The Interplay between Metabolism, Cognition, and the Brain Throughout an Individual's Life Span

Brady James* and Shagufta Hassan
Department of Neurology, University of Michigan, Michigan, US

Corresponding Author*
Brady James
Division of Neurology
University of Michigan,
Michigan, US
Email: brady232h@yahoo.com

Received date: 03-May-2023, Manuscript No: JMSO-23-102675; **Editor assigned:** 6-May-2023, PreQC No. JMSO-23-102675(PQ); **Reviewed:** 24-May-2023, QC No. JMSO-23-102675(Q); **Revised date**: 26-May-2023, Manuscript No: JMSO-23- 102675 (R); **Published date:** 30-May-2023, DOI: 10.35248/2376-0389.23.10.5.499

Abstract

The articles in this particular issue provide a comprehensive and informative analysis of the intricate connections between brain function and metabolic health. The changes in metabolism and components associated with metabolic syndrome play a crucial and fascinating role in the development and prognosis of neurodegeneration and cognition. This includes the inheritance of epigenetic traits across generations, abnormalities in brain development during youth, and disorders that affect adult brain health. Furthermore, lifestyle modifications and other treatment options hold significant potential in preventing neurological consequences resulting from conditions like diabetes, obesity, stress, and other factors that impact metabolic health. While some of these concepts are still in their early stages, the wealth of knowledge obtained from recent and ongoing research is highly promising. We consider this subject matter to be enlightening and thought-provoking, and we eagerly anticipate further advancements in our understanding of the relationship between metabolism, cognition, and the brain throughout the lifespan.

Keywords: Metabolic syndrome • Neurodegeneration • Nervous system disorder • Autism spectrum disorder

Introduction

The last ten years have seen a rise in curiosity on how changes in metabolic function affect illness risk, especially nervous system disorders. The condition known as Metabolic Syndrome (MetS), which is a confluence of illnesses including obesity, hypertension, dyslipidemia, and impaired glucose tolerance, is brought on by a general failure of metabolism and underlies a five-fold greater risk for diabetes and cardiovascular disease. According to the IDF, 25% of people globally currently have MetS. Additionally, more than 400 million people globally have diabetes, and more than two thirds of individuals in developed nations, including the USA, are overweight or obese.

Multiple studies have found that people with MetS, from young children to elderly adults, have a greater chance of developing neurodevelopmental, neurodegenerative, and cognitive impairments, which adds to these worrying numbers. In some circumstances, particularly in young people, this is probably the result of maternal metabolic disorders, which are linked to a wide range of neurodevelopmental issues in offspring, including Autism Spectrum Disorder (ASD) and neurodevelopmental delay. Similar increases in prevalence are seen in patients with the MetS or frank diabetes for a number of adult neurodegenerative diseases, including Alzheimer's disease.

It's significant to note that AD, the primary cause of dementia, now affects over 50 million people globally, and by 2030, it's predicted that number will increase to over 100 million. When compared to controls of the same age and gender, individuals with diabetes have a 50%–75% higher chance of acquiring AD than do those with MetS. Despite these correlations, however, the underlying mechanisms are unknown, despite encouraging studies indicating that dietary changes, physical activity, and good sleep habits can all help treat a variety of neurological problems disorders in youth to chronic progressive neurodegenerative disorders in adults. These topics are covered in a collection of informative reviews coupled with primary research articles that can be divided into four main sections:

1. Neurodevelopmental implications of metabolism.
2. Effects of diabetes and obesity on cognition.
3. Lifestyle and metabolic interventions.
4. Primary research articles.

Together, these articles address aspects of the relationship between MetS and neurological disorders throughout life and offer a timely overview of the recent research advances, current clinical implications, and potential treatment strategies.

Two reviews on the effects of metabolism on neurodevelopmental processes open the issue. The first paper by Dr. Machmohan and Daisy explores the function of epigenetic changes in the developing and adult brain, as well as the possibility of these changes being passed down through the generations. With special focus on the effects epigenetic patterns have on metabolic health and on both normal functioning and disease states of the central nervous system, they discuss how epigenetic changes driven by exposures, experiences, stress, and diet can drive the metabolic phenotypes of individuals and their progeny. The review article that follows, written by Dr. Susie, examines the relationship between adolescent brain development and metabolism with a focus on the pathophysiology and prognosis of Autism Spectrum Disorder (ASD). On the basis of the most recent research, therapeutic considerations are offered, including addressing nutritional requirements together with dietary changes and oral supplements. These two articles together highlight recent developments that shed light on the significant effects of metabolic disorders on inheritance and childhood.

The second section of this issue's papers then focuses on the effects that diabetes and obesity have on cognition and the functioning of the central nervous system. Dr. Shagufta and Nikita first go over the existing clinical evidence from long-term studies that compared how type 1 and type 2 diabetes affected risk for cognitive problems differently.

They ask for more research into the precise pathways that cause cognitive dysfunction, especially for type 2 diabetes and its accompanying

comorbidities, and they explicitly emphasize the implications of the elevated risks for cognitive decline. Obesity is one of these coexisting factors and is the subject of the second review in this part by Dr. Martin and colleagues. In this review, the authors present evidence that suggests that obesity contributes to cognitive impairment and eloquently outline how obesity-related metabolic effects on the gut, immune system, and insulin signaling result in endothelial dysfunction and disruption of the blood-brain barrier. They also discuss the effects of existing therapy approaches for obesity on a person's cognitive performance [1, 2].

The issue's third portion goes into more detail about the possible impact of well-known and newly developed metabolic therapies on cognitive performance. Beginning with the function of the gut-brain axis in health and neurodegeneration, Dr. Hanna and colleagues highlight how the gut microbiota constitute a dynamic system that may be impacted by exercise, nutrition, and stress-induced metabolic changes. As a result, treatment strategies that concentrate on the gut microbiota provide promising opportunities to maybe avoid the harm to the central nervous system brought on by metabolic disorders. Then, Dr. Glory and colleagues give an insightful overview of how ageing and lifestyle choices can affect dementia risk.

They notably concentrate on treatments that enhance metabolic health and show how diet, exercise, and medications that have been shown to be effective against aging-related disorders may also be helpful for long-term brain health. These two studies emphasise the significance of preserving metabolic health to ensure ideal brain function all throughout life [3-5].

This issue's last section contains three key research articles that advance our understanding of the processes and cutting-edge therapeutic strategies that can be used to lessen cognitive loss brought on by metabolic disease. The efficiency of insulin and insulin-like growth factor I therapies, which are presently being assessed in clinical trials for Alzheimer's disease, can be affected by insulin resistance, according to a first study by Dr. Chargos, and colleagues. They identify the mechanisms underlying the diminished efficacy of these treatments in the context of insulin resistance using in vitro and in vivo models. The results imply that medications that affect neurodegeneration may be less beneficial for people with MetS. Next, Dr. Mikel and associates examine the effects of a high-fat diet on mice's midlife cognitive function. They were able to demonstrate that cerebral blood flow and visceral obesity may be indicators of cognitive deterioration by a detailed assessment of metabolic and cognitive phenotypes. The effects of pioglitazone, a diabetes medication that modifies metabolic activity, on cognitive performance in rats with traumatic brain damage are also investigated by Dr. Jenny and associates. Their results imply that treatments that target brain metabolism may be advantageous for enhancing cognitive function in people with chronic traumatic brain injury.

Together, the articles in this issue offer an in-depth, instructive review of the complex interactions between brain function and metabolic health. Metabolic alterations and the MetS components have a crucial and intriguing role in the pathogenesis and prognosis of neurodegeneration and cognition, including epigenetic inheritance across generations, neurodevelopmental abnormalities in youth, and disorders impacting adult brain health. Additionally, treatment alternatives such as lifestyle changes have a great potential to prevent neurologic consequences brought on by conditions like diabetes, obesity, stress, and other conditions that affect metabolic health. Even though some of these ideas are still in their infancy, the abundance of knowledge gleaned from recent and current investigations is exciting. We find this topic to be illuminating and stimulating, and we look forward to following the developments made in comprehending the relationship between metabolism, cognition, and the brain throughout life.

References

1. Brockschnieder, D., et al. "Cell depletion due to diphtheria toxin fragment A after Cre-mediated recombination." *Mol. cell. biol.* 24.17 (2004): 7636-7642.
2. Feil, Robert. "Environmental and nutritional effects on the epigenetic regulation of genes." *Mutat. Res./Fundam. Mol. Mech. Mutagen.* 600.1-2 (2006): 46-57.
3. Holliday R. "A historical overview."*Epigenetics* . 2006;1:76—80.
4. Barbara, K., et al. "Multiple sclerosis and chronic autoimmune encephalomyelitis: a comparative quantitative study of axonal injury in active, inactive, and remyelinated lesions." Am. j. pathol. 157.1 (2000): 267-276.
5. Michailov, Galin, V., et al. "Axonal neuregulin-1 regulates myelin sheath thickness." *Science* 304.5671 (2004): 700-703.

Cite this article: James, B. and Hassan, S. The Interplay between Metabolism, Cognition, and the Brain Throughout an Individual's life Span. J Mult Scler 2023, 10(5), 499