Metabolic Syndrome's Effect on the Development of Neurodegenerative Diseases

Keith Herbert*

Editorial office, Journal of Neuroscience and Neuropharmacology, Brussels, Belgium

*Corresponding Author**

Keith Herbert
Department of Neuropharmacology
Belgium
Email: Herbert.k@gmail.com

Received: 5-Aug-2022, Manuscript No. NCOA-22-73502; **Editor assigned:** 8-Aug-2022, PreQC No. NCOA-22-73502 (PQ); **Reviewed**: 16-Aug-2022, QC No. NCOA-22-73502 (Q); **Revised**: 19-Aug-2022, Manuscript No. NCOA-22-73502 (R); **Published**: 27-Aug-2022, doi. 10.4172/2469-9780.2022.8.4.182

Abstract

Metabolic Syndrome (MetS) and Neurodegenerative Diseases (NDD), such as Alzheimer's disease, Huntington's disease, Parkinson's disease, and depression, are both impacted by the Brain-Derived Neurotrophic Factor (BDNF). If one element is crucial to the pathogenesis of two diseases, then it is likely that these two diseases share the same underlying cause. This study intended to emphasize the significant roles of BDNF in the pathogenesis of MetS and NDD and to propose two distinct preventative or therapeutic approaches, BDNF gene therapy, and BDFN administration, for managing MetS and NDD. One might list cardiometabolic disorders including atherosclerosis, hypertension, Type 2 Diabetes Mellitus (T2DM), obesity, diabetes, and Metabolic Syndrome (MetS) as some of the most prevalent worldwide pathologies. Based on the justification that obesity is one of the main causes of T2DM and that T2DM is linked to obesity, the term "diabesity" was added to this collection. One of the biggest clinical difficulties and worldwide health problems is the MetS, according to experts. It has etiologies connected to modernism, changing eating patterns, excess calorie intake, sedentary lifestyles, and decreased physical exercise. Patients with this group of metabolic disorders are more prone than healthy individuals to developing conditions like Cardiovascular Diseases (CVDs), coronary heart disease, Myocardial Infarction (MI), micro- and macrovascular dysfunction, type 2 diabetes, cognitive impairment, Central Nervous System (CNS) dysfunction, and early death.

Introduction

Metabolic Syndrome (MetS) is the concomitant accumulation of a number of functional abnormalities that often take place in persons over the age of 60. Type-2 Diabetes Mellitus (T2DM), dyslipidemia, and arterial hypertension are four conditions that must be present for MetS to be diagnosed. These conditions raise the risk of cardiovascular diseases as well as neurological sequelae including stroke and dementia. The insensitivity to hormones like leptin, adiponectin, and insulin results from these functional alterations, which often occur simultaneously. In addition to being linked to multiple diseases and diseases in the target organs, insulin resistance is a key factor in the emergence of metabolic dysfunctions, including T2DM. Therefore, a sedentary lifestyle, inherited tendencies, alcoholism, poor eating habits, as well as other environmental variables, raise the risk of obesity, encouraging the development of MetS. The production of pro-inflammatory mediators or adipokines is increased by hyperplasia of adipose tissue, which also increases the development of pre-adipocytes into adipocytes (adipogenesis). The Nuclear Factor Kappa B (NF-B), a potent transcription factor that is involved in the expression of several related genes and the inflammatory response in adipose tissue and the liver, is activated by the excess of free fatty acids in the blood plasma from adipocytes or the high intestinal inflow of fat from the diet. Therefore, it leads to an increase in the expression of pro-inflammatory cytokines like tumor necrosis factor-, Interleukin (IL)-1 and IL6, chemokines, prostaglandins, and adhesion molecules that act on particular targets and result in macrophage infiltration, promoting systemic inflammation and insulin resistance.

Central inflammation is brought on by circulating pro-inflammatory cytokines that are elevated due to systemic inflammation brought on by a high-fat diet. These circulating pro-inflammatory cytokines go through the blood-brain barrier and into the brain and hypothalamus, activating the NF-B in glial cells and the hypothalamus as a consequence. This causes inflammation in the hypothalamus and leptin resistance. MetS can be caused by risk factors for depressive symptoms, white matter lesions in the brain, and aging-related cognitive impairment. Alarmingly, dementia in older persons is on the rise globally, and those with MetS may be more vulnerable to this disease. Cardiovascular illness is another concern for those with dementia. Therefore, it becomes important to comprehend how the diseases that make up the MetS condition interact with one another and how this results in the progression of neurodegenerative disorders, as well as potential mitigation measures for this painful process.

Along with dementia, clinical consequences from metabolic diseases like T2DM, such as depression and strokes, are known to occur. Microvascular damage, which directly affects the brain and promotes mental diseases, must be taken into consideration even though the process of neurological diseases connected to T2DM is complicated and multiple. Additionally, arterial hypertension exacerbates diseases in target organs by increasing arterial stiffness, which boosts blood flow. However, the microvasculature is impacted by arterial stiffness, and brain diseases are therefore directly influenced. As a result, among other harmful disorders brought on by pressure overload, the long-term effects of AH might result in encephalopathies because of global cerebral hypoperfusion, cerebral infarction with microinfarctions, and other pressure-related conditions. The person with MetS, however, has a well-documented inflammation and is affected by all of the clinical disorders listed above in an integrated manner. Recent research indicates that chronic neuroinflammation and insulin signaling abnormalities are both risk factors for brain diseases including dementia and other brain disorders. The anti-inflammatory cytokine adiponectin, which is generated by adipocytes and can increase insulin sensitivity, is relevant in this setting. Adiponectin acts as a crucial regulator of brain physiology in the brain, maintaining and enhancing cognitive processes. Therefore, methods that lower a MetS patient's inflammatory profile seem effective in managing their metabolism. One of the successful techniques that are linked to a reduction in all risk variables, including the risk for T2DM, is weight loss. However, alterations in eating behaviours, such as fewer calories consumed and more physical activity, which raises energy expenditure, are preferable to weight loss. Other approaches, such using medications to regulate obesity or lower body weight, don't seem to be as effective over the long run. Surprisingly, regular exercise provides several advantages for people with MetS, including better lipid profiles, lower blood pressure, body weight control, and glycemic control. Exercise is also essential for preventing and slowing the development of neuronal disorders like Alzheimer's disease. As a result, regular exercise training increases the process of neurogenesis and synaptogenesis because it increases blood flow, enhances insulin sensitivity, lowers the inflammatory profile, and raises concentrations of hormones and second messengers such as adiponectin, brain-derived neurotrophic factor, and insulin-like growth factor. Though working in a complimentary manner in accordance with the clinical picture, exercise cannot replace the prescription of medications to treat diseases connected to MetS despite the positive effects. The use of peptides is particularly prominent in the treatment of oncological and metabolic diseases. The first stands out owing to the T2DM and obesity pandemic expansion, and the latter because of the rise in mortality and the need for novel chemotherapeutic treatment choices. In the pharmaceutical sector, isolated medications, particularly peptides, are being used to treat uncommon disorders. Captopril, one of the best-selling medications in the world and a successful treatment for hypertension, is a classic example of 1a medication created based on the observation of the triggering of the systemic impact brought on by pairing. An oligopeptide -10- (pyroglutamyl), rich in proline and obtained from the venom of the Bothrops jararaca snake, serves as the active component in this medication.

Cite this article: Herbert K, Metabolic Syndrome's Effect on the Development of Neurodegenerative Diseases .J Neurosci Neuropharmacol. 2022, 8.4, 001.