explain the etiology behind the reduction in hippocampal volume including poor diet, stress and lack of neurotrophic factors [37-39]. However, a poor diet can exacerbate the effect of stress and can further decrease neurotrophic factor release [37]. This fact is evidenced by many reports that describe the etiology behind an unhealthy diet in promotion of cognitive decline and mental disorders [2,40,41]. Equally, emerging reports support the role of an MD in reducing risk of anxiety and depression as well as of cognitive decline [42-44]. Although hippocampal atrophy could be due to an amalgamation of homeostatic disturbances, a healthy diet has the potential to reduce inflammation, oxidative stress and improve neurogenesis necessary for promotion a healthy hippocampal volume [45-47]. In fact, large hippocampal volume in men associates with increased behavioral inhibition such as regulation of defensive approach behaviors and anxiety traits [48]. Taken all together, deficiencies of key nutrients necessary for the dynamics of hippocampal neurogenesis and synaptogenesis may impact cognitive functions and mental health [49,50].

Cortical Thickness

Cortical thickness has been linked to higher cognitive abilities and mental wellbeing in human models [51]. The frontal cortex region regulates impulsivity, emotions and other aspects of executive functions [52]. Consequently, The thickness of cortices produces an efficient control over the subcortical amygdala-hippocampal system that

jointly regulates mental wellbeing [53]. The thickness of the cerebral cortex is dependent on the density of GM and sub-WM structures. The dense WM fibers, part of the CLC, are critical for regulation of emotional information. Reduced GM volume in PC regions and/or WM lesions in the CLC induce dysregulation of the limbic system. Subsequently, hyper-activation of the amygdala leads to mental diseases [54-56]. Furthermore, cerebral cortical structural abnormalities, including a decrease in cortical thickness, associate with major depressive disorders and other psychiatric conditions [57,58]. However, activities that increase cortical thickness, such as meditation and exercise, improve cognitive function and mental wellbeing [59-61].

Diet and Cortical Thickness

Several dietary bioactive compounds promote cortical thickness. Macronutrients and micronutrients of MD strongly correlate with brain cortical thickness [62]. Fish, nuts, leafy vegetables, low intake of meat and olive oil were described to contribute to cortical thickness and to a slower rate of brain volume loss [63]. Increasing evidence suggests that adherence to MD may be inducing epigenetic modifications protective against mental disease and cognitive decline with a trans-generational effect [1,64,65]. Epigenetic modifications in key genes may confer protection against day-to-day and seasonal alterations in dietary patterns inducing resilience against minor cellular insults. The MD is also rich in folate which is an integral part of the one-carbon metabolism pathway (OCMP). In the OCMP, 5-methyl tetrahydrofolate assists with generation of S-adenosylmethionine (SAM), the universal methyl donor for DNA and histone methylation reactions as well as for dopamine biosynthesis [66]. Epigenetic modifications are increasingly explaining causality of complex diseases. Adaptation to environmental factors, including diet, modulates cellular signaling to alter gene expression. Therefore, the epigenome is a potential contributor to cognitive function and mental health [67]. Consequently, a competent OCMP has the potential to promote dopamine biosynthesis as well as to induce advantageous epigenetics marks that support mental wellbeing and cognitive functions.

The Western Diet

The hallmark of WD are fast food, meat and starchy carbohydrates. WD is typically devoid of a spectrum of nutrients needed for optimal brain structure and function (such as omega-3 fats, polyphenols and antioxidants). Deficiencies of these nutrients increase risk of mental distress and promote brain atrophy [2]. Aging is a risk factor for hippocampal atrophy due to increased brain oxidative stress with age [68]. However, consumption of a WD seems to accelerate brain atrophy. A cross-sectional study included 5731 mid-aged and older participants reported that a Western-diet style associated with anxiety in men and women. Conversely, a healthy diet quality score was inversely associated with depression and anxiety [69]. In a four-year longitudinal study, led by the same research team, individuals who followed 'an unhealthy diet' similar to WD exhibited a rapid atrophy of the left and right hippocampi [37]. Therefore, regular consumption of WD with age may induce rapid brain structural changes that could potentially lead to cognitive decline and mental distress. Additionally, consumption of animal lipids with age may increase serum low density lipoprotein (LDL). LDL is commonly known as the 'bad cholesterol' because it comprises of high ratio of lipids to protein; hence the low-density nomenclature. Therefore, LDL is intrinsically prone to oxidation which may start the process of atherogenesis [70]. Atherosclerosis is an age-dependent phenomenon, and build-up of plaque in cerebral arteries has been linked to neurodegenerative diseases and brain atrophy [71,72]. Nevertheless, a Swedish study also reported a negative association between meat intake and brain volumes in older adults and a positive association with eicosapentaenoic acid (EPA; 20:5n-3) and docosahexaenoic acid (DHA; 22:6n-3) from marine fish [73]. Although meat consumption may be beneficial for mental wellbeing in younger adults as depicted previously from our laboratory, it may be not as much advantageous with increasing age [74]. However, many factors could dictate individual responses to animal fat consumption like genetic factors (such Apo E4 genotype) and diseases that promote vascular pathogenesis (such as diabetes and cardiovascular diseases) [75,76].

Marine Fish Fat: The Forgotten Shield

With the emergence of fish farming and the increasing reports about potential mercury contamination of marine fish, farm-raised fish (FRF) are becoming a growing commodity. Unlike marine fish, FRF may be devoid of EPA and DHA when marine ingredients in FRF diets are replaced with non-marine feed. EPA and DHA are essential fats from the omega-3 family which contribute significantly to brain health. DHA promotes neurite outgrowth in the hippocampus and synaptic function [77]. DHA has gained lots of interest from the scientific community for its role in the evolution of the human intelligence and higher cognitive functions [8,78,79].

Additionally, DHA controls a wide-spectrum of physiological processes including neurotransmitter release, transmembrane receptor function, intracellular signaling, axon myelination, neuroinflammation, and neuronal differentiation [80]. It is mostly located in membrane phospholipids and especially concentrated in GM which contributes extensively to subcortical volume [81]. Therefore, deficiency in DHA may induce homeostatic disturbances potentially by altering synaptic transmission and reducing GM volume [82,83]. EPA also plays a significant role in maintaining functional integrities of brain structures. EPA mediates neuroprotective functions through cascade of cellular events that culminate in the production of anti-inflammatory molecules [84]. Both EPA and DHA contribute anti-oxidative stress, anti-apoptotic and pro-neurogenesis properties [49]. Furthermore, EPA promotes proliferation of progenitor neural cells while DHA stimulates their differentiation [85].

Considering this information jointly, the combination of EPA and DHA deficiencies in the WD may have a significant effect on the HC and the subsequent cross-talk with the amygdala. The innate brain clock coupled with reduction in neurogenesis culminate in atrophy of subcortical brain regions over time that increases risk of mental distress in humans. This proposed scenario may suggest that absence of dietary components that support the structural integrity of the GM in the limbic system may increase risk of cognitive functions and mental distress with age.

Effect of an Active Lifestyle

It is well known that our ancestors led a very active lifestyle. Therefore, physical activity may have contributed to the evolution of the brain [86]. Physical exercise (PE) promotes constant cognitive and motor functions which lead to increased neuroplasticity in the human brain. The positive effects of PE are described to be mediated via Brain-Derived Neurotrophic Factor (BDNF). BDNF is a growth factor that reinforces synaptogenesis and neurogenesis which preserve existing structures and promote brain volume, respectively. Therefore, regular PE may avert some of the changes in hippocampal synaptic plasticity and may promote hippocampal volume preservation [87]. Similarly, BDNF controls emotional learning in the amygdala and enhances neurotransmission [88]. Therefore, absence of exercise in the contemporary societies contributes to the swift decline in hippocampal volume and to dysregulation of amygdala functionality [89]. Additionally, WD is deficient in essential nutrients and antioxidants needed to fight oxidative stress (OS). OS in the brain, which typically increases with age and reduces levels of BDNF; accordingly, it adds to the absence of neuroplasticity [90].

Conclusion

Based on the collective evidence, it appears that there is a mismatch between the evolved brain and the modern diet and lifestyle. The evidence suggests that nutrient-dense diets and an active lifestyle led to the evolution of the human brain and to the development of complex neural circuitry that control cognitive functions and emotions. In return, this complex network requires ample of high-quality dietary components that contribute to the growth, maintenance and repair of brain structures. Additionally, it appears that the sexual dimorphic state of brain connectivity may be adding to the differential dietary needs between male and female brain. Although, it is commonly known that eating healthy and exercising are warranted for a healthy body and mind, this review provides compelling evidence that describes the disturbances that ensue in the human brain from a poor diet and lack of physical activity. These disturbances, as described in the literature, are linked to cognitive decline and mental distress in contemporary humans.

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