

Opinion Article

Impact of Metabolic Syndrome on the Development of Neurodegenerative Disorders

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DESCRIPTION

The Brain-Derived Neurotropic Factor affects both Metabolic Syndrome (MetS) and Neurodegenerative Disorders (NDD) such as Alzheimer's disease, Huntington's disease, Parkinson's disease and depression. If one ingredient is critical to the development of two diseases, it is likely that the two diseases have the same underlying etiology.

Metabolic Syndrome (MetS) is the concurrent accumulation of a number of functional impairments that commonly occur in people over the age of 60. Type 2 Diabetes Mellitus (T2DM) and arterial hypertension are the four criteria that must be present for MetS to be diagnosed. These disorders increase the risk of cardiovascular disease as well as neurological consequences such as strokes and alzheimer. Hyperplasia of adipose tissue enhances pro-inflammatory mediators synthesis of simultaneously promoting the growth of pre-adipocytes into adipocytes. The Nuclear Factor Kappa B (NF-B), a potent transcription factor involved in the expression of several related genes as well as the inflammatory response in adipose tissue and the liver. As a result, it causes an increase in the expression of pro-inflammatory cytokines such as tumour necrosis factor-Interleukin IL-1 and IL-6, chemokines, prostaglandins and adhesion molecules which act on specific targets and result in macrophage infiltration, promoting systemic inflammation and insulin resistance.

Central inflammation is caused by circulating pro-inflammatory cytokines, which are raised as a result of systemic inflammation caused by a high-fat diet. These circulating pro-inflammatory cytokines cross the blood-brain barrier and enter the brain and hypothalamus, where they activate NF-B in glial cells and the hypothalamus. This results in hypothalamic inflammation and

leptin resistance. MetS can be caused by variables such as a family history of depression, white matter lesions in the brain and aging-related cognitive decline. Dementia in elderly people is on the rise worldwide and those with MetS may be especially sensitive to it. Clinical implications of metabolic illnesses like T2DM, such as depression and strokes, are known to occur in addition to dementia. Even if the process of neurological disorders associated with T2DM is complicated and multifaceted, micro vascular damage, which directly affects the brain and increases mental diseases, must be considered. Furthermore, arterial hypertension worsens disorders in target organs by increasing arterial stiffness, which increases blood flow. Nevertheless, arterial stiffness has an effect on the microvasculature and brain illnesses are therefore directly affected. The person with MetS, on the other hand has welldocumented inflammation and is affected by all of the clinical disorders listed above in a coordinated fashion, indicating that chronic neuroinflammation and insulin signaling abnormalities are both risk factors for brain diseases such as dementia and other brain disorders.

The anti-inflammatory cytokine adiponectin, which is produced by adipocytes and can promote insulin sensitivity, is important. Adiponectin is a critical regulator of brain physiology in the brain, helping to maintain and improve cognitive processes. Weight loss is one of the successful approaches connected to a reduction in all risk variables, including the risk of T2DM. Changes in eating habits, such as consuming less calories and increasing physical activity, which increase energy expenditure are preferred to weight reduction. Other treatments, such as utilizing drugs to control obesity or lose weight do not appear to be as beneficial in the long run.

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