ARC Journal of Nutrition and Growth
Volume 10, Issue 1, 2024, PP 23-25
ISSN No. (Online) 2455-2550
DOI: https://doi.org/10.20431/2455-2550.1001004
www.arcjournals.org

The Impact of Western Diet on Traumatic Brain Injury Outcomes

Betul Aslan*

Maltepe University, Faculty of Medicine, Anatomy Department, Turkey

Corresponding Author:** ***Betul Aslan Frances, *Maltepe University, Faculty of Medicine, Anatomy Department, Turkey.*

Abstract: *The relationship between Western diet (WD) and traumatic brain injury (TBI) underscores the critical role of nutrition in brain health. WD, characterized by high saturated fats, processed sugars, and low fiber content, can adversely affect long-term neurological outcomes following TBI. Effects such as cognitive impairments and synaptic damage highlight the lasting impact of WD on brain health. Therefore, this study aims to examine the relationship between WD and TBI.*

Keywords: *WD, TBI, cognitive functions*

1. INTRODUCTION

Dietary factors are increasingly recognized as potent regulators of brain plasticity, capable of influencing the progressiin of brain disorders [1].

In recent years, one of the lifestyle changes observed in Westernized societies is the increasing consumption of WD, which are deficient in fibers, vitamins, and minerals but rich in processed foods, fast food, convenience products, snacks, and sugary beverages. This dietary trend has spread increasingly from high-income countries to low-income countries. Concurrently, there has been a rise in diseases associated with WD, such as obesity, type 2 diabetes cardiovascular diseases, and neurodegenerative and autoimmune diseases [2, 3]. WD is highly heterogeneous in terms of its content and quality of fats carbohydrates, and proteins, representing calorie-dense foods. Given its significant role in regulating energy metabolism, WD is recognized as a significant risk factor in pathophysiology [4]. Prolonged adherence to a WD can disrupt normal physiology and impact health by promoting weight gain, pathological changes in lipids and energy metabolism, and activation of the immune system. Thus, a compromised immune-metabolic system associated with WD can contribute to a range of chronic metabolic diseases, including obesity, cardiovascular diseases, neurodegenerative and autoimmune diseases [5].

Evidence suggests that systemic changes associated with WD can lead to neuroinflammation parallel to disruption of the blood-brain barrier. Subsequently, these changes may impair synaptic transmission, lead to neurodegeneration, and ultimately result in memory and cognitive impairment [6].

The brain requires a significant amount of energy and can use either glucose or ketone bodies depending on metabolic availability. Thus, regulating metabolic rates is crucial for the central nervous system. Interestingly, metabolic dysfunction directly increases the risk of behavioral, cognitive, and mood disorders [7]. Additionally, obesity is associated with a higher risk of neurodegenerative diseases like Alzheimer's disease. Mechanistically, consuming a WD and obesity lead to systemic inflammatory responses, which can contribute to cognitive decline and worsen outcomes in brain injuries [8].

TBI is a critical and progressive brain injury caused by a mechanical impact, resulting in neurobehavioral dysfunction [9]. The pathophysiological processes in TBI are categorized into primary and secondary brain injuries. Primary injury refers to the immediate mechanical damage at the injury site, including tissue damage, disruption of cerebral blood flow regulation, subarachnoid hemorrhage, epidural hematoma, subdural hematoma, and

contusion. Secondary injury, on the other hand, involves a cascade of subsequent cellular and molecular events triggered by the primary insult [10]. These secondary mechanisms include diffuse axonal injury, inflammation, ischemia, and energy depletion [11].

WD has been implicated in influencing the outcomes and severity of TBI through several mechanisms.

- **Inflammation**

WD is known to induce systemic inflammation due to its high content of saturated fats, sugars, and processed foods. In the context of TBI, this inflammatory response can exacerbate secondary brain injury by amplifying neuroinflammation, oxidative stress, and immune responses [12].

- **Metabolic Dysfunction**

WD contributes to metabolic dysfunction, including insulin resistance and dyslipidemia, which are also risk factors for poor outcomes in TBI. These metabolic disturbances can impair cellular repair mechanisms and worsen neuronal damage post-injury [13].

- **Gut-Brain Axis**

WD affects the gut microbiota composition and intestinal barrier integrity. Disruption of the gut microbiota (dysbiosis) and increased intestinal permeability can lead to systemic inflammation and exacerbate neuroinflammation in TBI [14].

- **Brain Health**

Long-term consumption of WD is associated with cognitive decline and neurodegenerative diseases. When combined with TBI, WD may accelerate neurodegenerative processes and hinder recovery by promoting neuronal dysfunction and synaptic impairment [15].

Overall, the relationship between WD and TBI underscores the importance of nutrition in brain injury outcomes. A diet rich in whole foods, antioxidants, and healthy fats may offer neuroprotective benefits and improve recovery after TBI.

2. Discussion

Most studies examining how changes in diet affect traumatic brain injury (TBI) have primarily focused on understanding their impact on neuropathological and behavioral outcomes, regardless of the specific modes or severity of the brain injury context.

In a study, the secondary injury outcomes following a closed head injury with a single hit TBI were assessed in obese mice fed with WD, compared to lean mice. At a chronic time point (30 days), the obese mice exhibited significant microglial activation and a chronic inflammatory condition, which researchers attributed to disruption of the hypothalamic-pituitary-adrenal axis [16]. Likewise, feeding mice a high-fat and high-sucrose diet for four weeks prior to inducing experimental mild diffuse brain injury exacerbated the impairment of spatial learning capacity caused by the TBI [17].

Another study suggest that the additive effect of high fat diet (HFD) and TBI worsens short term memory and sensation deficits, and may be driven by enhanced oxidative stress and inflammation [18]. Adherence to a HFD has been found to exacerbate neurocognitive impairments triggered by TBI and worsen reductions in hippocampal mRNA and BDNF protein expression following TBI [19].

Consumption of a HFD could also drive secondary injury mechanisms such as chronic neuroinflammation. In obese rats subjected to experimental repetitive TBI, neuroinflammation characterized by microglial activation and chronic insulin resistance has been observed to coincide with increased anxiety levels [20].

There is also substantial evidence supporting the neuroprotective effectiveness of various vitamins, minerals, and other compounds derived from animal models of TBI [19].

3. Conclusion

It is well-established that consumption of HFDs such as WD, linked to neurodegenerative diseases, can negatively impact long-term neurological outcomes following TBI. WD can exacerbate post-injury outcomes by impacting molecular pathways similar to those affected by TBI, thereby increasing neuroinflammation. A healthy diet enriched with beneficial components like antioxidants and healthy fats can reduce the risk of complications after TBI and promote recovery. Finally, investigating broader dietary patterns and long-term intake of neuroprotective and neurotoxic compounds is essential for understanding their influence on post-TBI outcomes, particularly among older adults who are often affected by nutritional deficiencies.

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Citation: *Betul Aslan. The Impact of Western Diet on Traumatic Brain Injury Outcomes. ARC Journal of Nutrition and Growth. 2024; 10(1):23-25. DOI: https://doi.org/ 10.20431/2455-2550.101004.*