Treatment for Bipolar Disorder with Ketogenic and Low Carbohydrate Diet

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Description

Bipolar Disorder (BD) is a severe, multifaceted neurodevelopmental condition that impairs functional capacity and is characterized by abrupt, severe biphasic fluctuations in mood, energy, and thinking. About 9.2 million (2.8%) adults in the United States are affected by this condition each year, making it the 17th greatest cause of disease burden globally and the affective disorder with the highest suicide incidence. Grandiosity, increased activity levels, a decreased need for sleep, and more intense emotional responses are all possible symptoms of a manic bipolar disorder. A depressed episode can be recognized by changes in appetite, a sense of worthlessness, exhaustion, and recurrent suicide thoughts. Continuous treatment for BD is frequently required because of its cyclical and recurrent character and entails a mix of medication and psychotherapy. Mood stabilizers and antipsychotics, which predominantly target sodium channels, dopamine D2 and serotonergic 5-HT2 receptor targets, are among the pharmaceuticals used to treat symptoms. Inadvertent unfavorable metabolic side effects, such as weight gain or other neurological or cardiovascular problems, can happen when these fundamental mechanistic objectives are addressed.

Numerous biological processes have been suggested as possible underlying factors in BD. These include neurotransmitter disturbance, oxidative stress, and mitochondrial malfunction. In recent years, an increasing number of genetic, biochemical, and neuroimaging research have started to tackle these theories. Energy metabolism, cellular signaling, and circadian rhythms are some of the main activities that have been proven to be altered by biological mechanisms that are malfunctioning. A possible prior to BD has been found as mitochondrial malfunction. Reactive Oxidative Species (ROS) protection, modulation of intracellular calcium (Ca^{2+}), synaptic plasticity, and energy production are all crucial functions of mitochondria. Changes in aspartate, glutamate, choline, myo-inositol, lactate, phosphocreatine, phosphomonoesters, and intracellular pH, which alter cell signaling, are signs of aberrant metabolism in the brain, according to magnetic resonance studies. Additional biochemical and genetic evidence supports the idea that mitochondrial malfunction has a role in BD. The amount of energy used by brain functions can be affected by mitochondrial malfunction. Energy output may be impacted if Ca^{2+} concentrations cannot be controlled. Ca^{2+} levels increase and ATP (Adenosine Triphosphate) levels decrease when the Na^{+}/K^{+-} ATP is dysfunctional. The duration of neurotransmitter release can fluctuate in response to slight drops in ATP levels, which can also send neurons into excitatory or refractory states. Therefore, a shift in the threshold required for neuronal activity may have an impact on the manic and depressive phases of BD. It is likely that a decrease in Na^{+}/K^{+-}ATPase function results in the impairment of oxidative phosphorylation since normal ATP levels can only be achieved by both oxidative phosphorylation and glycolysis. But as of now, it is unclear how oxidative phosphorylation can malfunction. Since the majority of ATP is produced during the last phase of cellular respiration, a loss of function in key proteins might potentially result in a substantial drop in ATP levels.

The brain's ability to sustain energy levels could change. It has been proposed that BD may be caused by disruption of the ATP synthesis pathway between glycolysis, the Tricarboxylic Acid (TCA) cycle, and oxidative phosphorylation. At the conclusion of glycolysis, pyruvate is created and transformed into acetyl-CoA for usage in the TCA cycle. The process of conversion is carried out by the Pyruvate Dehydrogenase Complex (PDC). Pyruvate concentrations have been found to be elevated in BD, which suggests that the PDC is malfunctioning. Another biomarker that has been linked to BD is lactate, a consequence of glycolysis. Damage to the enzyme could cause oxidative phosphorylation to stop and glycolysis to become the main source of energy. Since glycolysis alone cannot produce sufficient quantities of ATP, PDC failure is expected to have negative knock-on consequences on the neuronal activity that regulates mood states in BD. A low-carbohydrate, high-fat, or KD diet has tentatively been demonstrated to positively affect mood, behavior, and cognition; as such, it represents a promising non-pharmacological treatment for BD that calls for more investigation.

A KD may have therapeutic effects on psychiatric symptoms in addition to metabolic health, according to supporting evidence from animal research and early results from human models. However, due to the dearth of open label and randomised controlled trials up to this point, the effectiveness of KD in patients with BD remains uncertain. To better understand how a KD works and how ketosis affects energy production, neural signaling pathways, and cellular defense, more clinical study is required.

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