Editorial

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Editorial Note on Alzheimer Disease

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Editorial Note

Alzheimer's disease is a progressive neurological illness marked by a loss of social adaptability due to personal and environmental confusion, and finally death of the patient. The death rate for Alzheimer's disease is 37.3 per 100,000 people. However, the global morbidity of this disease has increased considerably in the previous 20 years, with an average increase of 55.47%. There appears to be a link between educational attainment and the occurrence of dementia illness.

Because the brain is one of the most energy-intensive organs, we may conclude that this trend of low education in the background of individuals with Alzheimer's disease is due to a medical issue. Thus, there is a strong case to be made that this condition is a sort of metabolic disorder caused by persistent subclinical hypoglycemia, which results in insufficient production of ATP and other high-energy molecules by neurons and glia. As a result, the release of acetylcholine exceeds the receptors capacity, and they prevent depolarization, resulting in receptor desensitization to acetylcholine.

Hypoglycemia causes the release of certain glucose-related hormones (such as serotonin) to be reduced indirectly, and it can also steal the pentose phosphate pathway by causing the production of lactate in neurons. The lack of excitability of neurons produces a drop in metabolism in the surrounding tissue, which, in turn, leads to the death of astrocytes, which then leads to the death of neurons. Furthermore, Tryptophan catabolism culminates in the synthesis of kynurenine and its subsequent breakdown products, which predominantly increase ATP and glycolysis while also influencing serotonin release and may be considered a compensatory mechanism. Furthermore, excessive Tryptophan breakdown is thought to produce irreversible alterations in the function of NMDA receptors, as well as direct neurodegenerative effects in the brain. Furthermore, when this process occurs via nAChreceptors in the presence of Mg^{2+} and hypopolarization, it can cause the Ca^{2+} current to be blocked, resulting in exocytosis. Knowing that brains afflicted by this illness frequently contain Li^{+} ions emphasizes the importance of Mg^{2+} in the inhibition of neurotransmissions, as Li has comparable characteristics to Mg^{2+}.

According to this theory, the affected brains contain elements such as Li, Al, and others, which may be the result of pyrrole catabolism, which in some conditions can exhibit the main properties, while -amyloid plaque and tau-tangles are the result of a breakdown of the bilipid layer of the membrane of the victim neurons and the remains of glia. We can understand why the diffuse distribution of plaques and tangles reflects the most severe brain damage in this situation since it shows the death of mainly interneurons. But arguably the most pressing concern is why, in the previous 20 years, the global morbidity rate for this disease has grown to 55.47%. Without further, broad use of in-depth methods of evaluation, none of the non-communicable illnesses may show such considerable development.

This is thought to be related to people's insatiable need to reduce weight using a variety of weight-loss medications, an imbalance of proteins and carbs in their daily diet, and the fact that only around 5% of Alzheimer's disease is caused by an original proteinopathy (family Alzheimer's disease). That logically opposes the Japanese notion of "excessive" protein in the daily diet as a cause of dementia, and that the first proteinopathy is the issue of overwhelming numbers of dementia family illnesses.

As a result, this is the inverse of diabetes mellitus, where mortality rises proportionately with an increase in carbohydrate consumption in a daily diet from 49% to 74%, regardless of overall calorie intake, from 52 to 380 per 100000, p-value=0.001. The major cause of this alarming rise in dementia is widespread dieting and the use of weight-loss medicines that affect appetite and calorie absorption processes. Initial proteinopathy is uncommon in dementia familial illness, accounting for fewer than 5% of cases. Chronic hypoglycemia is the primary cause of neurodegeneration, as it inhibits glycolysis in glia and prevents neurons from depolarizing.

The production of kynurenine is potentiated by a lack of excitability, as is the excessive breakdown of Tryptophan. The nonreversible alterations in the activities of NMDA receptors caused by kynurenine and its breakdown products produce direct and indirect neurodegenerative changes in the brain. -amyloid plaque and tau-tangles are thought to be the result of a breakdown of the bilipid layer of the membranes of the brain's fundamental structures, most likely due to excessive pyrrole degradation.

