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Original Article

Role of Vitamin B9 (Folate) In Neuronal Plasticity

Sewanu Stpehen Godonu*

All American Institute of Medical Sciences

ABSTRACT

Folate as widely known is one of the B vitamins(vitamin B9). All B vitamins are water soluble vitamins(the body those not store them) that help the body conversion of of food into fuels(e.g,Carbohydrate to glucose) which is used to produce energy. These group of vitamins are the B complex vitamin. Folic acid a synthetic form of vitamin B9, found in supplements and fortified foods and folate which occurs naturally in foods is crucial for the proper brain functions and neurological health. Vitamin B9 have been a crucial nutrient during early pregnancy to reduce the risk of birth defects of the brain and spine. It involvement in DNA synthesis and RNA, the body genetic material has made it a critical vitamin in cellular health also a role in cellular detoxification. In recent research studies role of vitamin B9 in neuronal plasticity (commonly referred to ability of the CNS(Central Nervous System) to change it connectivity in terms of learning and memory,damage and development) as proofed to be an essential nutritional factor in playing a multifaceted roles in the brain health. Deficiency of folate (vitamin B9) have been associated with cognitive defects, depression and neurodegenerative diseases etc edifying the importance of folate in neuronal integrity. Biosynthesis of folate and importance, evidence, and impact of folate in neuronal plasticity will be reviewed in this systematic review with information accessed from databases such as Mount Sinai.org,CNBC make it(cnbc.com),Mayo clinic.org,CDC.gov,Havard T.H.CHAN school of public health and recent research studies and articles from 2003-present.

Keywords:CNS(Central Nervous System), Neurodegenerative, Neurotoxicity, Neuropsychiatry.

INTRODUCTION

Folic acid plays an important role in neuroplasticity and in the maintenance of neuronal integrity. Folate is a co-factor in one-carbon metabolism during which it promotes the regeneration ,of methionine from homocysteine, a highly reactive sulfur-containing amino acid. Methionine may then be converted to S-adenosylmethionine (SAM), the principal methyl donor in most biosynthetic methylation reactions. On the cellular level, folate deficiency and hyper homocysteinemia exert multiple detrimental effects(Golo Kronenberg, Micheal Colla and Mattias Endres, 2009).Low folate status and elevated homocysteine increase the generation of reactive oxygen species and contribute to excitotoxicity and mitochondrial dysfunction which may lead to apoptosis. Strong epidemiological and experimental evidence links derangements of one-carbon metabolism to vascular, neurodegenerative and neuropsychiatric disease, including most prominently cerebral ischemia, Alzheimer's dementia and depression(Golo Kronenberg, Micheal Colla and Mattias Endres, 2009).

Biosynthesis Of Vitamin B9 (Folate) And Importance

Folates exist in many forms. The main circulating form is 5-methyltetrahydrofolate. The natural forms, in plant foods, are prone to oxidation. Folic acid is the stable synthetic form. Folate works as a methyl donor for cellular methylation and protein synthesis, particularly for hematopoiesis. It is also directly involved in DNA and RNA synthesis, and requirements increase during embryonic development. Deficiency states include dietary insufficiency and also altered folate metabolism, which may be genetic(Shandshand, AG;Lean, MEJ et al; Davidson's principles and practice of medicine, 22, 755-780,2023).The human body cannot synthesize folic acid. Folic acid is a vitamin that gives rise to tetrahydro folic acid, which can carry a one-carbon group, such as a methyl group. Folic acid and its derivatives are found primarily in legumes, green leafy vegetables, and grains. The intestine releases

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mostly N 5 -methyl-tetrahydro folic acid into the blood. Folic acid , sometimes called vitamin B9 , is absorbed and then reduced to dihydro folic acid (DHF) and finally tetrahydro folic acid. Inside peripheral cells, tetrahydrofolate monoglutamates can be extended with glutamate to produce tetrahydrofolate polyglutamates .The term folates customarily includes all folate compounds regardless of one-carbon group or number of glutamate residues. Folates are present in the body only in sub-µM concentrations(Ronner, Peter, PhD;Netter's Essential Biochemistry,36,402-415,2018). Folic acid supplementation via pills and processed foods and natural folate intake through varied dietary sources are common approaches to combat folate deficiency(Jun Zheng, Xingsu Wang, Bangbang Wu, et al; Folate(vitamin B9) content analysis in bread wheat(Triticum aestivum L.),2022). During early or before pregnancy folate consumption helps to prevent deficiency of neural tubes disorders(NTD) such as spinal bifida and anencephaly etc. Although the exact mechanism by which FA facilitates the closure of the neural tube is not entirely known, it is estimated that up to 70% of the NTD can be prevented by supplements containing FA (De-Regil LM, Fernandez-Gaxiola AC, Doswell T, Rena-Rosas JP,2010). Systematic reviews based on clinical studies have elucidated a protective effect of FA on cleft lip and palate (CLP) formation (Blanco R, Colombo A, Pardo R, Suazo J, 2016; Millacura N, Pardo R, et al , 2017; Jahanbia A, Shandkam E, et al, 2018) which is the most common congenital craniofacial malformation and the fourth most prevalent congenital anomaly (American Cleft Palate-Craniofacial Association,2020). Although the protective effect of folate against CL and CLP is recognized, the mechanism due to which certain people possess intrinsically low levels of folate, presumably predisposing their offspring to NTD and other possible congenital pathologies, remains a contentious subject(Bendahan et al, Egyptian Journal of Human Genetics,2020). In 2022 research studies carried out by Hui Xu,Shanshan Wang, Feng Gao, Caihong Li proof that Adequate dietary vitamin B9 and B12 intakes are significantly associated with a better cognitive performance in immediate and delayed memory recall, categorical verbal fluency, processing speed, sustained attention, and working memory among elders assessed in the CERAD, AF, and DSST tests.

Role Of Folate(Vitamin B9) In Neuronal Plasticity

Folates are essential for key biosynthetic processes in mammalian cells and play a crucial role in the maintenance of central nervous system homeostasis. Mammals lack the metabolic capacity for folate biosynthesis; hence, folate requirements are largely met through dietary sources. To date, three major folate transport pathways have been characterized: the folate receptors (FRs), reduced folate carrier (RFC), and proton-coupled folate transporter (PCFT). Folate delivery to the brain primarily occurs at the choroid plexus through FR alpha and PCFT(Camille Alam et al,2020). Impairment in the transport of folate in the brain will lead to array of neurological disorders in early childhood development and also increased permeability of folate in the brain or finding alternative route for folate permeability in the brain could to lead significant therapeutic effect(Camille Alam et al,2020).DNA methylation: Folate helps to form DNA and RNA and is involved in protein metabolism. Folate DNA methylation is an important factor in playing a role influencing gene expression which is important in the synaptic plasticity of the CNS. Vitamin B9 and other vitamins such as vitamin B6(pyridoxine),B12(cobalamine) are the three vitamins that play role in the synthesis of homocysteine. Homocysteine, a sulfur containing amino acid, has established a putative relationship with cognitive decline(Beydoun MA, et al 2014). The association between elevated plasma homocysteine levels and cognitive impairments may be attributed to mechanisms involving the methylation pathways. It has been proposed that homocysteine is associated with brain atrophy, oxidative stress, DNA damage, increased apoptosis, excitotoxicity, and neurodegeneration(Mikkelsen K, et al,2016). Another crucial factor is aging. Aging is commonly accompanied by cognitive function declines, such as processing speed, attention, certain memories, language, visuospatial abilities, and executive functioning(Harada CN et al, 2013). Cognitive changes or deficits that affect one's ability to perform daily activities are considered major neurocognitive disorders(Hui Xu, et al, 2022). Impairment in synaptic remodeling and cognitive function could be possible effect of dysregulated DNA methylation.

Neurogenesis: This is the generation of new neurons from neural stem cells. Folate(vitamin B9) have been found to have essential properties in the formation of new neurons during neurogenesis. Role of folate have been emphasized in neuronal development in different journals and articles. In 2013 studies carried out by Zhang, Guowei Huang, et al, 2013 reveals that folic acid enhances the stimulation of neurons by ischemia of Notch signaling and hippocampal neurogenesis in adult brain and lessens the impairment of cognitive function that occurs after experimental stroke following the study of hippocampal neurogenesis and cognitive function in a rat model of cerbral ischemia. Folate is an important regulator of hippocampal neurogenesis, and folic acid is needed prenatally to reduce the risk of neural tube defects(Wansu Qiu, etal, 2019).

Neuronal plasticity: Brain's ability to adapt to external and internal stimuli mitigating learning, cognition and mood etc. Folate is a cofactor in one-carbon metabolism, during which it promotes the remethylation of homocysteine – a cytotoxic sulfur-containing amino acid that can induce DNA strand breakage, oxidative stress and apoptosis(Mark P Mattsan and Thomas B Shea, 2003). Studies carried out by Lee,J.H., et al in 2010 investigated the effects of dietary folic acid on the expression of myelin basic protein (MBP) in the maternal brain and spinal cord during pregnancy and lactation in rat. From the experiment it was noted that Folic acid deficiency did not increase the expression level of MBP in the cerebral cortex during pregnancy, suggesting that folate intake during pregnancy plays an important role in the maintenance of myelin. Depression is characterized by significant and low mood (Yue Zhou, Yu Cong and Han Liu, 2020), Classical antidepressants are still not adequate in treating depression because of undesirable side effects. Folic acid, a member of the vitamin B complex, in considered to be strongly associated with the function and development of the central nervous system. Studies have showed preclinical evidence that folic acid plays an antidepressant-like role in several pathways involving monoamine neurotransmitters. Thus, folic acid may be used as a potential antidepressant(Yue Zhou, Yu Cong and Han Liu, 2020). In neurodegenerative conditions, including Alzheimer's and Parkinson's diseases, folate deficiency exacerbates cognitive decline through elevated homocysteine levels, contributing to neuronal damage(Ana Filipa Sobral et al,2024). Thus, dietary folate is required for normal development of the nervous system, playing important roles regulating neurogenesis and programmed cell death (Mark P Mattsan and Thomas B Shea, 2003). Dysregulation of folate due to elevated homocysteine can induces defective damage to the CNS causing oxidative stress, excitotoxicity and inflammation.

DISCUSSION

Evidence from animal studies and clinical studies has proofed the importance of folate(vitamin B9) in onset of neuroplasticity. In 2008 from the journal of neuroscience Golo Kronenberg et al studied that folate deficiencies induces neurodegeneration and brain dysfunction in mice lacking Uracil DNA Glycosylase. This study proof that folate deficiency impacts brain function. Folate deficiency leads to increased homocysteine, alters brain monoamine levels, and strongly impairs hippocampal neurogenesis. Whereas these effects of low folate are largely compensated for in wild-type animals, in animals with defective uracil excision repair, along with changes in BDNF, GSH, and selective neurodegeneration, anxious and despair-like behaviors develop. Studies correlating vitamin B9(folate), vitamin B12(cobalamin) , and peripheral neuropathy in children with beta-thelassemia major carried out by Uni Gamayani et al in 2021 showed that cobalamin level correlates with the number of affected nerves in beta thelassemia major patient, and folic acid level correlate the number of affected nerves, especially motoric nerves. Vitamin B9 and vitamin B12 play a role in the hematopoiesis and maintaining function of peripherial nerves (Uni Gamayani et al, 2021). The evidence about whether folic acid can help relieve depression is mixed. Some studies show that 15 to 38% of people with depression have low folate levels in their bodies, and those with very low levels tend to be the most depressed. One study found that people who did not get better when taking antidepressants had low levels of folic acid. A double-blind, placebo-controlled study found that taking 500 mcg of folic acid daily helped the antidepressant Prozac work better in women, but possibly not men. Another study found that taking folic acid and vitamin B12 was no better than placebo in relieving depression in older people(https://www.mountsinai.org/health-

library/supplement/vitamin-b9-folic-acid). RDA: The Recommended Dietary Allowance for folate is listed as micrograms (mcg) of dietary folate equivalents (DFE). Men and women ages 19 years and older should aim for 400 mcg DFE. Pregnant and lactating women require 600 mcg DFE and 500 mcg DFE, respectively. People who regularly drink alcohol should aim for at least 600 mcg DFE of folate daily since alcohol can impair its absorption. UL: A Tolerable Upper Intake Level (UL) is the maximum daily dose unlikely to cause adverse side effects in the general population. The UL for adults for folic acid from fortified food or supplements (not including folate from food) is set at 1,000 mcg a day (Source: https://nutritionsource.hsph.harvard.edu/folic-acid/). Some evidence suggests that folic acid might be helpful in treating depression. Generally, vitamins contain 400 to 800 mcg of folic acid, but some contain other forms of folate (such as 5-MTHF) instead. Folic acid is the only form of folate shown to help prevent NTDs. No scientific studies exist that show that supplements containing other forms of folate can prevent NTDs. It's important to remind patients to check supplement labels to ensure they contain folic acid (https://www.cdc.gov/folic-acid/hcp/clinical-overview/index.html).

CONCLUSION

It is evident that vitamin B9(folate) play an important factor in neuroplasticity through mechanism involving neurogenesis, neurotransmitter synthesis, epigenetic factors and neurotoxicity in the protection of the CNS and brain. Study of cognitive impairment and neurotoxicity, neurodegeneration, brain atrophy, oxidative stress, DNA damage and apoptopsis and excitotoxicity have been established through the study of folate in synthesis of homocysteine with relationship to other crucial B vitamins such as cobalamin (vitamn B12) and pyridoxine(vitamin B6). Folate also demonstrate important factor during pregnancy preventing conditions such as cleft palate formation and anecephaly also reducing the risk of spinal bifida in infant.

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